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Evolutionary Adaptation of Parasites to Anthropogenic Stressors Including Pollution, Habitat Alteration, and Climate Shifts

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Abstract *— Parasites are increasingly recognized as dynamic evolutionary agents rather than passive indicators of environmental degradation. This review synthesizes current evidence for evolutionary adaptation of parasitic taxa to three dominant anthropogenic stressors chemical pollution, habitat alteration, and climate shifts and critically examines how such adaptation modulates their utility as bioindicators. Evidence from field surveys, experimental evolution, and population genomics demonstrates that acanthocephalans and trematodes can evolve metal tolerance within tens of generations, arthropod parasites develop target-site resistance to pesticides with measurable fitness costs, and trematode populations harbor significant genotypic variation in thermal optima that enables selection under warming regimes. Habitat fragmentation consistently favors generalist over specialist parasite lineages, driving biotic homogenization with implications for disease emergence risk. However, the capacity for adaptation is highly heterogeneous across taxa and contexts, constrained by generation time, effective population size, and standing genetic variation. Critically, evolutionary adaptation can decouple parasite responses from current environmental conditions, potentially compromising their sensitivity as early-warning indicators if not explicitly accounted for. The review concludes that parasites retain substantial bioindicator value, but only within an evolutionary-aware framework that incorporates local adaptation, population genetic structure, and the quantification of trade-offs. Future priorities include long-term multigenerational experiments, comparative genomics along environmental gradients, and the development of adaptive bioindicator indices that integrate evolutionary trajectories rather than static prevalence metrics.*

Keywords *— **Evolutionary adaptation; Anthropogenic stressors; Parasite bioindicators; Pollution tolerance; Habitat fragmentation; Climate change***

I. INTRODUCTION

The Anthropocene is characterized by an unprecedented acceleration in the rate and spatial extent of environmental change, with pollution, habitat alteration, and climate shifts representing the three dominant axes of anthropogenic pressure [1, 2]. These stressors exert profound selective forces on all trophic levels, yet the evolutionary responses of parasitic organisms a group often neglected in mainstream conservation and evolutionary biology remain comparatively underexplored. This oversight is paradoxical, as parasites constitute a substantial proportion of global biodiversity, mediate ecosystem function through host population regulation, and drive coevolutionary dynamics [3, 4].

The conventional perspective in ecotoxicology and disease ecology has historically treated parasites as passive indicators of environmental degradation

rather than as active evolutionary agents capable of adaptive change [5]. Early field surveys documented alterations in parasite community structure along pollution gradients, typically reporting declines in species richness and shifts in prevalence without mechanistic interrogation of the underlying evolutionary processes [6]. Subsequent work, however, has fundamentally revised this view: parasites possess generation times often shorter than those of their hosts, large population sizes that generate standing genetic variation, and life cycles that expose free-living stages directly to ambient stressors [7]. These characteristics render parasitic taxa particularly susceptible to rapid evolutionary change under anthropogenic selection.

The existing literature reveals a fragmented understanding of how parasites adapt to anthropogenic stressors. Research on pesticide resistance in arthropod parasites has provided robust evidence of target-site mutations and metabolic detoxification mechanisms, with documented fitness costs and trade-offs [8, 9]. Studies of heavy metal pollution have demonstrated that certain helminth taxa can accumulate metals to concentrations orders of magnitude higher than those in host tissues, suggesting either the evolution of tolerance or physiological sequestration strategies [10]. Investigations of climate change impacts have primarily focused on phenological mismatches and thermal optima, with emerging evidence of genotypic variation in thermal responses across trematode populations [11]. Habitat fragmentation and deforestation have been linked to shifts in parasite community composition favoring generalist over specialist lineages, but the genetic basis of such shifts remains largely speculative [12].

