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Evolution of parasites in the Anthropocene: new pressures, new adaptive directions

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ABSTRACT

The Anthropocene is seeing the human footprint rapidly spreading to all of Earth's ecosystems. The fast-changing biotic and abiotic conditions experienced by all organisms are exerting new and strong selective pressures, and there is a growing list of examples of human-induced evolution in response to anthropogenic impacts. No organism is exempt from these novel selective pressures. Here, we synthesise current knowledge on human-induced evolution in eukaryotic parasites of animals, and present a multidisciplinary framework for its study and monitoring. Parasites generally have short generation times and huge fecundity, features that predispose them for rapid evolution. We begin by reviewing evidence that parasites often have substantial standing genetic variation, and examples of their rapid evolution both under conditions of livestock production and in serial passage experiments. We then present a two-step conceptual overview of the causal chain linking anthropogenic impacts to parasite evolution. First, we review the major anthropogenic factors impacting parasites, and identify the selective pressures they exert on parasites through increased mortality of either infective stages or adult parasites, or through changes in host density, quality or immunity. Second, we discuss what new phenotypic traits are likely to be favoured by the new selective pressures resulting from altered parasite mortality or host changes; we focus mostly on parasite virulence and basic life-history traits, as these most directly influence the transmission success of parasites and the pathology they induce. To illustrate the kinds of evolutionary changes in parasites anticipated in the Anthropocene, we present a few scenarios, either already documented or hypothetical but plausible, involving parasite taxa in livestock, aquaculture and natural systems. Finally, we offer several approaches for investigations and real-time monitoring of rapid, human-induced evolution in parasites, ranging from controlled experiments to the use of state-of-the-art genomic tools. The implications of fast-evolving parasites in the Anthropocene for disease emergence and the dynamics of infections in domestic animals and wildlife are concerning. Broader recognition that it is not only the conditions for parasite transmission that are changing, but the parasites themselves, is needed to meet better the challenges ahead.

Key words: anthelmintics, aquaculture, climate change, fisheries, environmental stressors, life-history traits, pollution, vaccination, virulence.

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I. INTRODUCTION

The Earth's ecosystems are changing rapidly, and so are the organisms they harbour. There are now multiple examples that anthropogenic impacts on the environment exert selective pressures and drive human-induced evolution in wild organisms (Palumbi, 2001; Law, 2007; Allendorf & Hard, 2009; Hendry, Gotanda & Svensson, 2017; Pelletier & Coltman, 2018; Therkildsen *et al.*, 2019; Baltazar-Soares, Brans & Eizaguirre, 2021). The classical example of industrial melanism provides a well-documented case (Kettlewell, 1973). Although the story is complex (Cook & Saccheri, 2013), the simple version involves the replacement of a pale morph of the peppered moth, *Biston betularia*, by a darker morph, as the tree trunks on which the moths rest during the day became darker due to deposition of soot during Britain's Industrial Revolution. Through frequency-dependent selection exerted by avian predators, the genes of the better-camouflaged dark morph spread rapidly through the population, as an indirect consequence of industrial pollution (Kettlewell, 1973). Similarly, different populations of the butterfly *Euphydryas editha* have rapidly adapted to different plants as food sources, after the plants they originally fed on disappeared due to human-induced habitat modifications (logging and cattle ranching); the butterflies no longer accept their ancestral plant food (Singer, Thomas & Parmesan, 1993). Human activities can also impact populations more directly, with equally pronounced evolutionary consequences. For example, the selective targeting by trophy hunters of male bighorn sheep, *Ovis canadensis*, with the largest horns has led to an evolutionary decline in body size and horn size in rams within just a few generations (Coltman *et al.*, 2003). These kinds of rapid evolutionary responses of wildlife to human activities are likely to become more frequent and more pronounced during the next decades of the Anthropocene, as the breadth and severity of human impacts intensify.

Although its official recognition remains controversial (Witze, 2024), the Anthropocene is a widely accepted geological epoch in the history of Earth, starting with the commencement of significant human impacts on the planet's ecosystems. Although various start dates have been proposed for this new age in the geological record, most agree that the mid-twentieth century is the clearest boundary marking the end of the Holocene and the start of the Anthropocene (Lewis & Maslin, 2015; Monastersky, 2015; Subramanian, 2019; Prillaman, 2023). This time coincided with the rapid expansion of the human population and the accelerated pace of industrial production, rapidly growing use of agricultural chemicals, and the rise in other human activities with broad environmental impacts. Here, we will consider the mid-twentieth century as the approximate start of the Anthropocene.

Human-induced evolution can affect all organisms in the Anthropocene, regardless of size, taxon, or trophic level. In particular, in all habitat types, parasites also face the same anthropogenic pressures that impact their hosts (Sures *et al.*, 2017, 2023; Marcogliese, 2023). As hosts and parasites exert strong selective pressures on one another while simultaneously facing new and rapidly changing external pressures, ecological and evolutionary processes can occur within contemporary timeframes (Hendry, 2020). Parasite evolution may therefore take place on ecological timescales in response to rapidly changing conditions. As a result, strong selection can drive rapid evolutionary changes in key parasite life-history traits. The parasites of the butterfly, peppered moth and bighorn sheep mentioned above are also evolving under changing conditions and new selective pressures. This has important implications for the huge research effort aimed at quantifying and/or predicting the impact of anthropogenic stressors on host–parasite interactions and disease risk (see reviews in Vidal-Martinez *et al.*, 2010; Wells & Flynn, 2022; Vicente-Santos *et al.*, 2023). As with free-living

organisms, all current experimental research conducted on the responses of parasites to future environmental conditions consists of exposing today's parasites to tomorrow's conditions. If tomorrow's conditions fall well within the eco-physiological tolerance range of parasites, then tomorrow's parasites may well be the same as today's parasites. However, if changing conditions require rapid adaptation by today's parasites, it will be tomorrow's parasites, not today's, that experience those future conditions. What will these future parasites look like?

Previous reviews of human-driven evolution of host–parasite interactions have mostly focused on how it may change the emergence and dynamics of parasitic diseases (Lebarbenchon *et al.*, 2008; Jones *et al.*, 2013; Rogalski *et al.*, 2017). Here, we focus instead on how anthropogenic impacts might shape the rapid evolution of key parasite traits, leading to future parasites that are genetically, phenotypically, and/or functionally different from today's parasites. We mainly limit our review to life-history traits and closely associated traits, that is the suite of traits associated with the age-dependent schedule of growth, maturation, host exploitation, survivorship and reproduction that are the most pertinent to parasite fitness (Stearns, 1992). We restrict our use of examples to eukaryotic parasites, mostly metazoans; however, the general principles covered here apply broadly to all parasite taxa. We begin with a brief overview of the intrinsic potential of parasites for rapid evolution, illustrated with selected examples. We then connect human impacts to parasite evolution in a two-step chain of causation. First, we review the main anthropogenic factors known to impact living systems and how they affect parasite biology at both individual and population levels. Second, we discuss the selective pressures these factors exert on parasites and the parasite traits most likely to show an evolutionary response. To explore some of these evolutionary responses in greater depth, we provide a few case studies, involving documented examples of contemporary changes in parasite traits under anthropogenic pressures, as well as hypothetical but plausible scenarios predicted from basic theoretical principles. Finally, we propose a set of complementary approaches to study human-induced evolution in parasites exposed to anthropogenic stressors, to allow real-time tracking of changing parasites in a changing world.

II. PARASITES CAN EVOLVE RAPIDLY

Most parasite taxa are characterised by short generation times, huge fecundity for their body size (Calow, 1983), and in some cases also relatively high mutation rates (M'Gonigle, Shen & Otto, 2009). These features should predispose them for rapid evolution. The key life-history traits of parasites are generally quantitative traits (Lande, 1982) whose expression is determined by both the small effects of many genes and environmental influences. Quantitative genetic theory tells us that the short-term selection response of a quantitative trait, that is the change in its mean value from one generation to the next, under directional selection depends on both its heritability and the observed selection differential (Falconer & Mackay, 1996; Maynard Smith, 1998). The heritability of a trait is the ratio of its additive genetic variance to the total phenotypic variance, whereas the selection differential is the difference in the mean value of the trait in a population before and after selection within a generation (Falconer & Mackay, 1996). There is very little existing information regarding the heritability of the parasite traits considered here. However, generally speaking, heritability is lower for life-history traits in comparison with traits less tightly coupled to fitness, such as morphological or physiological traits (Mousseau & Roff, 1987). Nevertheless, under sufficiently strong selective pressure, such as that exerted by anthropogenic factors, life-history traits can show short-term selective changes.

