THE IN A HURRY SERIES

Parasites

in a Hurry

The freeloaders that shape everything

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The Whole Thing in One Page

The word parasite makes most people picture something pale and wriggling in an intestine. That is a spectacularly narrow sample.

Parasitism is not one branch of life. It is a relationship that evolution has invented again and again. Worms, insects, mites, crustaceans, fungi, single-celled eukaryotes and thousands of flowering plants all contain lineages that live at another organism's expense. Some feed for minutes. Some remain for years. Some steal blood, some sap, some tissue, some food already digested by the host, and some use a host's body as a nursery. What unites them is dependence on living infrastructure.

That dependence changes the problem of survival. A free-living organism must find food, water, shelter and the right physical conditions. A parasite can outsource much of that work to a host. The bargain is severe. The resource moves, defends itself and can evolve. A useful host is therefore both opportunity and obstacle.

Every parasite must solve four problems. It must reach a suitable host, establish on or inside it, take enough without losing the relationship too soon, and send descendants onwards. Those problems explain most of the apparent strangeness. Tapeworms can lose a gut because the host has already digested the meal. Parasitic plants build haustoria that tap another plant's vascular system. African trypanosomes repeatedly change the surface molecules seen by antibodies. Trematodes can divide their life cycle among snails, fish and birds because each host performs a different job. Complexity is often dependency made visible.

Damage is a consequence, not the parasite's objective. Natural selection rewards transmission. That can favour restraint, heavy exploitation or something between them depending on route, timing, host behaviour, vectors, immunity and competition among parasite strains. The old idea that parasites inevitably evolve towards harmlessness is no better than the idea that successful parasites maximise sickness.

Some parasites alter host behaviour, but the evidence must earn the word manipulation. A changed animal may be sick, defending itself or suffering collateral damage. The strongest cases link a stage-specific change to increased parasite transmission. The weakest become stories about mind control because spectacle outruns mechanism.

The subtitle matters most at larger scales. Parasites are not biological litter around the edges of ecosystems. They add large numbers of links to food webs, can regulate host populations, redirect energy and disappear when their hosts disappear. They also shape evolution. Malaria helped maintain human haemoglobin variants with severe consequences. Parasites can favour rare host genotypes, reward recombination and drive repeated changes in immunity, behaviour and life history.

The central reversal is this: a host is not merely a victim. It is a living environment that detects, repairs, resists, tolerates and evolves. Once life becomes useful to other life, parasitism becomes likely. Once parasitism appears, hosts themselves are changed by the pressure. The freeloaders help design the building they live in. That is why parasites belong in any serious account of adaptation, immunity, population dynamics and biological diversity. They are consumers, passengers, competitors and selective pressures at once, often hidden because their habitat is another organism. Their invisibility has repeatedly made biology underestimate them.

That is the book.

Why You Should Care

A single change in haemoglobin shows what parasites can do to a species that imagines itself in charge of its own body. In many malaria-exposed populations, inheriting one copy of the sickle haemoglobin variant reduces the risk of severe *Plasmodium falciparum* malaria. Inheriting two copies can cause sickle-cell disease. The same variant has been maintained because a parasite altered the balance of survival and reproduction strongly enough to outweigh a terrible genetic cost in some descendants.

That is more than disease. It is evolution written into human blood. The pressure also runs in the other direction: modern genomic work shows that the protection associated with sickle haemoglobin varies partly with parasite genotype. Host and parasite are not fixed opponents. Each changes the environment in which the other evolves.

The immediate burden is still immense. The World Health Organization estimated 282 million malaria cases and 610,000 deaths in 2024. It estimated that at least 253.7 million people required preventive treatment for schistosomiasis in the same year. Those figures are not a map of biological destiny. They are produced by parasites interacting with water systems, housing, poverty, vectors, land use, conflict, public-health capacity and access to care. Biology establishes possible routes. Infrastructure and politics determine how many stay open.

But human disease is only one reason to care. A broad comparative study identified at least 223 independent origins of parasitism among animals. Parasitic flowering plants have arisen in about a dozen independent lineages and include roughly 4,750 species. Fungi parasitise plants, animals and other fungi. Most of these organisms will never infect a person. They matter because parasitism is one of life's recurrent economic strategies: let another organism gather the resources, then evolve a way to tap the flow.

That breadth prevents the subject from becoming tropical medicine with extra animals attached. The same logic appears in forests, farms, ponds, oceans and soils, at scales ranging from a single cell to a food web.

That strategy exposes mechanisms that are otherwise easy to miss. A tapeworm makes the value of a digestive tract obvious by showing what happens when digestion is outsourced. A parasitic plant shows that roots and leaves are negotiable if another plant supplies their function. A trypanosome makes immune recognition legible by surviving through antigenic change. A trematode turns a food web into a transport network. Parasites are explanatory stress tests for bodies and ecosystems.