Three major gaps persist. First, comparative analyses across multiple stressor types are rare, limiting our ability to generalize about the tempo and mode of parasite adaptation. Second, the evolutionary costs of adaptation to a single stressor, particularly with respect to virulence, transmission efficiency, and host specificity, remain poorly quantified. Third, the implications of parasite evolutionary adaptation for their utility as bioindicators have received minimal critical scrutiny. If parasite populations are evolving tolerance to anthropogenic stressors, then their sensitivity as sentinels of environmental change may be compromised, a possibility with significant practical consequences for biomonitoring programs.

This review synthesizes the current evidence for evolutionary adaptation of parasites to three primary anthropogenic stressors: chemical pollution (with emphasis on heavy metals and pesticides), habitat alteration (including fragmentation and deforestation), and climate shifts (focusing on temperature change and phenological disruption). For each stressor category, I critically evaluate the mechanisms, rates, and trade-offs of adaptation, drawing upon experimental evolution studies, population genomic analyses, and field surveys. Throughout, I examine how these evolutionary processes alter the utility of parasites as bioindicators in changing ecosystems. The central thesis advanced herein is that parasite adaptation to anthropogenic stressors is not merely an epiphenomenon but a fundamental force reshaping host–parasite dynamics, disease risk, and the informational value of parasitic organisms in environmental monitoring.

II. CHEMICAL POLLUTION AS AN EVOLUTIONARY DRIVER

Chemical pollutants, including heavy metals, pesticides, and emerging contaminants such as microplastics, constitute one of the most pervasive selective pressures in contemporary ecosystems. Parasites, by virtue of their intimate physiological association with host tissues and their exposure to environmental contaminants during free-living stages, experience pollution-mediated selection across multiple life-cycle stages [13].

2.1 Heavy Metals and the Evolution of Tolerance

The capacity of parasites to bioaccumulate heavy metals has been recognized for decades, but only recently have studies demonstrated that such accumulation may reflect evolved tolerance rather than passive absorption [10, 14]. Acanthocephalans, in particular, exhibit remarkable metal bioconcentration capacities, with parasite-to-host tissue concentration ratios exceeding 100-fold for elements such as cadmium and lead [15]. Whether these high accumulation levels represent an adaptive mechanism for metal detoxification or a non-adaptive consequence of parasite physiology remains debated,

but several lines of evidence support the former interpretation.

First, comparative studies across polluted and reference sites reveal elevated metal tolerance in parasite populations from contaminated habitats. For instance, acanthocephalans collected from fish in metal-polluted rivers exhibit higher survivorship following experimental metal exposure compared to conspecifics from clean sites, suggesting local adaptation [16]. Second, transcriptomic analyses have identified upregulation of metal-binding proteins (e.g., metallothioneins) and heat shock proteins in parasites from contaminated environments, indicating a regulated physiological response rather than passive accumulation [17]. Third, experimental evolution studies with trematode–snail systems have demonstrated that multigenerational exposure to cadmium selects for increased tolerance, although this tolerance may trade off against resistance to other stressors [18].

However, the evolutionary rate of metal tolerance in parasites appears highly variable across taxa and contexts. Parasites with complex life cycles and low effective population sizes may exhibit limited adaptive capacity, whereas those with high fecundity and short generation times can respond within tens of generations [19]. Table 1 summarizes key findings on metal tolerance evolution across parasitic taxa.

Table 1. Comparative evidence for heavy metal tolerance evolution in parasitic taxa

Parasite taxon	Metal	Experimental design	Evolutionary response observed	Estimated selection coefficient	Citation
Pomphorhynchus laevis (Acanthocephala)	Cd, Pb, Zn	Field comparison (polluted vs. reference rivers)	Elevated metal tolerance in polluted populations; bioaccumulation factors 100–400× host tissue	Not quantified	[15]
Biomphalaria glabrata (snail host) infected with *Schistosoma mansoni*	Cd	Multigeneration laboratory exposure (10 generations)	Parasite-resistant strain more metal-sensitive than susceptible strain; trade-off between resistance and tolerance	~0.12 per generation	[18]
Philometra ovata (Nematoda)	Cr, Cu, Pb, Cd, Ni, Zn	Field biomonitoring (Jhelum River)	Parasite-to-muscle ratios 3.2–121.7; highest accumulation in most polluted sites	Not quantified	[20]
Dentitruncus truttae (Acanthocephala)	Multiple metals	Transcriptomic analysis	Upregulation of metal-binding proteins; evidence for adaptive molecular mechanisms	Not applicable	[17]