There is evidence of phenotypic, and probably genetic, variation in a range of traits among conspecific parasites. For example, in the trematode *Maritrema novaezealandense*, there are significant differences among different genotypes in how rates of asexual multiplication in the snail intermediate host respond to rising temperatures (Berkhout *et al.*, 2014), in the morphology and phototactic responses of infective stages (Koehler *et al.*, 2011), and in the success of infective stages at penetrating and establishing in various species of crustacean second intermediate hosts (Koehler *et al.*, 2012). In these studies, all parasite genotypes originated from the same locality: they were collected within a few metres of each other. Therefore, in this and other parasite species (e.g. Cwiklinski *et al.*, 2015), substantial phenotypic and genetic variation can exist in local parasite populations, providing the necessary raw material for natural selection and rapid evolution under changing conditions.

The emergence of anthelmintic resistance in parasites of livestock is probably the best-known example of (relatively) rapid evolution in parasites (Sangster, Cowling & Woodgate, 2018). Anthelmintic resistance is a heritable trait through which members of a parasite population become resistant to a particular concentration of a given drug. Resistance to anthelmintic drugs has evolved independently multiple times around the world, in many species of nematodes and trematodes infecting sheep, goats, cattle and other ruminants (Jackson & Coop, 2000; Kaplan & Vidyashankar, 2012). For example, the first reports of resistance to the drug benzimidazole in nematodes of sheep appeared in the early 1960s, just a few short years after this drug was first used on farms; by the early 1990s, resistance to benzimidazole was seen worldwide, with the prevalence of resistance very high in many countries (Waller, 1994). The time needed for resistance to emerge in a parasite population depends on multiple factors acting independently or synergistically: parasite and host species, frequency and dosage of treatments, repeated use of a single drug *versus* alternation of drug types, stock management strategy, environmental factors, etc. (Barnes, Dobson & Barger, 1995; Jackson & Coop, 2000). Resistance to a particular drug is also likely to

evolve more rapidly if it is monogenic, that is conferred by a single gene, and inherited as a dominant trait (Jackson & Coop, 2000). Whatever the underlying genetic mechanisms, the high mortality imposed by chemotherapy gave a clear advantage to individuals carrying resistance genes, driving the rapid increase in their frequency within any population subjected to sustained anthelmintic exposure.

Rapid parasite evolution has also been observed under more controlled conditions, when experimental populations undergo evolutionary changes as a consequence of new selective pressures imposed by researchers (Kawecki *et al.*, 2012). For example, serial passage experiments have shown that parasite virulence can evolve rapidly, increasing substantially in just a few generations when the transmission success of a parasite is experimentally decoupled from its impact on the host (Ebert, 1998, 2000). In these experiments, transmission was ensured by the researchers regardless of how severely host resources were exploited by the parasites, relaxing any selection against high levels of virulence. Laboratory conditions generally exert very different selective pressures on parasites than those they face in nature. For example, after 30 years of serial passage through snail intermediate hosts and mouse definitive hosts in the laboratory, an experimental population of the trematode *Schistosoma mansoni* diverged significantly in virulence and other key life-history traits from the natural population from which it originated (Dias *et al.*, 2023). Some of the best examples of rapid parasite evolution under artificial selection come from experimental studies of the cestode *Schistocephalus solidus*. After only four generations of selection for fast development rate in the copepod intermediate host, faster development indeed evolved, although heritability for this trait was relatively low (Benesh, 2023). This came at no cost to the copepod host, and allowed the parasite to spend less time in the copepod host before reaching infectivity to the next host in its life cycle (Benesh, 2023). In a separate selection experiment, only three generations of artificial selection were sufficient to produce significant changes in the extent to which the cestode manipulated the behaviour of its copepod host (Hafer-Hahmann, 2019).

It remains unclear whether such increases and decreases in trait expression after just a few generations of selection are simply the product of phenotypic plasticity, or whether they reflect genetic changes. However, the latter seem inevitable as phenotypic divergence is often the prelude of genetic divergence (Pfennig *et al.*, 2010). This would be the case if the new selective pressures persist for many generations, such as those currently exerted on natural populations by anthropogenic factors.

III. ANTHROPOGENIC FACTORS AFFECTING PARASITES

Research on the impact of anthropogenic factors on parasites has mostly focused on their responses at two levels: individual-level phenotypic expression of key life-history traits (e.g. stage-specific survivorship, growth rate, body size, fecundity) and population-level epidemiological parameters (e.g. prevalence and intensity of infection). In this section, we briefly review some of the best-studied anthropogenic factors impacting parasites and provide examples of their effects at the individual and/or population level. Many of these impacts are taxon specific, and the review we present is not meant to be exhaustive. Anthropogenic factors are grouped and discussed below according to their most obvious, most frequently observed, or best-documented individual-level impact on parasites. Specifically, in this section we examine the causal relationships linking anthropogenic factors to the selective pressures that they indirectly exert on parasites (i.e. arrows in Fig. 1), the first step in the two-step causal chain leading to rapid parasite evolution.

(1) Chemotherapy

The purpose of anthelmintics and other anti-parasite drugs is to kill parasites. Since they are administered to infected vertebrate hosts, generally these drugs result in increased mortality of adult parasite stages only, not free-living infective stages or juvenile stages within intermediate hosts in the case of parasites with complex life cycles. In susceptible parasite populations (i.e. those where drug resistance is not widespread), drug-induced adult mortality rates can be much higher than natural mortality. For example, a relatively newly synthesised class of anthelmintic compounds (amino-acetonitrile derivatives) can achieve elimination of >90% of adult nematodes of all the major species infecting sheep and cattle (Kaminsky *et al.*, 2008). Similar results are reported for the efficacy of chemotherapy against drug-naïve parasite populations, ranging from anthelmintics used against trematodes in cattle (e.g. Richards *et al.*, 1990) to similar compounds used against ectoparasitic monogeneans and copepods infecting fish in aquaculture (Buchmann, 2022). In the absence of resistance, chemotherapy can thus exert strong selective pressures if sustained across parasite generations.

(2) Host vaccination

Unlike chemotherapy, which exerts totally new selective pressures, vaccines work by eliciting and boosting immune responses that parasites would naturally encounter upon infecting a host. Vaccines improve host immunity, and as a consequence they increase parasite mortality in the early stages of infection, before the parasite can reproduce. For example, vaccination of cattle against the cestode *Echinococcus granulosus* prevented the establishment of the parasite, whereas unvaccinated control animals all became infected (Heath *et al.*, 2012). Vaccines can also reduce parasite growth and production of infective stages. For instance, immunisation of dogs with various types of vaccines against *E. granulosus* did not fully block the establishment of the parasite, nevertheless it impaired worm development and resulted

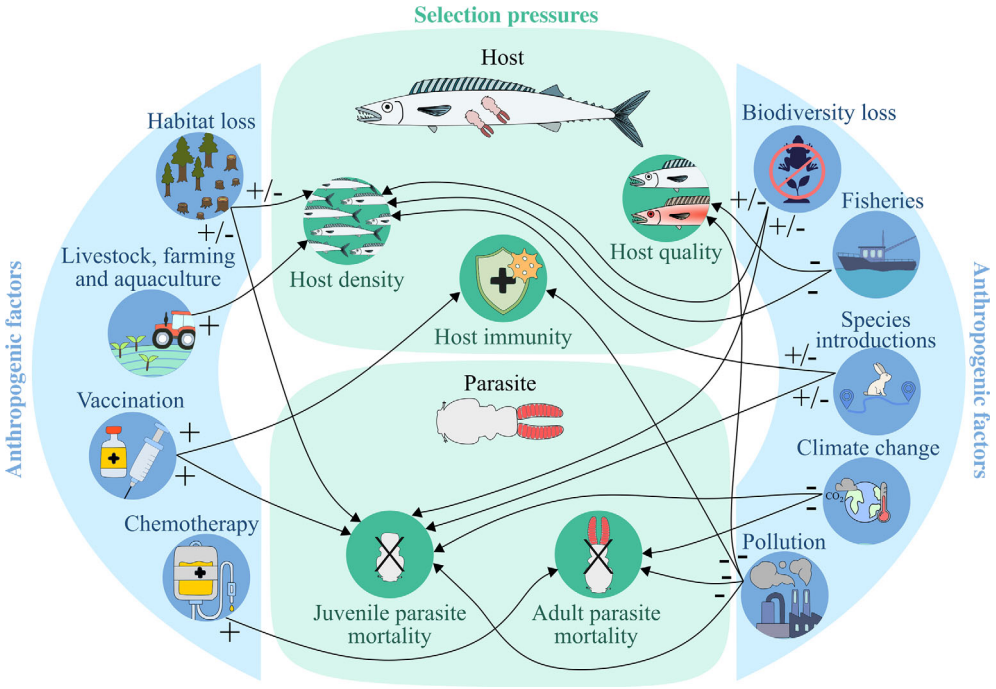

Fig. 1. First part of the two-step causal chain linking anthropogenic factors to rapid parasite evolution. Here, anthropogenic factors impacting the environment are connected to the selective pressures they can exert. For illustrative purposes only, host and parasite are depicted as fish and ectoparasitic copepods; and for simplicity, not all possible connections are shown. The direction of likely effects is indicated as positive (+), negative (−), or (+/−).

in a massive drop in the proportion of worms reaching the egg-producing stage (Zhang *et al.*, 2006). However, not all vaccines are perfect. So-called "leaky" vaccines reduce the pathology experienced by the host, but allow the parasite to survive, reproduce and release infective stages (Read & Mackinnon, 2007). The evolutionary consequences of perfect *versus* imperfect or leaky vaccines can be quite different (see Section IV). Still, vaccines generally prevent the successful establishment of pre-reproductive parasites following infection of the host, and thus cause heavy mortality at this stage.