They also teach the importance of routes. Guinea worm disease has no

vaccine and no curative drug that made eradication possible. The campaign attacked the life cycle: contaminated drinking water, infected copepods, contact between an emerging worm and water, case detection and later animal reservoirs. Annual reported human cases fell from an estimated 3.5 million in 1986 to ten in 2025. The remaining animal infections show why the final few cases can be harder than the first few million.

There is a final reason to care, and it corrects the instinct to treat parasite as a moral category. Some parasitic relationships cause severe human, agricultural or conservation harm and should be controlled or, where feasible, eradicated. Others are native components of ecosystems. A host-specific parasite can be rarer than its host and can disappear with it. Removing parasites indiscriminately would not cleanse nature. It would alter food webs, competition and evolutionary history.

By the end of this hour, you should be able to see a parasite as a biological strategy rather than a monster, read a life cycle as a chain of dependencies, separate infection from disease and transmission, judge mind-control claims by evidence rather than theatre, and understand why hosts themselves bear the fingerprints of the organisms that exploit them. Parasites begin by borrowing life. Over evolutionary time, they help reshape it.

The Core Ideas

1. Parasitism Is a Relationship, Not a Lineage

A wolf is a wolf whether it is hungry, asleep or chasing prey. Parasite is different. The label describes what an organism is doing in relation to another living organism. A parasite lives in or on a host, takes resources through that association and reduces the host's fitness on average. Change the relationship and the ecological label can change with it.

That is why there is no parasite branch on the tree of life. A fungus may decompose dead wood, invade living tissue or exchange nutrients with plant roots depending on the lineage and circumstances. Among animals, parasitism has evolved repeatedly in flatworms, roundworms, insects, mites,

crustaceans, molluscs and other groups. A 2016 synthesis identified at least 223 independent animal origins across fifteen phyla. Flowering plants have evolved parasitism in about twelve separate lineages, producing roughly 4,750 known species. Protists include *Plasmodium*, trypanosomes and *Toxoplasma*, alongside an enormous diversity of parasites that cause no human disease.

The boundary is useful rather than absolute. Ticks feed briefly and leave, so some ecologists place them near micropredators. Parasitoid wasps develop in or on one host and usually kill it, putting them between parasites and predators. Brood parasites such as cuckoos exploit parental care rather than tissue. Mutualisms can slide towards exploitation when one partner takes more than it returns. Biological relationships form continua that classification tidies after the fact.

What makes parasitism recur is the value of living matter. A host has already concentrated nutrients, maintained water balance, built tissues, found energy and moved into a suitable environment. To another organism, that body can be a rich patch of habitat. Blood is transport fluid full of nutrients. Intestinal contents are pre-processed food. Plant xylem and phloem move water, minerals and sugars. A cell maintains chemical gradients that an intracellular parasite can exploit.

The host is not passive ground. It detects, grooms, walls off, poisons, starves, encapsulates or kills intruders. Plants deploy chemical and structural defences. Vertebrates add complex innate and adaptive immunity. Insects can melanise invaders. Snails and fish mount their own responses. The resource therefore fights back.

This creates the defining asymmetry. A parasite often depends heavily on a host, but the host can survive without that particular parasite. The parasite gains by specialising; the host gains by preventing or tolerating exploitation. From that tension come host specificity, elaborate life cycles, immune evasion, manipulation, virulence and coevolution.

The useful starting question is therefore not what sort of creature is this

parasite? It is what living system has it become dependent on, what resource does it extract, and what does that dependence force it to do?

2. Dependence Rewards Specialisation, Reduction and Precision

Parasites often look anatomically odd because independence is expensive. If the host reliably supplies a function, natural selection can favour losing the machinery that once performed it.

Tapeworms are the clearest example. Adult cestodes live in a nutrient-rich intestine and absorb food across their outer surface. They do not need a digestive tract because the host has already broken the meal down. What they retain and elaborate are the structures that matter inside that habitat: attachment, an absorptive surface and enormous reproductive capacity. Reduction is not primitiveness. It is specialisation after outsourcing.

Parasitic plants show the same logic in a different kingdom. Hemiparasites such as many mistletoes still photosynthesise but tap hosts for water and minerals. Holoparasites may obtain carbon as well and can lose much of the ordinary machinery of independent plant life. Their decisive organ is the haustorium, which attaches to host tissue and establishes a physiological bridge to vascular supplies. The parasite has not stopped being a plant. It has reorganised its priorities around another plant's plumbing.

Fungi can specialise just as sharply. Rusts and mildews form feeding structures in living plant tissue while avoiding rapid destruction of the cells on which they depend. Entomopathogenic fungi must recognise an insect cuticle, adhere, germinate, penetrate and then proliferate in an environment unlike the leaf or soil from which the spore arrived. Each transition calls for a different set of genes and structures.