2.2 Pesticide Resistance and Its Fitness Consequences

The evolutionary response of parasites to pesticides has been most intensively studied in arthropod vectors and agricultural pests, where resistance mutations have been documented to spread rapidly under strong selection [8, 21]. In the malaria vector *Anopheles gambiae*, pyrethroid resistance mediated by mutations in the voltage-gated sodium channel (Vgsc-1014F and Vgsc-1014S) has increased from <5% allele frequency in 2013 to >70% in some Senegalese populations by 2018, representing a remarkable rate of evolutionary change [21]. However, such resistance often carries substantial fitness costs in the absence of the selecting agent, including delayed development and reduced survivorship [22].

For parasitic flatworms, the picture is more complex. Trematodes exposed to sublethal concentrations of pesticides exhibit altered cercarial emergence

patterns, reduced infectivity, and shifts in life-history allocation [23, 24]. Experimental studies with *Echinostoma* spp. have demonstrated that pesticide exposure selects for faster cercarial emergence but at the cost of reduced post-encystment survival, consistent with a trade-off between transmission rate and longevity [25]. Importantly, the direction and magnitude of these evolutionary responses depend critically on the specific pesticide, the parasite species, and the environmental context, precluding simple generalizations.

The collateral consequences of the evolution of pesticide resistance for host–parasite interactions have only recently been appreciated. In an elegant experimental evolution study, Jansen et al. [26] showed that selection for carbaryl resistance in a non-parasitic invertebrate inadvertently increased susceptibility to parasite infection, suggesting that resistance to one stressor may compromise defense against another. Conversely, parasitism itself can modify the fitness costs of insecticide resistance, with resistant mosquitoes experiencing higher or lower relative fitness depending on the specific resistance mechanism and the parasite species involved [27]. These context-dependent interactions underscore the complexity of predicting evolutionary trajectories under multifactorial anthropogenic selection.

Table 2 presents a synthesis of the evolution of pesticide resistance in parasitic and vector organisms.

Table 2. Pesticide resistance evolution in parasitic arthropods: mechanisms and fitness costs

Species	Parasite/vector type	Pesticide class	Resistance mechanism	Fitness cost	Reference
Anopheles gambiae	Malaria vector	Pyrethroids	Vgsc-1014F target-site mutation	Delayed larval development (2–3 days longer); reduced pupation rate	[22]
Anopheles gambiae s.l.	Malaria vector	Pyrethroids	Vgsc-1014F and Vgsc-1014S; allele frequencies 3–90% across Senegal	Increased generation time; geographic variation in cost magnitude	[21]
Tetranychus urticae	Agricultural pest	Etoxazole, pyridaben	Point mutations in target site	Not quantified; rapid resistance development within 16 years	[28]
Cimex hemipterus	Ectoparasite	Pyrethroids	Super-kdr mutations	Not quantified; resistance associated with global range expansion	[29]
Daphnia magna (host) infected with *Pasteuria ramosa*	Host–parasite system	Carbaryl	Exposure-induced resistance evolution	Increased susceptibility to parasite infection (collateral damage)	[26]

2.3 Microplastics: An Emerging Selective Agent

Microplastic pollution represents a novel and rapidly expanding environmental stressor whose evolutionary implications for parasites are only beginning to be explored [30]. Unlike conventional chemical pollutants, microplastics exert their effects through physical interference, vector-borne pathogen transport, and indirect alteration of host susceptibility [31, 32]. Laboratory studies have demonstrated that microplastic ingestion can increase amphibian

susceptibility to chytrid fungal infection in a dose-dependent manner, suggesting that microplastics may modulate host–parasite dynamics through immunomodulatory effects [31]. Conversely, exposure to nanoplastics has been shown to reduce the prevalence of phytoplankton infections by physically aggregating around host cells and interfering with parasite attachment [33].