(3) Livestock production and aquaculture

Animal production has greatly intensified in the past century to meet the increasing demands of our growing population (Thornton, 2010; Naylor *et al.*, 2021). From sheep and cattle to fish and shellfish, the conditions for their parasites are generally the same: a high density of suitable hosts, often genetically and phenotypically much more homogeneous than hosts in natural populations. Maximum host lifespan is also often shorter than in nature, as adult individuals are removed for human consumption, however they are replaced by young, immunologically naïve hosts. In the absence of parasite control measures, these conditions, especially unnaturally high host densities, can greatly facilitate transmission of parasites with direct life cycles. As animal production ramps up and host densities increase both in local facilities and on regional scales, threshold host densities are reached above which parasite outbreaks can occur (Krkošek, 2010). For example, the number of farmed Atlantic salmon, *Salmo salar*, increased gradually in the Bay of Fundy, Canada, as the number and sizes of fish farms increased throughout the 1980s and 1990s. While host density rose, the prevalence and intensity of sea lice, that is the parasitic copepod *Lepeophtheirus salmonis*, on farmed fish remained steady for many years at <20% fish infected with <5 lice per fish on average. However, in the mid-1990s, presumably when host numbers exceeded the threshold density, prevalence suddenly rose to 100% and intensities to >20 lice per fish (Hogans, 1995). In addition to the immediate epidemiological effects of high host densities in animal production systems, there may also be longer-term evolutionary consequences as selection will adjust parasite life-history traits to the new conditions of abundant hosts and facilitated transmission.

(4) Fisheries and intensive harvesting

Harvesting of natural animal populations, if not managed properly, can cause substantial reductions in animal population densities. There is strong evidence of this occurring as a consequence of commercial fishing, with overexploitation of fish populations and ineffective stock management having caused large declines in marine fish populations over the past century (Murawski, 2010; Le Pape *et al.*, 2017; Hilborn *et al.*, 2020). Size-selective harvesting can lead to qualitative

as well as quantitative changes in exploited populations. For example, average body sizes of many fish populations following many years of sustained harvest are often significantly lower than pre-harvest, because of selection on growth rate exerted by biased captures of larger size classes (Allendorf & Hard, 2009; Audzijonyte *et al.*, 2013; Therkildsen *et al.*, 2019). For parasites, lower host densities and host size distributions biased towards smaller sizes may translate into lower transmission probabilities and lower-quality hosts to exploit as resources (Wood, Lafferty & Micheli, 2010). Beyond these immediate epidemiological effects, lower densities and quality of hosts, if they persist across generations, could select for different parasite strategies of reproduction and host exploitation, including host-switching.

(5) Habitat loss and fragmentation

Habitat loss and fragmentation, in their many forms, have multiple negative impacts on natural populations and ecosystems (Fahrig, 1997; Haddad *et al.*, 2015). Destruction of natural habitats or their fragmentation, due to urbanisation and expansion of agricultural land, generally lead to decreases in local biodiversity. Since many free-living species become locally extinct as a result, for some parasites this can mean co-extinction (Colwell, Dunn & Harris, 2012). For parasite species that persist in smaller and patchy habitats, the consequences are often changes in either or both the genetic diversity and the density of their essential host species. Habitat fragmentation can reduce host genetic diversity in some cases (e.g. Dixo *et al.*, 2009), which may lead to more homogenous hosts among which parasites can spread more readily (King & Lively, 2012). Perhaps more importantly, depending on the particular habitat types and host taxa, host density may be higher in small habitat fragments than it was prior to habitat destruction, or it may be lower. In some cases, habitat suitability for essential host species may improve following anthropogenic habitat modification. For example, the formation of reservoirs along river ecosystems in Africa has created ideal habitats for snails serving as intermediate hosts for the trematodes *Schistosoma* spp., resulting in high snail densities and higher infection rates of definitive hosts, including humans (Steinmann *et al.*, 2006). Classical epidemiological models reveal that parasite transmission success and population dynamics are tightly coupled to host population density (Anderson & May, 1978; May & Anderson, 1979). Therefore, depending on whether or not host density is higher or lower in remaining habitat fragments, parasite transmission success can be higher or lower than in the previously undisturbed habitat (McCallum & Dobson, 2002). Several studies report either increases or decreases in the prevalence or intensity of infection by parasites following habitat fragmentation, depending on the type of life cycle, the size and isolation of habitat patches, etc. (see examples in Marcogliese, 2023). In general, specialist parasites with complex life cycles are more likely to be impacted negatively by changes in host density following urbanisation and habitat fragmentation, than are generalist parasites with direct life cycles (see Werner & Dunn, 2020). With changes in host density being the most immediate impacts of habitat modification and fragmentation, the survivorship and host-finding success of infective or juvenile stages of parasites appear to be the aspects of parasite biology most likely to be affected by this anthropogenic factor.

(6) Biotic impacts

From the perspective of altered selective pressures, the two main human-induced biotic changes likely to impact parasites are biodiversity loss and species introductions. Global rates of species extinctions have recently risen dramatically above the background rates prevailing at times between mass extinctions, and significant biodiversity loss is being recorded in a range of habitats across the world (Ceballos *et al.*, 2015; Díaz *et al.*, 2019). Assuming that a parasite and its host(s) persist, changes to local biodiversity can have a range of impacts, depending on the specific circumstances. In many systems, greater biodiversity can dilute infection risk for a focal host species (Civitello *et al.*, 2015; Keesing & Ostfeld, 2021). Although the mechanisms vary from system to system, often they involve the infective stages of parasites either attempting and failing to infect unsuitable host species, in which they perish quickly (Thieltges, Jensen & Poulin, 2008), or being consumed by non-host organisms (Johnson *et al.*, 2010; Thieltges *et al.*, 2013; Koprivnikar, Thieltges & Johnson, 2023). In both instances, local biodiversity loss could mean lower mortality of infective stages and greater infection of the focal host, again assuming the necessary host species persists. If the latter do not, for some parasites with complex life cycles, the extinction of one of their required host species could have very different consequences. If the parasite avoids local extinction, the disappearance of a previously used host species could favour individuals in the parasite population capable of skipping that lost host and adopting an abbreviated life cycle. For example, some trematodes are capable of facultative truncation of their life cycle by accelerating their development in one host and dropping the need to pass through another host (Lefebvre & Poulin, 2005; Lagrue & Poulin, 2007). Therefore, there are many ways in which decreased biodiversity might impact parasite transmission and general biology.

Similarly, species introductions can have a range of effects on parasites (Dunn, 2009; Goedknegt *et al.*, 2016). If a new animal species is introduced into a local community, it may turn out to be an unsuitable host in which the infective stages of a native parasite perish, or it may directly prey on the parasite's infective stages (Stanicka *et al.*, 2021). In both cases, this would increase the mortality of infective stages. By contrast, the newcomer may prove to be a suitable host species that could serve as an alternative host for the parasite, or it may facilitate parasite transmission among native hosts through habitat modification or some other process (Chalkowski, Lepczyk & Zohdy, 2018). For instance, certain introduced species, in particular those that are closely related to a parasite's original hosts, readily become part of the

parasite's host range (Paterson *et al.*, 2012). Parasites may evolve rapidly to exploit successfully a novel host species following an introduction, and use of the novel host may in turn alter their epidemiology and lead to new adaptations (Woolhouse, Haydon & Antia, 2005). If a parasite is itself introduced to a new geographical area along with its original host species, and assuming both parasite and host become established, similar scenarios are possible (Dunn, 2009; Goedknegt *et al.*, 2016). The genetic diversity of the introduced parasite population may be lower than that of its population of origin, due to founder effects (Blakeslee *et al.*, 2020). Nevertheless, either the new community will impose greater (or lower) mortality of infective stages, or it will offer suitable alternative hosts for the parasite to exploit. Many other outcomes are possible, of course, as the circumstances surrounding species introductions vary greatly. What will always be true, however, is that the parasite will find itself in new conditions, experience novel selection pressures and evolve life-history adaptations in response.