They also matter wherever humans depend on other species. Crop plants can be exploited by parasitic flowering plants, nematodes, fungi and protists. Livestock carry worms, ticks and single-celled parasites that divert energy, damage tissue or reduce reproduction. Wildlife management can fail when an apparently healthy population is being shaped by an infection that

changes survival or breeding. None of this requires treating every pathogen as a parasite for the purposes of this book. The useful point is narrower: agriculture and conservation operate on living systems, and living systems contain exploiters. A field can therefore be thought of as crops plus soil, weather and competitors, but also as hosts connected to parasites whose own life cycles may involve seeds, vectors, alternate plant hosts or persistent stages.

This changes what counts as control. Killing the organism found on the damaged host may be insufficient if the next stage is in soil, a vector or another species. Removing an alternate host may work biologically and fail ecologically if it causes greater damage elsewhere. Breeding resistance can suppress disease and then select parasite genotypes able to overcome it. Parasites turn management into an evolutionary problem because the target population responds to the pressure placed on it.

Dependency also changes which traits are worth keeping. Structures that help a free-living ancestor find food, disperse independently or digest a meal can become liabilities when the host performs those jobs more reliably. What survives is the machinery needed to locate, attach, extract, reproduce and move to the next host.

Specialisation creates host specificity. Receptors, surface chemistry, immune defences, body temperature, diet, behaviour and associated microbes can make one host permissive and a close relative useless. Some parasites remain generalists, but many occupy an evolutionary corridor narrower than their apparent opportunity. A seed of a parasitic plant can land on the wrong host. A trematode larva can encounter the wrong snail. A malaria parasite can enter a mosquito species in which development stalls. Contact is not compatibility.

Dependency also encourages stage specialisation. One form penetrates; another feeds; another reproduces; another survives outside; another disperses. The same genome can build bodies so different that early parasitologists sometimes named separate life stages as separate species. A complex parasite is often several ecological specialists connected in time.

This is the first major cost of freeloading. The more completely a lineage is adapted to a host, the more dangerous host loss becomes. A parasite restricted to one endangered species can vanish with it. A lineage dependent on several hosts can be broken by the loss of any compulsory link. Specialisation can make exploitation efficient and the lineage fragile at the same time.

The apparent simplicity of parasites is therefore deceptive. They may carry less general-purpose anatomy while requiring exquisite precision about where they are, which stage they are in and what their host is doing. Independence uses spare capacity. Dependence replaces it with fit.

3. Life Cycles Turn Ecology into Transport

A parasite can live comfortably inside a host and still fail completely. If its descendants cannot leave, survive the outside interval and enter another suitable host, the lineage ends. Life cycles are the transport systems that solve this problem.

Some are direct. Head lice hatch, feed and reproduce on one human host, with movement between people carrying the lineage onward. Many intestinal worms release eggs or larvae that pass into the environment and later infect another host of the same kind. Even these apparently straightforward routes require stages built for incompatible jobs: feeding inside tissue, enduring outside it, finding a host and establishing again.

Other cycles divide the jobs among species. In parasitology, the definitive host is where sexual reproduction occurs, not the host that appears most important to us. The intermediate host carries a developmental or asexual stage. A vector transports the parasite and may also provide an essential site of development. The malaria parasite reproduces asexually in humans and sexually in *Anopheles* mosquitoes, making the mosquito the definitive host. Human size, suffering and narrative attention do not alter the definition.

A biological vector is more than a contaminated syringe. The parasite must survive the vector's gut, cross tissues, develop at the available temperature

and reach the organ from which it can leave. Mosquito immunity and physiology filter which parasite genotypes complete that journey. Time matters as well: if development takes longer than the vector lives, transmission fails. Vector competence is therefore a property of a particular parasite-vector-environment combination, not a permanent label attached to every mosquito or tick species.

Trematode worms turn host sequence into an art form. Many use a snail as a first intermediate host, multiplying there before moving into a second host or directly into a vertebrate. If an infected fish or crustacean must be eaten by a bird or mammal, the route is trophic transmission. The parasite has attached its journey to a food chain. Tapeworm larvae can wait in prey tissue until a predator supplies the gut in which the adult can mature. The prey is a vehicle with a timetable.

Complexity can be advantageous because one host is good for growth and another for reproduction. A small intermediate host may be abundant and easy to enter. A larger definitive host may live longer, move farther or offer mates for sexual reproduction. Different stages can specialise in penetration, multiplication, persistence or dispersal rather than forcing one body plan to do everything badly.

There are costs. Every additional host is another point of failure. The hosts must overlap in place and time. A stage that matures before the next host arrives is wasted. Parasites compensate with huge reproductive output, long-lived resistant stages, host specificity, behavioural cues or manipulation. Many eggs never complete the route. The spectacular fertility of worms is less extravagance than insurance against an itinerary with terrible connections.

Guinea worm shows both the elegance and vulnerability of a cycle. People or other animals drink water containing infected copepods, small crustaceans often called water fleas. Larvae are released, mature in the body and, roughly a year later, a fertilised female emerges through a painful skin lesion. Contact with water releases larvae that infect copepods and reset the route. Filtering drinking water, preventing infected people and

animals from entering water, treating ponds and finding cases quickly can break the chain without killing the adult worm with a drug.