The evolutionary consequences of microplastic exposure for parasites remain speculative, but several plausible scenarios exist. First, if microplastics reduce transmission efficiency, selection may favor parasites with alternative transmission strategies or enhanced host-seeking behaviors. Second, microplastics that serve as vectors for opportunistic pathogens [34] could create novel selective pressures for parasites capable of exploiting plastic-associated microbial communities. Third, the immunomodulatory effects of microplastic ingestion on hosts could alter the within-host competitive environment, favoring parasite genotypes with enhanced immune evasion capabilities. Addressing these hypotheses will require integrated experimental evolution and mechanistic studies.

III. HABITAT ALTERATION AND PARASITE EVOLUTIONARY RESPONSES

Habitat alteration encompassing deforestation, fragmentation, agricultural intensification, and urbanization represent a dominant anthropogenic driver of biodiversity change with profound implications for host–parasite systems [12, 35]. Unlike pollution, which acts primarily through direct physiological toxicity, habitat alteration modifies the ecological context in which parasites complete their life cycles, altering host availability, transmission pathways, and selective regimes.

3.1 Fragmentation and the Shifting Balance between Specialist and Generalist Parasites

Theoretical models predict that habitat fragmentation should favor generalist over specialist parasites because generalists can exploit a broader range of host species, thereby buffering against local host extinctions [36]. Empirical evidence strongly supports this prediction. In a large-scale study of avian haemosporidian parasites in the Western Ghats, India, Dharmarajan et al. [12] demonstrated that anthropogenic disturbance reduces the prevalence of specialist parasite lineages while increasing that of generalist lineages. This biotic homogenization of parasite communities has important implications: generalist parasites are more likely to cause emerging infectious diseases, suggesting that habitat alteration may increase the risk of disease emergence in the Anthropocene [37].

The evolutionary mechanisms underlying this shift remain incompletely resolved. One possibility is that generalist parasites possess greater phenotypic plasticity, enabling them to track host resources across fragmented landscapes [38]. Alternatively, fragmentation may impose stronger selection for host-switching ability in specialist parasites, leading to the evolution of broader host ranges, a process that could occur within tens of generations, given the high fecundity of many parasite taxa. A third, non-mutually exclusive explanation is that fragmentation reduces gene flow among parasite populations, facilitating local adaptation to the host community present in each fragment [39]. The geography of parasite local adaptation can operate at continental scales, with parasites performing best on the host species most common in their location of origin [40].

Table 3 synthesizes key findings on habitat alteration and parasite community structure.

Table 3. Effects of habitat fragmentation and deforestation on parasite community composition and evolutionary trajectories

Parasite system	Habitat alteration type	Observed effect	Proposed evolutionary mechanism	Reference
Avian haemosporidians (*Plasmodium*, *Haemoproteus*)	Deforestation (Atlantic Forest, Brazil)	Increased prevalence in deforested sites; negative relationship with host functional diversity	Reduced host functional diversity increases encounter rates; vector abundance changes	[41]

Avian haemosporidians (Western Ghats, India)	Anthropogenic disturbance gradient	Specialist lineages decline; generalist lineages increase	Broader host range buffers against local host extinctions	[12]
Multiple parasite taxa	Forest fragmentation (thesis)	Increased parasite prevalence in fragmented habitats	Altered host density and movement patterns	[35]
Striga hermonthica (parasitic plant)	Agricultural landscape	Local adaptation to dominant cereal host species	Parasite performs best on host species common in origin location	[40]
Cavity-nesting bees and their parasites	Reforestation types (Amazonia)	Parasite interaction networks differ across reforestation types	Host functional diversity mediates parasite specialization	[42]

3.2 Deforestation and Vector-Borne Parasites

Deforestation exerts particularly pronounced effects on vector-borne parasites, as forest clearance alters the abundance and composition of both vector and reservoir host communities [41, 43]. In the Brazilian Atlantic Forest, Fecchio et al. [41] documented that avian malaria prevalence increases with deforestation, and both *Plasmodium* prevalence and taxonomic diversity are negatively related to host functional diversity. This pattern suggests that deforestation reduces the functional redundancy of host communities, thereby increasing encounter rates between susceptible hosts and infected vectors. Importantly, temperature and host phylogenetic diversity did not influence prevalence, indicating that ecological rather than evolutionary filters dominate the response at least over the timescale of the study.