(7) Climate and abiotic impacts

Changes in external abiotic conditions resulting from climate change, chemical pollution, and any other form of anthropogenic impact on the environment, can directly affect parasites in at least three ways. Firstly, altered abiotic conditions may prove stressful for parasite eggs and infective stages outside the host, which are exposed to these conditions during transmission (Pietrock & Marcogliese, 2003; Moerman *et al.*, 2023). For example, lower survival, activity and/or infectivity of parasite infective stages has been reported in response to higher temperatures (Shostak & Samuel, 1984; Thieltges & Rick, 2006; Koprivnikar *et al.*, 2010), ocean acidification (Koprivnikar *et al.*, 2010; MacLeod & Poulin, 2015; Franzova *et al.*, 2019), herbicides (Koprivnikar, Forbes & Baker, 2006), and toxic pollution (Evans, 1982; Pietrock & Goater, 2005; Bourgeois, Koski & Scott, 2007). Secondly, abiotic conditions can impact adult ectoparasites since they remain exposed to external factors for their entire life. Higher temperatures generally decrease the lifespan of adult ectoparasites in both aquatic and terrestrial systems (Sereno-Uribe, Zambrano & García-Varela, 2012; Nielebeck *et al.*, 2023), as does exposure to a range of pollutants (Gilbert & Avenant-Oldewage, 2021). Thirdly, external conditions can also affect parasites within their host. In particular, external temperatures can influence the biology of parasites within ectothermic hosts. Notably, the rate at which trematodes multiply asexually inside their molluscan intermediate host is very sensitive to external temperatures (Poulin, 2006). More generally, higher metabolism and rates of resource use in response to higher temperature can accelerate the growth of parasitic worms within ectothermic hosts (e.g. Franke *et al.*, 2017). The combined population-level impacts of the three above ways through which anthropogenically modified external conditions affect parasites vary depending on the factor and parasite taxon involved. However, it is often a negative one. For example, in response to most forms of chemical pollution, many parasite taxa experience a decline in prevalence and intensity of infection in local host populations (Blanar *et al.*, 2009).

In addition to their direct impacts on parasites, anthropogenic environmental changes may indirectly impact parasites by affecting their hosts. On the one hand, suboptimal abiotic conditions can lead to decreases in host abundance, or to more subtle changes in host behaviour (Scott & Sloman, 2004; Jacquin *et al.*, 2020; Nagelkerken *et al.*, 2023), in both cases potentially reducing parasite transmission success by causing greater losses of infective stages. On the other hand, stressful external conditions may also impact the conditions experienced by parasites within their host. For instance, alterations in temperature and other ocean biogeochemical properties associated with global climate change exert stress on fish eco-physiological processes (Pörtner, 2010; Nagelkerken *et al.*, 2023), induce varying levels of immunosuppression (Makrinos & Bowden, 2016; Scharsack & Franke, 2022), and are even predicted to cause a reduction of fish body sizes (Sheridan & Bickford, 2011; Cheung *et al.*, 2012). Similarly, toxic pollutants can impair fish immune responses (Bols *et al.*, 2001). Internal parasites of endothermic hosts are not necessarily spared the effects of climate change; for instance, they are affected indirectly by the thermal stress incurred by their host during heat waves and through global warming (Morley & Lewis, 2014). These kinds of altered within-host living conditions can have not only immediate effects on parasite development and reproduction, but also longer-term evolutionary consequences.

No doubt several other anthropogenic disturbances also affect the biology and transmission of parasites. For instance, recent research has revealed the possible impact of nanoplastic pollution (Mavrianos *et al.*, 2023), noise pollution (Berkhout *et al.*, 2023) and light pollution (Poulin, 2023) on host–parasite interactions. However, the factors reviewed above are sufficient to illustrate the wide-ranging impacts of anthropogenic factors on parasites, highlighting the significant influence of human activities on parasite ecology and, by extension, on parasite evolution.

IV. SELECTIVE PRESSURES AND EXPECTED ADAPTIVE RESPONSES

Through their impact in ecological time, anthropogenic factors exert selective pressures leading to genetic adaptations over evolutionary time, as well as altered disease dynamics (Rogalski *et al.*, 2017). Following from the previous section, here we discuss the second step in the two-step causal chain linking anthropogenic factors to rapid parasite evolution, namely, the relationships linking the selective pressures exerted by anthropogenic factors to the adaptive responses expected in parasite populations subjected to those pressures (i.e. arrows in Fig. 2). We focus on the four main selective pressures exerted by the anthropogenic factors discussed in the previous section. As with the immediate effects of

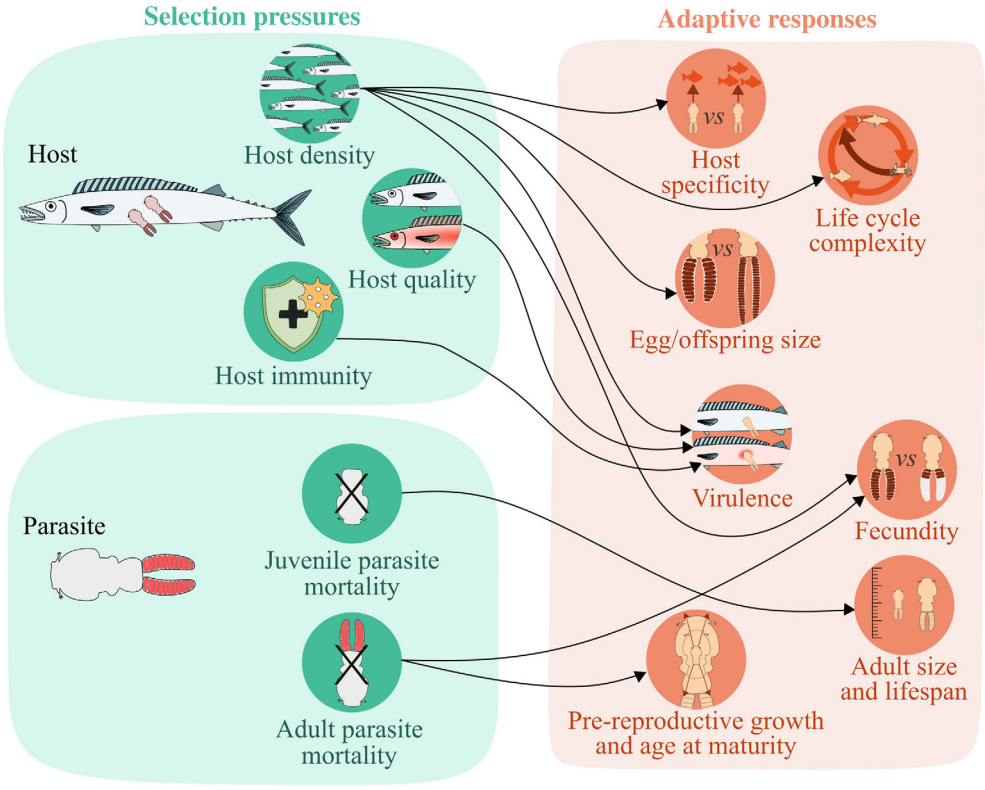

Fig. 2. Second part of the two-step causal chain linking anthropogenic factors to rapid parasite evolution. Here, selective pressures exerted by anthropogenic factors are connected to the expected adaptive changes in key parasite life-history traits. For illustrative purposes only, host and parasite are depicted as fish and ectoparasitic copepods; and for simplicity, not all possible connections are shown. The direction of likely effects is highly contingent on particular conditions and is therefore not shown; it is discussed in Section IV.

anthropogenic factors on parasites, there are no simple generalisations: the resulting adaptive responses are likely to be taxon specific and/or contingent on idiosyncrasies of particular systems, therefore what follows is an illustrative rather than exhaustive list of possible scenarios.

(1) Changes in host density

Host density is a key determinant of parasite transmission success (Anderson & May, 1978; May & Anderson, 1979), and can therefore influence selection for a range of parasite traits. In particular, large increases in host density may select for higher parasite virulence. Here, virulence should be thought of as the rate at which resources are extracted from the host, that is as a parasite trait; the visible consequences in terms of pathology and reduction in host fitness are just means of quantifying virulence. A trade-off is assumed to direct the evolution of virulence towards its optimal level under particular conditions (Anderson & May, 1982; Frank, 1996; Mackinnon, Gandon & Read, 2008; Ebert & Bull, 2007; Alizon *et al.*, 2009). The cost of a shorter transmission window due to reduced host survival at high virulence must be offset by the production of more transmission stages per unit time prior to host death. However, increasing host densities can shift the virulence trade-off by improving the probability of transmission stages finding a new host (Fig. 3). This relaxes the constraint on the evolution of high virulence, because the cost of reduced host lifespan is no longer strictly limiting transmission success. In conditions of high host density, more virulent genotypes, which extract resources from their host at a high rate and therefore have more severe pathological effects, can thrive and spread through a parasite population. Intensive animal production systems provide such conditions. For instance, the facilitated host-to-host transmission experienced by parasites within aquaculture facilities relaxes the constraints normally preventing the evolution of increased virulence (Nowak, 2007; Pulkkinen *et al.*, 2010; Kennedy *et al.*, 2016). We might therefore expect more virulent strains of parasites to spread within aquaculture populations and spill over to adjacent natural populations (Kennedy *et al.*, 2016). Other anthropogenic changes that lead to increased host densities, for example the concentration of host populations in small habitat patches following habitat fragmentation, may have similar evolutionary consequences.