A life cycle diagram can look like decorative complexity until each arrow is treated as a survival problem. Then it becomes a map of dependencies. Every arrow is also a possible intervention.

Cycles also determine where genetic mixing occurs. Sexual reproduction in a definitive host can bring together lineages acquired from several intermediate hosts, creating new combinations on which selection can act. Bottlenecks between stages can do the opposite, allowing only a few genotypes through. The same itinerary therefore governs population genetics as well as anatomy. To know where a parasite evolves, one must know where it meets, reproduces and is reduced to a small travelling sample.

4. The Host Is Defended Territory

A host is attractive because it maintains itself. The same maintenance system can make it hostile. Skin blocks entry. Mucus traps invaders. Stomach acid destroys many swallowed organisms. Plants thicken cell walls and produce toxic chemicals. Animals groom, avoid contaminated food, abandon infected nests and alter social contact. Once a parasite crosses the outer border, immune recognition creates another set of walls.

Hosts can resist or tolerate. Resistance reduces parasite numbers through exclusion, killing or removal. Tolerance limits damage without necessarily reducing the burden. Both can improve host fitness, but they exert different pressure on the parasite. Strong resistance rewards evasion. Tolerance may allow high burdens while reducing the selective benefit of escalating exploitation. What looks like a weak defence may be a strategy of damage control.

Parasites answer with location, disguise, change and persuasion. Some live inside host cells, hiding from parts of the immune system while exposing themselves to others. Some coat themselves in host molecules or occupy sites with limited immune access. African trypanosomes cover themselves

in a dense layer of variant surface glycoprotein. Antibodies rise against the current coat and parasite numbers fall. A minority has switched the expressed variant, survives and expands, producing repeated waves of infection. The defence learns, but the target changes.

Helminths often face a different problem. They are too large to hide inside a cell, and long residence is valuable. Many release molecules that alter host immunity. The mouse intestinal worm *Heligmosomoides polygyrus* produces a structurally distinct mimic of the host signalling molecule TGF-beta. The mimic can induce regulatory T cells, which restrain immune attack. This is molecular imitation with a practical result: the intruder persuades part of the security system to lower the alarm.

The host response can become costly in its own right. Schistosome eggs trapped in tissues prompt granulomas, organised immune structures that wall off the eggs but can also produce fibrosis and organ damage. Fever can inhibit some parasites but taxes the host. Inflammation recruits weapons that harm surrounding tissue. Immunity is therefore an allocation problem, not a free weapon. Too little response permits exploitation. Too much can destroy what it is meant to protect.

Parasites have helped shape the complexity of immune systems because recognition cannot be solved once. A defence that targets a common parasite genotype makes rarer variants more valuable. A parasite that suppresses one pathway creates pressure for backup pathways or different forms of recognition. Hosts also face many parasites at once, so a response effective against one may worsen another or damage beneficial microbes. The system is layered because the threat is diverse and moving.

This is why elimination from one body does not settle the evolutionary contest. The parasite's descendants encounter other genotypes, other immune histories and other environments. Defence changes the habitat. The parasite then evolves inside the changed habitat.

Immune memory adds time to that habitat. A first infection can make later encounters harder, but protection may be incomplete, stage-specific or

short-lived. Parasites that change antigens, occupy long-lived tissue stages or suppress memory can reopen a route that appeared closed. Hosts, meanwhile, carry immune histories shaped by previous infections, nutrition, age and coinfection. Two genetically similar bodies can therefore present different environments to the same parasite.

5. Virulence Is a Consequence of the Route

A parasite's success is counted in new infections, not in symptoms. Pain, fever, anaemia, diarrhoea, tissue damage and death matter to the parasite only through their effect on survival and transmission. They may help, hinder or have little connection to the route out.

Three terms need separating. Infection means that a parasite has established. Disease is the impaired function experienced by the host. Virulence is the degree of harm associated with infection. One can occur without the others lining up neatly. A person may carry a parasite with few symptoms. A small parasite burden may provoke severe immune damage. A parasite can reproduce heavily while leaving its host mobile, or render the host incapacitated while gaining no extra route to the next body.

The old intuition says natural selection should make parasites gentler because killing the host destroys their home. That can happen, especially when transmission depends on prolonged contact, parental transfer or host reproduction. It is not a rule. If greater replication produces more transmissible stages but also harms the host, selection can favour an intermediate balance. In monarch butterflies infected by the protozoan *Ophryocystis elektroscirrha*, strains that produced more spores also imposed greater costs on their hosts, while damage that prevented successful flight and survival reduced onward transmission. The evolutionary optimum lay in the relationship between both effects, not at minimum harm.

Change the route and the balance changes. A parasite carried by a biting vector may still spread from a host too ill to walk. A durable stage shed into soil or water can outlast the body that produced it. A parasite that needs its

host to be eaten may benefit from altered movement or conspicuous behaviour. One transmitted from parent to offspring has a stronger interest in host reproduction. Multiple strains sharing a host may compete so aggressively that all damage their common vehicle. There is no universal endpoint called benign coexistence.