Whether deforestation drives evolutionary adaptation in parasites, rather than merely filtering existing lineages, remains an open question. The loss of forest cover reduces the effective population size of many host species, potentially imposing bottlenecks that reduce genetic diversity in associated parasites [44]. However, parasites with rapid generation times and large population sizes may respond to deforestation-induced changes in host community composition through selection on host recognition loci [45]. Genomic studies comparing parasite populations across deforestation gradients could resolve whether observed shifts in prevalence and lineage composition reflect plastic responses, ecological filtering, or genuine evolutionary change.

3.3 Urbanization as a Selective Filter

Urban environments impose a unique suite of selective pressures, including elevated temperatures (urban heat island effect), altered resource availability, and novel pollutant cocktails [46]. Parasites inhabiting urban hosts face distinct challenges: reduced host population connectivity, increased host densities in some contexts, and exposure to anthropogenic food sources that may alter host immunity. Studies of avian malaria parasites in urban versus rural habitats have yielded mixed results, with some reporting higher prevalence in urban areas and others detecting no difference [47]. The most consistent finding is a reduction in parasite species richness in urban habitats, suggesting that urbanization acts as a selective filter favoring tolerant or generalist parasite lineages.

IV. CLIMATE SHIFTS AND PARASITE EVOLUTIONARY TRAJECTORIES

Climate change is altering temperature regimes, precipitation patterns, and the phenological synchrony of interacting species. For parasites with complex life cycles involving multiple hosts and free-living stages, climate shifts can disrupt transmission pathways, shift thermal optima, and impose novel selective pressures [48, 49].

4.1 Thermal Adaptation and Its Limits

The potential of parasite populations to adapt to rising temperatures depends critically on the amount of standing genetic variation for thermal tolerance traits

[50]. Studies of trematode parasites have demonstrated significant genotypic variation in cercarial emergence rates and activity across temperature gradients, indicating the raw material for evolutionary response [11]. For the marine trematode *Maritrema novaezealandensis*, different genotypes exhibit divergent responses to elevated temperature (20°C versus 15°C), with some maintaining high cercarial output while others show marked declines [51]. This genotypic variation suggests that natural selection could act to increase thermal tolerance over multiple generations.

However, the limits of thermal adaptation are defined by physiological constraints. Trematode cercarial emergence is temperature-dependent, peaking at a species-specific thermal optimum and declining rapidly at supraoptimal temperatures [52]. If these optima are constrained by fundamental biochemical processes such as the thermal stability of key enzymes or the integrity of membrane structures, then evolutionary shifts may be limited. A comparative analysis across trematode species found substantial variation in trait means and variances across temperatures, suggesting that some taxa possess greater evolutionary potential than others [52]. Parasites with narrow thermal windows and low genetic diversity may be particularly vulnerable to climate-driven extinction, whereas those with broad thermal tolerances may expand their geographic ranges.

The thermal optimum for transmission can be altered by interventions that target temperature-sensitive life stages. In a modeling study of *Schistosoma mansoni*, Aslan et al. [49] showed that transmission risk peaks at 21.7°C, and simulated snail control interventions increased this optimum by up to 1.3°C because intervention-related mortality overrode natural thermal constraints. This finding has important implications: climate change will increase the risk of schistosomiasis in regions where surface water temperatures approach the thermal optimum, but the magnitude of this effect depends on the intensity of vector control efforts.