By contrast, habitat loss and fragmentation may also lead to decreases in host density on both local and regional scales; so too could unsustainable fisheries and other forms of over-

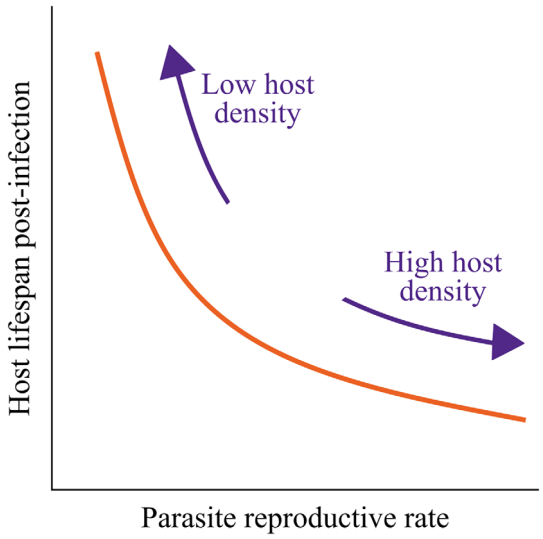

Fig. 3. Trade-off between the rate at which a parasite reproduces within its host by extracting resources from it, and the expected lifespan of the host post-infection. Host density can affect what position along this virulence trade-off curve will be favoured by natural selection for a given parasite. At high host densities, the greater transmission success of infective stages allows more virulent genotypes to thrive and spread, pushing the optimal strategy toward higher reproduction rates at the expense of host survival.

harvesting, as well as host population declines in suboptimal abiotic conditions. Even if the survivorship of infective stages is not affected directly, their probability of finding a suitable host and thus achieving transmission will be lower because there are fewer suitable hosts. In such cases, the above trade-off may push parasite virulence towards lower levels, ensuring a production of infective stages maintained over a longer period even if the daily production rate is lower. However, the directionality of virulence evolution remains difficult to predict following reductions in host density (Bolzoni & De Leo, 2013; Cressler *et al.*, 2015). Alternatively, or additionally, reduced host densities may also select for altered reproductive strategies. In particular, reproductive effort is assumed to be shaped by a trade-off between egg size and fecundity (Roff, 1992; Stearns, 1992). Larger eggs may produce offspring that are better equipped to survive, while high fecundity means more offspring produced. For a given amount of resources and energy allocated to reproduction, egg size and egg numbers cannot both be maximised: the strategy along the continuum from many small eggs to few large eggs that maximises overall fitness will be favoured by selection. For parasites, a decrease in host density should signify a lower probability of transmission, a new pressure that could shift the optimal reproductive strategy along this continuum. For example, populations of the nematode *Rhabdias pseudosphaerocephala* have evolved larger eggs, larger infective larvae and a reduced age at maturity along the edges of the expanding spatial range of their host, the invasive cane toad *Rhinella marina*, in Australia, where host densities are much lower than those in the centre of the host range (Kelehear, Brown & Shine, 2012). These new life-history traits have evolved rapidly, better equipping the parasite's eggs and larvae to survive the longer periods in the external environment they endure before encountering a host in conditions of low host density.

Anthropogenic impacts can also lead to increases in the local density of animal species that were not previously used as hosts, but that are immunologically and physiologically suitable, possibly leading to host-switching and the expansion of the parasite's host range. This can result from species introduction, for instance when a parasite is introduced to a new area and experiences what is, from its perspective, a sudden increase in the local density of potential host species it has not coevolved with (e.g. Lefebvre *et al.*, 2012; Kuchta, Choudhury & Scholz, 2018). Pre-adaptations must allow the parasite to exploit the new host species in the first place, however natural selection would no doubt hone those adaptations to improve the parasite's ability to avoid the new host's defences and successfully exploit its resources.

In the case of extreme reductions in host density or the total disappearance of a required host species, the evolutionary consequences for some parasites with complex life cycles may be different. Species like some trematodes that are capable of skipping a host in their life cycle when this host is not available (see Lefebvre & Poulin, 2005) might evolve from a facultative to an obligate shorter life cycle, possibly even losing adaptations necessary to exploit a host that is no longer available.

(2) Changes in host immunity or quality

In principle, when vaccination extends to a large proportion of the host population (as is often the case in animal production systems), it increases the probability that parasites will face a stronger than normal immune response upon infecting a host. By contrast, a range of abiotic stressors can cause immune depression in vertebrate populations (Bols *et al.*, 2001; Makrinos & Bowden, 2016; Scharsack & Franke, 2022), leading to a higher probability that a parasite encounters a weaker than normal immune response upon infecting a host. In theory, host immunocompetence can be a factor involved in the selection of parasite strains of varying virulence (Alizon *et al.*, 2009; Metz & Boldin, 2023). Although generalisations are not possible, anthropogenic factors leading to stronger host immunity may promote the evolution of less-virulent parasites (but see discussion of "leaky" vaccines in Section IV.3), while those inducing lower host immunity may promote the evolution of more virulent parasites.

Several anthropogenic impacts may also result in hosts of smaller sizes; for instance, both intensive fishing (Allendorf & Hard, 2009; Audzijonyte *et al.*, 2013) and climate change (Sheridan & Bickford, 2011; Cheung *et al.*, 2012) are driving reductions in average and maximum sizes in many fish populations. All else being equal (for example, assuming no concurrent reduction in host density), a

reduction in host size may represent a drop in the average quality of hosts as resources. Host condition, and thus the host's general quality as a resource, can also affect selection on parasite virulence (Seppälä *et al.*, 2008; Cornet *et al.*, 2014). Populations of smaller-bodied hosts may therefore select for parasite genotypes that extract host resources at a lower rate, grow and reproduce more slowly, achieve a smaller body size, and are generally less virulent. This assumes that host body size and quality are limiting factors for parasite growth and reproduction; if that is not the case, changes in host sizes and quality may have no selective consequences for parasites.

(3) Increased mortality of infective/juvenile stages

Anthropogenic factors can increase the mortality of pre-reproductive parasites either when they are free in the environment (infective stages) or just after they have arrived in the host (juvenile stages). In other words, parasite survivorship may be compromised between the release of eggs or infective stages from the host and their entry into the next host, or between entry into that host and the onset of sexual maturity and reproduction.

A range of common anthropogenic pressures can directly decrease the survival and/or infectivity of parasite infective stages. In particular, these include abiotic impacts such as temperature increases, ocean acidification and toxic pollution (Pietrock & Marcogliese, 2003), as well as introduction of non-host species capable of feeding on large quantities of parasite infective stages (Stanicka *et al.*, 2021). Both life-history theory (Stearns, 1992) and empirical evidence (e.g. Lawson, Alvergne & Gibson, 2012) suggest that increased mortality of infective or juvenile stages, if sustained across generations, should select for increased adult lifespan and thus an extended and more prolific reproductive period. When reaching adulthood becomes less likely, selection should favour genotypes that can extend the duration of this life stage. Given the global extent of some of the human-induced changes affecting the survival of parasite infective stages, we may expect such adaptive adjustments in many parasite species.

In principle, a "perfect" vaccine prevents the successful establishment of all pre-reproductive parasites following their entry in the host, and thus results in near-total parasite mortality pre-reproduction. In such cases, the evolutionary consequences may be as described above. However, in reality most vaccines are imperfect, or "leaky", and allow some parasites to survive, establish in the host and reproduce. There are reasons to believe leaky vaccines could lead to evolutionary increases in parasite virulence, because they tend to select for immunity-adapted parasite individuals (Gandon *et al.*, 2001; Read & Mackinnon, 2007; Mackinnon *et al.*, 2008; Day *et al.*, 2022). The vaccine may attenuate the harm caused by the parasite, but without killing it, thus allowing survival of virulent genotypes that extract more resources from the host. In such situations, selection against high virulence may be relaxed, allowing genes associated with virulence to spread.