Much harm is also indirect. Schistosome adults can persist in blood vessels for years, while many chronic injuries arise when eggs become trapped in tissue and the immune system builds inflammatory reactions around them. Malaria symptoms and severe disease emerge from parasite multiplication, destruction and alteration of red blood cells, obstruction of small vessels in *Plasmodium falciparum* infection, and the host response. The parasite causes the situation, but the route from presence to damage passes through host biology.

This matters for prediction. A change that reduces symptoms need not reduce transmission. A treatment that clears severe stages may leave transmissible stages. A vaccine or drug can alter which parasite variants succeed. Crowding, immunity, host age, nutrition and coinfection can change the same parasite's apparent virulence. Asking whether a parasite is deadly is therefore weaker than asking which stage causes harm, how that harm affects transmission and what ecological route selection is rewarding.

Transmission is also uneven among hosts. Parasite burdens commonly cluster, with many parasites concentrated in a minority of individuals because exposure, immunity, age, behaviour and chance differ. Those heavily infected hosts can contribute a disproportionate share of onward transmission. Population averages then hide the mechanism. Ten hosts carrying one worm each are not epidemiologically equivalent to one host carrying ten and nine carrying none, even when the mean burden matches.

The parasite's central problem is not how to defeat one host. It is how to turn one host into the next.

6. Parasites Can Rewire Hosts and Food Webs

Parasitism happens inside ecology, so its effects rarely stop at the infected individual. A parasite can alter movement, appetite, reproduction, competitive ability and vulnerability to predators. Those changes can be incidental damage, host defence or parasite adaptation. Distinguishing among them is one of the most important disciplines in the subject.

The strongest cases of adaptive manipulation connect a changed host trait to the parasite's next transmission step. The trematode *Euhaplorchis californiensis* develops in California killifish and reaches sexual maturity in fish-eating birds. Infected fish perform more conspicuous movements and are eaten by birds at higher rates than uninfected fish. Later work found associated changes in brain monoamine systems. The altered behaviour appears at the point where predation is useful to the parasite, which makes the transmission argument unusually strong.

The fungus *Ophiocordyceps* produces a more theatrical effect in ants. Infected ants leave the colony, climb vegetation and bite into a position favourable for fungal development before dying. Three-dimensional imaging found fungal cells forming extensive networks through the body and around muscle fibres while the brain remained uninvaded during the manipulated stage. That finding weakens the cartoon of a fungus sitting in the brain and issuing commands. Altered muscle function, metabolism, chemical signalling and intact host neural circuitry may all contribute.

Toxoplasma gondii is a useful warning. Some experiments report reduced rodent aversion to cat odour, and sometimes attraction, which could favour transmission to cats, the definitive hosts. Results vary. Direct demonstrations that these changes increase predation by cats are much thinner than the popular story. Claims about human personality or psychiatric effects add further causal gaps. A striking phenotype is not enough. Adaptive manipulation requires stage, mechanism and fitness benefit to line up.

At community scale, parasites add relationships that older food webs often

omitted. In several highly resolved webs, parasite-host links outnumbered predator-prey links on average. Predators also consume parasites inside their prey, creating further links that may kill the parasite or deliver it to the correct final host. In three studied estuaries, total parasite biomass exceeded top-predator biomass. That is a result from those systems, not a planetary law, but it demonstrates what disappears when organisms inside other organisms are ignored.

Parasites can regulate abundance as well. In British red grouse, experimental reduction of the nematode *Trichostrongylus tenuis* prevented expected population crashes, showing that the worm helped drive the cycle rather than merely tracking it. A parasite can also sterilise hosts, weaken a dominant competitor, make prey easier to catch or alter grazing and movement.

This is what the subtitle means by shape everything. Parasites can modify bodies, relationships and population dynamics without becoming the largest organisms in the system. Their influence comes from occupying the connections through which energy, reproduction and transmission already flow.

7. Coevolution Leaves Parasites Inside the Host's Design

A parasite turns a host into an environment that evolves. A host turns a parasite into a pressure that evolves. Each side changes the conditions faced by the other, so adaptation can expire as soon as it becomes common.

This is coevolution. It need not look like a steady climb towards stronger weapons. A common parasite genotype favours host resistance variants that recognise it. As those host variants spread, parasite genotypes that evade them gain an advantage. Rare host types can then become valuable precisely because they are rare. Frequencies cycle, different places follow different paths, and victory in one generation can create vulnerability in the next.

The Red Queen image captures the motion: both sides run to remain in roughly the same relative position. Experiments and field studies have found this negative frequency-dependent pattern in several host-parasite systems. It also supplies one leading explanation for sexual reproduction. Sex breaks and recombines inherited combinations, producing offspring that differ from their parents and siblings. Against a parasite adapted to common host genotypes, novelty can be protective. Work on New Zealand freshwater snails and their sterilising trematodes helped make the case. Parasites are not the complete explanation for sex, whose costs and persistence have several proposed causes, but they can reward genetic reshuffling.