4.2 Phenological Mismatch and Host–Parasite Synchrony

Climate-driven shifts in phenology, the timing of seasonal life-cycle events, can disrupt the synchrony between parasites and their hosts, with cascading consequences for transmission dynamics [53]. Parasites with complex life cycles depend on the temporal overlap of susceptible host stages; if warming advances host emergence more rapidly than parasite emergence, transmission may decline. Conversely, if parasites exhibit greater phenological plasticity or respond more strongly to temperature cues, transmission may intensify.

Parasitoid–host systems provide particularly well-studied examples. In solitary bees and their parasitoids, warming may amplify the synchrony of emergence phenologies, potentially increasing parasitism rates and reducing host reproductive success [54]. However, community context modifies these responses: the presence of alternative host species can support interaction persistence across a wider range of phenological mismatches [55]. This finding suggests that biodiversity loss itself, exacerbated by climate change, may reduce the buffering capacity of ecological communities, making host–parasite systems more vulnerable to climate-driven disruption.

For parasites that rely on free-living transmission stages, precipitation changes may be as important as temperature. Drier conditions reduce the survival of many free-living parasite larvae, selecting for genotypes with enhanced desiccation resistance or altered transmission strategies [56]. The evolution of such traits has been documented in nematode parasites of livestock, where selection for enhanced survival off-host has occurred in response to changing grazing management practices [57]. Whether similar responses will occur under climate-driven shifts in precipitation remains to be determined.

Table 4 summarizes key findings on parasite responses to climate-related stressors.

Table 4. Evidence for thermal adaptation and phenological responses in parasitic taxa under climate change

Parasite species	Climate stressor	Response metric	Key finding	Reference
Maritrema novaezealandensis (Trematoda)	Elevated temperature (15°C → 20°C)	Cercarial output, activity	Significant genotypic variation in thermal response; evolutionary potential present	[51]
Multiple trematode species	Temperature gradient	Trait means and variances	Substantial interspecific variation in thermal optima and plasticity	[52]
Schistosoma mansoni	Temperature (modeled)	Transmission risk	Thermal optimum 21.7°C; snail control shifts optimum +1.3°C	[49]
Solitary bee parasitoids	Warming (phenological)	Parasitism rates	Warming may amplify emergence synchrony; community context modifies effect	[54, 55]
Biomphalaria spp. (snail host) and *S. mansoni*	Temperature + interventions	Thermal optimum	Intervention mortality overrides thermal constraints; Topt shifts	[49]

V. SYNTHESIS: PARASITE ADAPTATION AND BIOINDICATOR UTILITY

The preceding sections have established that parasites can evolve in response to anthropogenic stressors, albeit at rates and magnitudes that vary substantially across taxa, stressor types, and environmental contexts. This evolutionary capacity has profound implications for the utility of parasites as bioindicators of ecosystem health, a role that has been advocated for decades but rarely critically reexamined in light of contemporary evolutionary knowledge.

The conventional rationale for using parasites as bioindicators rests on three pillars: (1) parasites are sensitive to environmental change, often responding more rapidly than free-living organisms; (2) parasites integrate exposure over time and space because they accumulate pollutants from host tissues; and (3) parasite community structure reflects the cumulative impact of multiple stressors [5, 10]. Each of these assumptions is challenged by the evidence for parasite evolutionary adaptation.

First, if parasite populations evolve tolerance to pollutants, their sensitivity as early warning indicators may be compromised. A parasite population that has adapted to heavy metal contamination will no longer exhibit the same dose–response relationship as a naive population, potentially leading to false negatives in biomonitoring programs. This problem is particularly acute for acanthocephalans and other helminths proposed as sentinel organisms for metal pollution [15, 58]. The high metal accumulation factors reported for these parasites (often exceeding 100× host tissue concentrations) may reflect evolved tolerance mechanisms rather than passive bioaccumulation, and the magnitude of accumulation may vary with the degree of local adaptation.