(4) Increased mortality of adult stages

Formal evolutionary models of life-history strategies and of the interrelationships and trade-offs among life-history traits (Roff, 1992; Stearns, 1992) predict that if adult mortality is increased, selection should favour parasites that postpone maturity and extend their pre-adult growth period. For instance, chemotherapy targeting only or mostly adult parasites reduces adult life expectancy, and thus the duration of the active reproductive period (Skorping & Read, 1998). In many nematodes parasitic in mammals, juvenile stages either migrate through various tissues (e.g. *Ascaris*, *Strongylus*) or burrow into the intestinal wall, and only settle in the gut lumen as adults. They may therefore escape the action of orally administered anthelmintic drugs while in their juvenile stages. Under these circumstances, individuals that have a prolonged pre-reproductive growth period, either as pre-patent juveniles within the definitive host or (in the case of complex-life-cycle helminths) as juveniles in an intermediate host, will attain greater size at maturity. Because larger size results in greater fecundity, these individuals will achieve greater egg output, on average, during their shorter expected adult lifespan, than those with shorter juvenile developmental periods, under a sustained regime of drug exposure and assuming no drug resistance (see Skorping & Read, 1998; Read, Gemmill & Skorping, 2000). Increased adult mortality due to anthropogenic factors may therefore select for longer pre-adult developmental periods, larger adult sizes, and higher rates of egg production. In turn, these new traits may lead to higher virulence, as they are associated with higher rates of resource extraction from the host, and thus greater pathogenicity.

The above scenarios are meant as illustrative, and they are subject to much variation due to the idiosyncrasies of particular host–parasite systems and the variable impacts of anthropogenic factors, treated here as uniform. In addition, several distinct anthropogenic stressors may independently select for similar adaptive changes in parasite traits (see Figs 1 and 2). However, in combination, they may have synergistic effects resulting in even stronger selective pressures. Furthermore, when parasites are exposed to multiple stressors, each affecting the parasite in different ways and exerting different selective pressures, rapid evolution may involve a whole suite of traits.

V. DOCUMENTED AND PREDICTED HUMAN-INDUCED PARASITE EVOLUTION

To illustrate better the many ways in which human-induced factors are shaping parasite evolution in the Anthropocene, we discuss two documented case studies, and two hypothetical but plausible examples.

(1) Chemotherapy and nematodes of livestock

The widespread use of drugs to control nematodes parasitic in livestock has been likened to a global experiment in life-history evolution (Skorping & Read, 1998). As discussed in the previous section and as predicted years ago (Poulin, 1996; Skorping & Read, 1998; Read *et al.*, 2000), by reducing adult life expectancy, anthelmintics may broadly select for delayed maturity, prolonged pre-reproductive growth and larger body sizes (and thus higher daily egg output) in nematodes of ruminants. In the nematode *Teladorsagia circumcincta*, a common gastrointestinal parasite of sheep worldwide, populations that are either resistant or susceptible to anthelmintics (benzimidazoles) have evolved different life-history phenotypes after being subjected to drug treatments for a few generations (Leignel & Cabaret, 2001). Adult body size of susceptible nematodes increased by 6–10%, depending on the treatment, in populations subjected to anthelmintics other than benzimidazoles to which the worms are susceptible, relative to control populations not exposed to any drug (Leignel & Cabaret, 2001). This occurred after only 2 years of treatment. Given the tight correlation between adult body size and egg production, chemotherapy also resulted in more fecund parasites. In nematode species (*Cyathostomum* spp.) parasitic in horses, it has been suggested that the shorter time taken between administration of anthelmintics and the reappearance of eggs in host faeces seen in recent field studies compared to earlier ones may indicate faster parasite development times (Nielsen *et al.*, 2023). In this case, parasites that develop more quickly to maturity and produce offspring prior to the next drug treatment would achieve greater fitness than slow-developing parasites. These two contrasting examples illustrate the selective power of anthelmintic-induced mortality, with the resulting adaptive responses varying according to the parasite taxon under selection and its particular biology.

(2) Aquaculture conditions and sea lice on fish

The sea louse, *Lepeophtheirus salmonis*, an ectoparasitic copepod of Atlantic salmon, *Salmo salar*, is a major pest in aquaculture systems that has been the subject of sustained control efforts, in particular using chemotherapy (Abolofia, Asche & Wilen, 2017). The evolution of drug-resistant sea lice populations was therefore no surprise (e.g. Treasurer, Wadsworth & Grant, 2000; Aaen *et al.*, 2015). This is not the only adaptation of sea lice to the unnatural conditions experienced in aquaculture facilities. As discussed earlier, high host densities within fish farms promote host-to-host transmission and thereby relax the constraints that would otherwise prevent the evolution of high virulence (Nowak, 2007; Pulkkinen *et al.*, 2010; Kennedy *et al.*, 2016). In Norwegian salmon farms, there is evidence that sea lice have indeed evolved towards greater virulence (Ugelvik *et al.*, 2017). This is inferred from the greater extent of skin lesions and reduced growth incurred by parasitised fish hosts in recent years, compared to previous generations, indicating a higher extraction rate of host resources. In turn, this can lead to faster growth of the parasite on its fish host, and earlier maturation and production of transmission stages (Mennerat *et al.*, 2010). In experiments where lice from either salmon farms or from wild fish caught in locations at least 200 km from the nearest salmon farm were allowed to infect fish from a "neutral" batch, lice originating from salmon farms produced more eggs in their first clutch, fewer eggs in later clutches, and died earlier than lice originating from wild fish (Mennerat *et al.*, 2017). These results suggest that aquaculture conditions have selected not just for higher parasite virulence, but also for greater investment into early reproduction at the expense of future fecundity and survival. This may also have been exacerbated by chemotherapy against sea lice, since the drug treatments target both pre-reproductive and adult individuals on fish, the kind of indiscriminate mortality that should favour heavy investment in early reproduction.

(3) Overfishing and ectoparasites

Commercial fishing has caused, and continues to cause, substantial declines in marine fish populations (Murawski, 2010; Le Pape *et al.*, 2017; Hilborn *et al.*, 2020). Because lower host densities decrease the probability of successful transmission for parasites with direct transmission by free-swimming infective stages, they should select for adjustments to reproductive strategies. In particular, the egg size *versus* egg number trade-off may shift towards the production of a greater number of smaller eggs. Some directly transmitted parasites, such as monogeneans, produce eggs more or less continuously; a modified trade-off may be difficult to observe in their case. However, ectoparasitic copepods structure their reproductive output into discrete clutches of eggs, with each female producing paired egg sacs a few times in her lifetime. Assuming little or no change in resource availability (i.e. a female copepod can always obtain sufficient energy from its fish host), for a given investment into a clutch producing more eggs should entail that they must be smaller. There is evidence that selection can shape the egg size *versus* egg number trade-off in parasitic copepods (Poulin, 1995). Taxa parasitic on colonial and sessile invertebrates, where host-to-host transmission is expected to be easy, produce very few but very large eggs relative to their body size. When transmission is almost guaranteed, producing larger offspring well provisioned with energy gives them a head start once they establish on a host. By contrast, taxa parasitic on pelagic fishes, where host-finding and transmission are likely much more challenging, produce many tiny eggs (Poulin, 1995). In those species, a greater number of offspring increases the chances that at least a few will find a host and survive, even if their post-attachment developmental period is longer. There is also ample intraspecific variation in both egg size and numbers of eggs per egg sac in some parasitic copepods (e.g. Timi, Lanfranchi & Poulin, 2005; Cavaleiro & Santos, 2014), providing the raw material for natural selection. We therefore predict that in commercial fish species that have undergone substantial and sustained stock decline, selection will favour

copepod genotypes that produce more and smaller eggs than their ancestors did prior to the overexploitation of their host populations (Fig. 4), a change that may have occurred already.

(4) Global warming and trematodes

The epidemiological responses of parasites to rising ambient temperatures are likely to be idiosyncratic and taxon specific (Marcogliese, 2001; Rohr & Cohen, 2020). However, in the case of trematodes, very similar responses to warmer temperatures are seen in most species. First, rising water temperatures are almost always associated with higher rates of asexual multiplication within the snail intermediate host and therefore with increased production of cercariae, that is infective stages, up to some maximum temperature beyond which there is a sharp decline (Poulin, 2006; Thieltges & Rick, 2006; Studer, Thieltges & Poulin, 2010). Second, the survival of cercariae is almost invariably negatively affected by rising temperature; these are non-feeding stages which use up their glycogen energy reserves faster at higher temperatures (Morley, 2011; Studer *et al.*, 2010; Born-Torrijos *et al.*, 2022). However, within trematode species, some genotypes perform much better at warmer temperatures and others perform better at cooler temperatures (Berkhout *et al.*, 2014). This intraspecific genetic variation in sensitivity to temperature may result in a shift of the parasite's thermal performance curve towards a higher temperature range (see Byers, 2020). Thermal performance curves are reaction norms characterised by a hump-shaped relationship between a fitness component and temperature; selection experiments on various ectotherms have demonstrated that thermal performance curves shift towards higher temperatures across generations in response to warming (Malusare, Zilio & Fronhofer, 2023). Through such a microevolutionary change, genotypes that do better in slightly warmer waters may gradually increase in frequency and replace those that perform better in cooler conditions as global warming intensifies (Fig. 5). Anthropogenic alterations to the environment can thus exert strong pressures on parasite evolution that will not necessarily lead to novel traits, but instead to adjustments in condition-dependent performance (Wolinska & King, 2009).