Human blood carries a sharper example. The sickle haemoglobin variant changes red blood cells in ways that reduce the risk of severe *Plasmodium falciparum* malaria in people with one copy. Where malaria has imposed intense mortality, that benefit helped maintain the variant despite the severe cost of sickle-cell disease in many people who inherit two copies. This is balancing selection with a human price. Recent work has also linked the strength of sickle-associated protection to parasite genotypes, evidence consistent with *Plasmodium* evolution within the genetic landscape created by its hosts.

The same logic reaches immune genes, behaviour and life history. Hosts may evolve faster maturation when infection threatens later reproduction. They may choose mates whose immune variants differ from their own, although specific claims require careful testing. Parasites evolve host specificity, timing, antigenic change and strategies for tolerating or redirecting defence. Neither side designs a perfect solution because every solution changes the game and carries costs elsewhere.

Coevolution can also produce dependence and restraint. A parasite that relies on a scarce host may evolve lower exploitation. A host may tolerate infection when resistance would be more damaging. Long associations can settle into low harm, then shift when a new host, route or environment changes the incentives. Novel spillovers are often dangerous because parasite and host enter without the adjustments produced by shared history,

though even that pattern has exceptions.

Core Idea 1 began with a rich living habitat. Its richness invited invasion. Defence then made the habitat harder to use, rewarding specialisation, disguise and change. Those countermeasures rewarded new defences, genetic diversity and altered behaviour. The feature that made hosts exploitable also made them evolutionarily unfinished.

No organism occupies its body alone in evolutionary terms. Its shape includes the enemies that failed to enter, the ones it learned to tolerate and the ones still changing beside it.

The contest is also geographically uneven. A parasite may be locally adapted to common host genotypes in one valley and poorly matched a few kilometres away. Migration mixes those histories. Climate, land use and host movement change who meets whom, shifting selection again. Coevolution is therefore a mosaic rather than one global race, with hot spots of intense reciprocal change, cold spots where interaction is weak, and continual movement between them.

How It Actually Works

One parasite, two hosts

A female *Anopheles* mosquito does not place a malaria parasite neatly into a blood vessel. She probes the skin for blood and releases saliva that helps the meal flow. Slender *Plasmodium* sporozoites leave the salivary glands with that fluid, move through skin and circulation, and reach the liver. The infection begins as a transport event measured in micrometres.

Inside liver cells, the parasites multiply without producing the familiar cycles of fever. The resulting merozoites enter the bloodstream and invade red blood cells. There they feed, develop and divide. Cells rupture, more merozoites spread, and some parasites take a different developmental route, becoming male or female gametocytes. Those forms do not complete sex inside the human. They wait to be taken by another mosquito.

A mosquito feeding on the infected person ingests gametocytes. In its gut they form gametes, fuse and pass through further stages before producing new sporozoites that migrate to the salivary glands. The loop is ready to begin again. The human supplies liver cells, red blood cells and a large site for asexual multiplication. The mosquito supplies movement between humans and the setting for sexual reproduction. Remove either host and this route stops.

The sequence shows why a parasite cannot be understood from one snapshot. A blood smear reveals one stage. A liver biopsy would reveal another. A mosquito gut contains forms absent from both. What looks like several organisms can be one genome moving through a schedule of specialised bodies.

Scale changes with the stage. A few sporozoites begin the human infection, liver multiplication expands the population, repeated blood cycles create immense numbers, and only a fraction become gametocytes capable of infecting a mosquito. Selection acts at each transition, but success is decided by the whole circuit. A variant that grows quickly in blood yet never reaches the mosquito is an evolutionary failure.

Find the right surface

Every parasite begins by reaching the right surface. That may be the skin, gut, lung, root, leaf or body cavity of an intermediate host. Most encounters fail. The wrong host lacks the required receptor, chemistry, temperature, behaviour or onward connection. Successful infection is a narrow match hidden inside a large number of dead ends.

Schistosome larvae called cercariae leave freshwater snails and penetrate human skin during contact with infested water. They discard their forked tails, alter their outer surface and enter circulation. Hookworms can also cross skin, while many other helminths enter as swallowed eggs or larvae. Ticks cut into skin and anchor mouthparts long enough to feed. Lice stay near the surface. Each route exposes the parasite to different barriers and determines which interventions can interrupt contact.

Parasitic plants solve the same problem without blood. A seed must germinate close enough to a suitable host, detect chemical or physical cues and build a haustorium. This specialised organ attaches to and penetrates host tissue, then connects with vascular conduits. Some species draw mainly water and minerals while continuing to photosynthesise. Others extract carbon as well. The host's xylem and phloem become supply lines, but establishing the connection requires developmental precision that an ordinary root does not possess.