Second, the assumption that parasites integrate exposure over time implicitly assumes that the parasite population is at evolutionary equilibrium with its environment. If adaptation is ongoing, the relationship between pollutant concentration and parasite response is time-dependent, complicating the interpretation of field data. Experimental evolution studies demonstrate that tolerance can evolve within tens of generations [18, 26]; over the timescale of typical biomonitoring programs (years to decades), such evolutionary change cannot be ignored.

Third, the use of parasite community composition as an indicator of ecosystem health assumes that the observed community reflects contemporary

environmental conditions rather than evolutionary history. However, if habitat fragmentation has selected for generalist parasites over specialists [12], the resulting community may reflect past selection as much as current conditions. Distinguishing between ecological filtering and evolutionary adaptation in parasite community data is methodologically challenging but necessary for accurate bioindication.

Despite these challenges, parasites retain considerable value as bioindicators provided that evolutionary considerations are explicitly incorporated into study design and interpretation. Several strategies can mitigate the confounding effects of adaptation. First, the use of molecular markers to assess population genetic structure and detect signatures of selection can identify whether observed patterns reflect plasticity, ecological filtering, or genuine evolutionary change. Second, reciprocal transplant experiments (collecting parasites from polluted and reference sites and exposing them to common conditions) can quantify the extent of local adaptation and adjust bioindicator thresholds accordingly. Third, focusing on parasite taxa with limited evolutionary potential, such as those with low genetic diversity, long generation times, or obligate sexual reproduction, may reduce the risk of adaptation confounding bioindicator signals.

VI. CONCLUSION

Parasites are not passive victims of anthropogenic environmental change but active evolutionary agents capable of adapting to pollution, habitat alteration, and climate shifts. The evidence reviewed herein demonstrates that parasitic taxa can evolve metal tolerance, pesticide resistance, altered transmission strategies, and shifted thermal optima over timescales relevant to ecological monitoring and disease management. However, the capacity for adaptation is not uniform: parasites with high fecundity, short generation times, large population sizes, and substantial standing genetic variation exhibit the greatest evolutionary potential, whereas those constrained by low diversity or complex life cycles may lag behind environmental change.

The evolutionary costs associated with adaptation are particularly consequential. Trade-offs between resistance and virulence, between tolerance to one stressor and susceptibility to another, and between transmission rate and longevity create complex fitness landscapes that defy simple predictions. Understanding these trade-offs requires integrated approaches combining experimental evolution, population genomics, and mechanistic physiology, a research agenda that remains largely unrealized.

For the use of parasites as bioindicators, evolutionary adaptation presents both challenges and opportunities. The challenge is that adaptation can decouple parasite response from current environmental conditions, potentially leading to misinterpretation of biomonitoring data. The opportunity is that the very processes of adaptation, shifts in allele frequencies, changes in life-history traits, and alterations in community composition provide rich information about the selective pressures operating in an ecosystem. By measuring not only the presence or absence of parasites but also their evolutionary trajectories, researchers can gain insights into the intensity, duration, and nature of anthropogenic stressors that are inaccessible from free-living bioindicators alone.

Future research must prioritize three directions. First, long-term experimental evolution studies across multiple generations are needed to quantify rates of adaptation, characterize the genetic architecture of tolerance traits, and map fitness trade-offs under realistic multifactorial stressor combinations. Second, comparative genomic analyses of parasite populations along pollution, fragmentation, and climatic gradients can identify loci under selection and reveal whether parallel evolutionary responses occur across independent populations. Third, the development of evolutionary-aware bioindicator frameworks incorporating estimates of local adaptation, population genetic structure, and evolutionary potential will be essential for maintaining the utility of parasites in environmental monitoring as anthropogenic pressures intensify.

The Anthropocene is an era of rapid evolutionary change, and parasites are at the forefront of this transformation. Recognizing their capacity for adaptation does not diminish their value as bioindicators but rather reframes it: parasites inform us not only about the state of the environment but also about the evolutionary processes that determine how organisms, including humans, will respond to a changing planet.

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