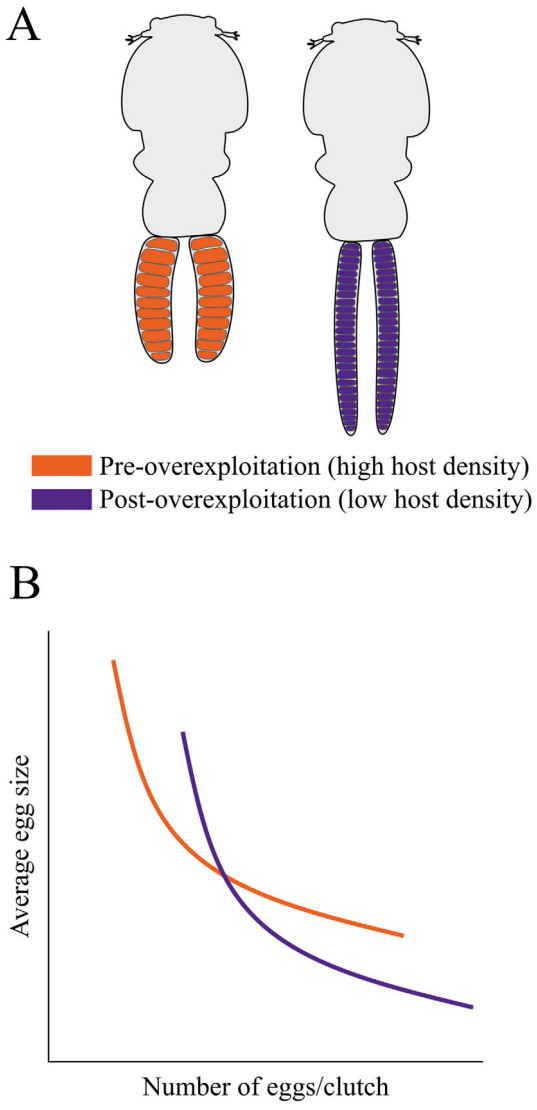

Fig. 4. Hypothetical evolutionary trends in a copepod ectoparasitic on fish as overexploitation from commercial fisheries causes a large decline in host population density. (A) For a given body size, female copepods (here, with uniseriate egg sacs) may be expected to produce more and smaller eggs after the overexploitation of host stocks. (B) The trade-off curve between egg numbers and egg sizes among individual females can be expected to shift towards the many-small-eggs end of the spectrum.

This last example highlights the possibility that, in the face of certain types of anthropogenic factors, parasites may evolve greater tolerance instead of, or in addition to, adjustments to life-history strategies. For example, in an increasingly polluted environment, the infective stages of some parasite species may evolve the ability to cope with higher concentrations of pollutants than they can at present. Future parasites of the Anthropocene may thus differ from past and present parasites in traits ranging from cellular function up to growth and reproduction schedules.

VI. STUDYING HUMAN-INDUCED PARASITE EVOLUTION

Anticipating evolutionary changes in parasite populations is crucial to our ability to mitigate their consequences for wildlife conservation, sustained food production, and human health. We propose five distinct approaches for the study and real-time tracking of human-induced evolutionary changes in parasites. These are not meant to be mutually exclusive; in fact, they are complementary and, combined, would provide a robust tool kit to monitor parasite evolution "as it happens".

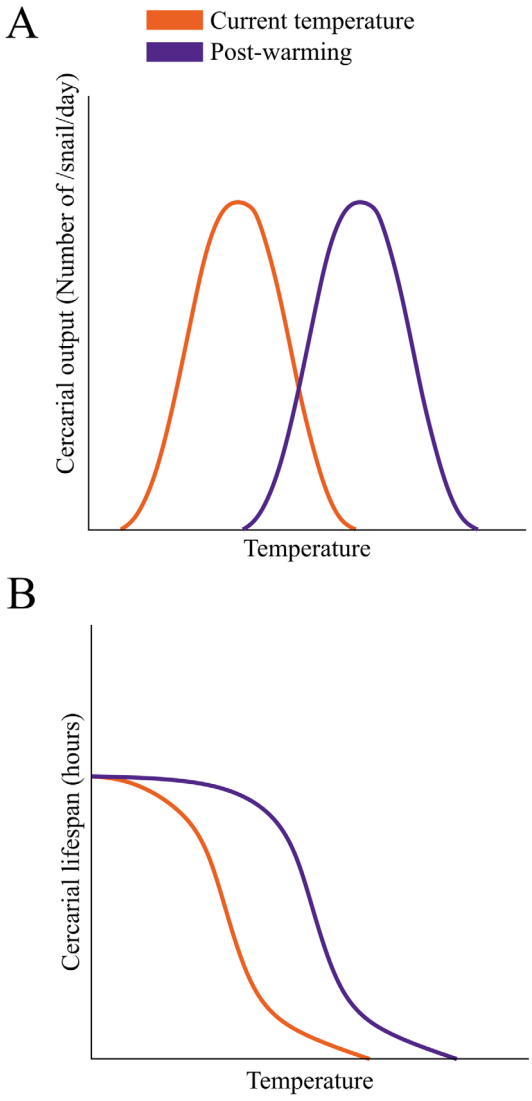

Fig. 5. Hypothetical evolutionary trends in a trematode parasite under a global warming scenario. The asexual production of cercariae (infective stages) within snail hosts (A) and their subsequent survival outside the snail (B) as they seek the next host in their life cycle are key fitness traits. The average population-level thermal performance curves for both traits are shown for today's trematode population and for that expected in a warmer future.

(1) Before-and-after comparisons using historical collections

Comparisons of parasites sampled at different times, for instance before and after the onset of anthropogenic selective pressures, can provide strong evidence of rapid evolution over short timescales. Specimen collections in natural history museums and other institutional repositories are a rich and under-utilised source of historical samples for evolutionary studies (Holmes *et al.*, 2016). Phenotypic changes in parasite populations can be quantified using historical material in a non-destructive manner. For example, historical samples of parasitic copepods collected prior to intensive fishing of their host species can be compared with modern samples obtained from the same location and fish species. As discussed in the previous section, and after accounting for possible shrinkage due to long-term preservation in ethanol or formaldehyde, one can contrast past and present traits such as body size, egg diameter, and number of eggs per ovisac. Paired with data on temporal changes in fish stocks, that is host density, evolutionary change in these life-history traits could be used to test hypotheses about fisheries-driven selection on parasites. Genotypic changes can also be studied using this approach, especially if the genetic basis of phenotypic variation is understood (Wandeler, Hoeck & Keller, 2007; Holmes *et al.*, 2016). This powerful approach has been successfully used to study rapid evolution of butterfly phenotypes in response to climate change (MacLean *et al.*, 2018) and that of avian genotypes in response to introduction to new environments (Stuart *et al.*, 2022). To date, the use of natural history collections in parasitology has been mainly focused on quantifying historical changes in parasite abundances (see Wood *et al.*, 2023), however there is no reason why they could not also serve to track temporal changes in key parasite traits in response to anthropogenic pressures.

(2) Spatial comparisons across populations

In addition to the study of rapid parasite evolution on a temporal scale, one can also seek evidence on a spatial scale (see Blois *et al.*, 2013; Lovell *et al.*, 2023). The nature and intensity of anthropogenic impacts are not uniform in space. For instance, not all comparable ecosystems have been subjected to the same extent of habitat destruction, not all conspecific marine fish populations have been overexploited to the same degree, and pollution levels vary extensively across lakes and rivers. Therefore, selective pressures of anthropogenic origins differ among conspecific parasite populations, allowing for geographical or inter-population comparisons. Local adaptation provides a powerful conceptual tool to study phenotypic and genetic divergence among populations exposed to different local conditions (Blanquart *et al.*, 2013; Wadgymar *et al.*, 2022). Relationships between variation in given traits and corresponding spatial variation in anthropogenic factors can be used to test *a priori* predictions about directional evolution under new selection pressures. To complement such correlations among populations experiencing different local conditions, reciprocal transplant experiments can test the performance and fitness of parasites from anthropogenically impacted localities on hosts from non-impacted localities, and *vice versa*. This approach has proved useful to detect rapid evolution and adaptation of invasive species in their introduced *versus* native range (Colautti & Lau, 2015), and could equally be applied to parasite populations experiencing different degrees of human-driven selection pressure. Alternatively, parasites from a number of localities ranging from non-impacted to highly impacted could be maintained together in mesocosms with a genetically

standard host stock to test whether environmental changes have affected the local evolution of parasite traits.