Fungal parasites enter through wounds, natural openings, direct penetration or ingestion. Insect-pathogenic fungi can attach spores to the cuticle, germinate and cross it with mechanical pressure and enzymes. Plant rust fungi build feeding structures within living plant tissue while keeping host cells alive. The goal is access without immediate collapse of the resource.

Entry leaves evidence. The parasite may shed a coat, switch gene expression, change metabolism or produce a structure used nowhere else. These transitions are not decorative names in a diagram. They are the engineering required to move from an exposed traveller to a resident.

Exposure is therefore more than proximity. Dose, duration and route matter. Swallowing a stage adapted to penetrate skin may do nothing. A parasite placed directly into blood may bypass barriers it could never cross naturally. Laboratory infections must reproduce the relevant route or risk describing an artificial organism-host relationship. In the field, small changes in footwear, water contact, housing or vector access can alter entry without changing the parasite itself.

Change body when the job changes

Once inside, growth and reproduction may occur in a body unlike the infective stage. Parasites often separate tasks more sharply than free-living animals because each host presents a different environment.

A tapeworm illustrates reduction. The adult attaches to an intestine with a scolex and absorbs nutrients across its outer surface. It has no digestive tract because digestion has been outsourced to the host. Behind the scolex,

repeated segments mature and produce reproductive organs. Eggs leave with host waste. In many species, grazing animals or other intermediate hosts ingest those eggs, larvae cross the gut and encyst in tissues, and the adult stage appears only if a suitable predator eats that tissue. The tapeworm's body is a conveyor built around attachment, absorption and reproduction.

Trematodes can use multiplication to compensate for improbable travel. A larva entering the correct snail may produce many descendants asexually, turning one successful entry into a large wave of stages released into water. Some species redirect host resources so strongly that the snail's reproduction is reduced or stopped. The parasite has converted a host from competitor for energy into a production site. Later stages may encyst in fish, crabs, vegetation or tissue and wait for a vertebrate to complete the route.

The word adult can therefore mislead. The sexually mature stage may be small and brief while an asexual stage generates most of the numbers. A larva can be the ecologically dominant form. Different hosts may see different consequences from the same parasite. A fish carrying encysted larvae may be harmed little until altered behaviour raises predation risk. A bird eating the fish supplies the intestine where adults mate, yet may carry them with modest damage.

Parasites also time development to host biology. Some wait for hormones associated with pregnancy, moulting or migration. Some remain dormant until a vector feeds or an intermediate host is eaten. The dormant stage is not inactivity in an evolutionary sense. It is a bet that waiting costs less than developing in the wrong place.

Parasitic plants show a quieter version of the same task-switching. A germinating dodder seedling has only a short reserve with which to find a host. Once contact is made, coiling and haustorial development convert a searching seedling into a vascular parasite. Flowering and seed production then hand dispersal back to ordinary plant machinery. The life cycle is less theatrical than a trematode's but follows the same rule: build different structures when the ecological job changes.

Stage changes can also partition conflict within the parasite population. Asexual multiplication rewards rapid expansion, while sexual stages depend on meeting compatible gametes and reaching the definitive host. One stage may monopolise host resources that later stages need. Developmental switches regulate that conflict, allocating some descendants to immediate growth and others to transmission. The life cycle is therefore an economy of bodies, with reproduction deferred or accelerated according to where future value lies.

Feed inside a defended system

Residence brings a new constraint: the host can detect change. Parasites must acquire resources while keeping enough tissue functional and avoiding elimination.

Plasmodium falciparum spends much of its human blood stage inside red blood cells, which lack a nucleus and much of the machinery used by other cells to signal infection. The parasite still faces splenic clearance, antibodies and other immune pressures. It remodels the infected cell and places adhesive proteins on its surface, allowing many mature infected cells to bind vessel walls rather than circulate through the spleen. That evasion contributes to severe disease when altered cells accumulate in small vessels.

African trypanosomes remain extracellular in blood and tissue fluids. Their dense surface coat presents one main antigenic identity at a time. As antibodies clear parasites bearing that coat, switched variants escape. Population waves follow, each provoking another response. The strategy is expensive: a large genomic archive and controlled switching machinery are devoted to staying one antigen ahead.

Large worms cannot rely on one trick. Schistosomes enter as larvae and mature into paired adults in blood vessels. Their surface is continually renewed and interacts closely with host molecules. Eggs are built to leave, but many become trapped. The host walls them off, containing dangerous secretions while creating chronic inflammation. The same immune response

protects tissue and scars it.

Long-lived helminths can alter the tone of immunity rather than evade every attack. Secreted proteins, glycans and vesicles affect host signalling. *Heligmosomoides polygyrus* in mice produces a TGF-beta mimic that activates regulatory pathways. Other helminths use different molecules and targets. The general pattern is convergence on the host's own brakes.

None of this means the host is helpless. Parasite loads often fall with acquired immunity. Resistant genotypes spread. Grooming and avoidance reduce exposure. Tolerance keeps hosts alive and reproductive despite infection. The stable-looking infection is an active balance of parasite growth, host control, tissue repair and transmission. Remove one pressure and the equilibrium can move quickly.