(3) Tracking ongoing natural experiments

The rapid evolution of parasites can also be tracked in real time in nature, by taking advantage of ongoing human-induced environmental changes in various ecosystems. Just as the widespread use of chemotherapy to control parasites in livestock represents a global experiment in life-history evolution (Skorping & Read, 1998), the gradually increasing acidification of our oceans, the rising concentrations of various toxic pollutants in fresh waters, and the decreasing densities of harvested host populations are all ongoing natural experiments taking place right now. Regular sampling of parasites from habitats undergoing anthropogenically induced changes would provide a time series that can serve to track phenotypic and genetic changes in a parasite population as they happen. As a tragic example, the disastrous release of radioactive isotopes into the atmosphere following the explosion of a reactor at the Chernobyl nuclear power plant in 1986 has provided biologists with a unique opportunity to monitor and study the effects of radiation on the evolution of wild organisms in the following decades (Møller & Mousseau, 2006). In other cases, by simultaneously sampling different and independent populations experiencing similar selective pressures, one can obtain replicate time series, and therefore determine whether parallel or divergent responses occur under similar conditions.

(4) Controlled long-term experiments

As an alternative to natural experiments, experimental evolution studies involve directly exposing populations of organisms to control and treatment conditions associated with different selective pressures, and test their capacity to evolve by measuring phenotypic and/or genetic changes across generations (Garland & Rose, 2009; Kawecki *et al.*, 2012). This approach has the advantages of allowing for standardised replication (using a series of independent aquaria, mesocosms, etc.) and factorial designs involving two or more factors. Selection experiments have recently proved popular to investigate the responses of marine organisms to climate change (Kelly & Griffiths, 2021). For parasites, long-term multi-generational selection experiments are actually possible over short timescales, given the typically short generation times of most parasitic taxa. Strong artificial selection can lead to noticeable phenotypic evolution in metazoan parasites after only a handful of generations (Hafer-Hahmann, 2019; Benesh, 2023). Model parasite species that can readily be maintained in laboratory conditions for tens or even hundreds of generations (e.g. Behnke, Menge & Noyes, 2009; Sulima-Celińska, Kalinowska & Młocicki, 2022) would be perfect candidates for such experiments.

(5) Genomic signatures of selection

Rapid evolution in response to novel environmental challenges can occur by selection acting on quantitative traits that are highly polygenic (Barton & Keightley, 2002). This probably applies to most of the parasite traits discussed above. However, rapid adaptation may also occur *via* a selective sweep, when alleles at a single locus with a large phenotypic effect (or at a few loci each with a moderate effect), which were already present as standing genetic variation or arose by mutations, sweep through the population to achieve high frequencies, or even fixation (Messer & Petrov, 2013; Stephan, 2019). These processes leave a footprint that can be detected in population genomic data. For example, genome sequencing revealed that selective sweeps were responsible for the rapid evolution of a novel behaviour following introduction of Africanised honeybees to a new country (Avalos *et al.*, 2017), and that of insecticide resistance in a pest moth (Calla *et al.*, 2021). The signature of rapid evolution may also be visible in the genomes of parasite populations experiencing strong selection from anthropogenic factors.

Rapid adaptation *via* selective sweeps can be identified in the genome by altered allele frequency spectra (i.e. the distribution of allele frequencies at each genomic site/location) and stronger linkage disequilibrium (i.e. non-random association of alleles) closer to the positively selected loci and weaker in other, more distant, genomic locations (Vitti, Grossman & Sabeti, 2013; Stephan, 2016; Abondio, Cilli & Luiselli, 2022). Furthermore, when comparing populations subject to different selective pressures, some loci may be more divergent among populations than the background (neutral) genetic differentiation in the majority of the loci, and one of the phenomena that can create these highly differentiated (outlier) loci is local adaptation (Nicholson *et al.*, 2002; Beaumont & Balding, 2004; Storz, 2005; Foll & Gaggiotti, 2008). Although rarely applied to eukaryotic parasites, methods to detect such genomic differences among populations, known as genome scans, are plentiful and have been reviewed elsewhere (Narum & Hess, 2011; Vitti *et al.*, 2013; Hoban *et al.*, 2016; Luu, Bazin & Blum, 2017). When correlated with phenotypic data, genome-wide association studies can help uncover the genomic architecture underlying complex traits that may be subjected to natural selection (Barghi, Hermisson & Schlotterer, 2020). An interesting application of such methods detected signatures of a selective sweep increasing the frequency of anthelmintic-resistance genes in the nematode *Haemonchus contortus* (Sallé *et al.*, 2019). Of particular interest would be to target such methods toward parasite traits predicted to respond to anthropogenic changes, such as virulence (which may increase in response to higher host densities, leaky vaccines, and chemotherapy, see Figs 1 and 2) and adult lifespan (which may be longer in response to juvenile mortality caused by perfect vaccines or other biotic/abiotic factors impacting infective and juvenile stages, see Figs 1 and 2).

Environmental factors (e.g. temperature, precipitation) may be expected to impact parasites and hosts equally given their co-adapted evolutionary dynamics, but in fact their impact has been shown to differ by a genotype–environment association (GEA) study in mistletoes (specialist parasites) and their host plant (Walters *et al.*, 2021). There are various methods to perform GEA under diverse population genetic scenarios (de Villemereuil *et al.*, 2014; Lotterhos & Whitlock, 2015; Rellstab *et al.*, 2015), but an interesting feature of GEA methods is their higher sensitivity to detect polygenic adaptation when compared with other genome scans (Forester *et al.*, 2018; De La Torre, Wilhite & Neale, 2019). GEA studies would be invaluable to understand the genomic basis of parasite responses to anthropogenic impacts (pollution, rising temperatures, etc.), even more if coupled with investigations of differential gene expression (such as done for snail temperature tolerance by Gleason & Burton, 2013, 2015, 2016). An extension of GEA methods is the estimation of genomic vulnerability, which uses climatic models and machine learning to predict the potential of current populations to adapt to future climatic conditions (Fitzpatrick & Keller, 2015; Fitzpatrick *et al.*, 2021; Hoffmann, Weeks & Saro, 2021). For example, genomic vulnerability assessments revealed that southern anadromous populations of Arctic charr, *Salvelinus alpinus*, in Canada have limited standing genetic variation and are particularly vulnerable to increasing temperatures, which means these populations are less likely to adapt to predicted future climatic conditions (Layton *et al.*, 2021). Genomic vulnerability estimates are defining source populations and predicting refuges for biodiversity management approaches in the face of climate change, such as for Sino-Himalayan birds and Asian rosewoods (Chen *et al.*, 2022; Hunga *et al.*, 2023). Applied to parasites, it may help predict responses of the "parasites of tomorrow" to different environmental conditions based on genomic patterns of the "parasites of today".

VII. CONCLUSIONS

(1) Life on planet Earth is under novel, strong, and increasing selective pressures as human activities impact all ecosystems. The evidence that many organisms have evolved rapidly since the start of the Anthropocene is mounting. To understand better what future organisms will look like and how they will perform under future conditions, we need to identify the new evolutionary trajectories they are following in response to anthropogenic pressures.

(2) Parasites often have short generation times, huge fecundity for their size, and in some cases substantial levels of standing genetic variation and relatively high mutation rates. Experimental studies have revealed that parasites can evolve rapidly under artificial selection. Multiple examples from the real world confirm this, from the rapid development of drug resistance in parasites of cattle, to the emergence of more virulent parasite strains in aquaculture conditions.

(3) Anthropogenic impacts such as chemotherapy, intensive animal production, fisheries, habitat destruction, pollution and climate change, can affect either host density, immunity or quality as a resource, or they can directly affect parasite survival or transmission. This represents the first part of a two-step causal chain leading to rapid parasite evolution, as these impacts on hosts and parasites translate into strong selective pressures.

(4) The second step in the causal chain links the new selective pressures exerted by anthropogenic factors to the adaptive responses expected in parasite populations, in particular changes in key phenotypic traits such as growth prior to maturity, adult body size, virulence, the egg size *versus* egg number trade-off, or the use of additional host species. The combined effects of distinct selective pressures exerted by multiple kinds of anthropogenic factors render predictions difficult, yet identifying likely evolutionary directions remains an important goal.

(5) Evidence of parasite evolution associated with anthropogenic impacts often emerges after the fact, when it may be too late to manage it. However, human-driven evolution of parasites in the Anthropocene can be studied, quantified, and tracked through a range of correlative and experimental approaches, focused on both phenotypic and genomic signatures of rapid change. Together, this important research can inform current efforts aimed at anticipating and mitigating the future dynamics of parasitic diseases.

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