Inside one host, parasites also compete and cooperate. Related stages may share molecules that alter immunity, while unrelated strains compete for cells, nutrients and access to vectors. High density can trigger developmental changes or reduce each parasite's growth. A host is not one parasite facing one immune system. It is an ecosystem in miniature, and within-host ecology can determine both disease and which genotypes reach the next host.

Leave at the right moment

A parasite that persists but never exits is an evolutionary cul-de-sac. Exit shapes pathology, timing and behaviour as strongly as entry.

Intestinal parasites can release eggs, cysts or larvae into faeces. Urinary schistosome eggs pass through the bladder wall and leave in urine. Respiratory parasites may exploit coughing. Blood parasites wait for vectors. Skin-dwelling stages can move through contact. Fungal spores use air, rain splash or the body of a manipulated insect. Parasitic plants often use ordinary flowers, fruits and seeds, keeping the host connection for nutrition while relying on wind or animals for dispersal.

Routes through predation create especially sharp incentives. *Euhaplorchis*

trematodes need infected killifish to enter birds. Conspicuous swimming becomes useful near the mature transmission stage. Too early, and the parasite dies before becoming infective. Too late, and the opportunity is missed. Manipulation therefore depends on timing, not a permanent takeover.

Parasitoid wasps use a terminal exit. An egg is placed in or on another arthropod. The developing larva consumes host resources, sometimes delaying lethal damage until growth is nearly complete. Emergence kills the host or follows its death. The relationship begins like parasitism but ends like predation. The host is a nursery with one occupant scheduled to leave.

Guinea worm makes exit visible at human scale. The mature female moves towards the skin and emerges through a painful blister. Water contact triggers release of larvae. The burning sensation encourages immersion, which can serve the parasite's route, though relief-seeking need not be described as precise behavioural manipulation. The crucial fact is ecological: one infected limb entering a pond can connect a human body to copepods and future drinkers.

Each exit stage faces a hostile interval. Eggs need shells, cysts need resistance, spores need dispersal, and vector-borne stages need to remain available in blood or skin. Reproductive excess reflects attrition. Millions of descendants may be produced because only a few complete the circuit.

The exit creates a bottleneck that can reshape the population. A mosquito ingests only a sample of circulating gametocytes. A predator eats one infected prey item. A few eggs reach the right water. Chance can remove common variants and carry rare ones forward. What evolves between hosts is therefore influenced by sampling as well as by selection, especially when transmission events are scarce.

Reconstruct the hidden route

For much of medical history, observers could see recurring fevers without seeing the organism or its journey. The route became clear through a sequence of observations across species and stages.

In 1880 Alphonse Laveran saw pigmented parasites in the blood of people with malaria. That established a living agent but not transmission. In 1897 Ronald Ross traced related malaria parasites in birds through mosquitoes and found developing forms in mosquito tissues. Italian investigators including Giovanni Battista Grassi, Amico Bignami and Giuseppe Bastianelli then demonstrated transmission of human malaria by *Anopheles* mosquitoes in 1898. The complete picture required blood microscopy, experimental infection, mosquito dissection and the willingness to study non-human malaria when parts of the human cycle were inaccessible.

The liver stage remained unknown until 1948, when Henry Shortt and Cyril Garnham demonstrated pre-blood development in liver tissue. Dormant liver stages responsible for relapse in some malaria species were confirmed later. Each addition changed intervention. Blood stages explained symptoms and diagnosis. Mosquito stages made vector control intelligible. Liver stages created targets and explained why an apparently cleared infection could return.

Parasitology repeatedly advances by joining fragments. Eggs found in faeces reveal exit. Larvae in snails reveal an intermediate host. Adult worms in predators reveal trophic transfer. Molecular sequences connect forms that look unrelated. A life cycle is often discovered backwards, one host at a time.

Names have sometimes hidden the connection. Adult and larval stages found in different hosts were described as separate species because they looked unrelated. Experimental feeding, rearing and later DNA sequencing reunited them. Modern barcoding can match a larva in a snail to an adult in a bird, but only if reference sequences and specimens are reliable. The map still depends on natural history: knowing where to look, what eats what and when stages appear.

Break a dependency, then watch evolution

Control works best when it attacks a dependency the parasite cannot bypass cheaply. The weakest link may be entry, an intermediate host, a vector, a developmental stage or the social conditions that connect them.

Guinea worm is the cleanest demonstration. The cycle requires contaminated drinking water, infected copepods and later contact between an emerging worm and water. Cloth and pipe filters remove copepods. Safer water removes the encounter. Larvicide can reduce copepods in selected ponds. Detecting cases and keeping affected people and animals away from water prevents release. No vaccine is needed because the route offers several physical points of failure. By 2025, only ten human cases were provisionally reported worldwide.

The final cases reveal the limits of a human-only model. The same worm infects dogs and other animals. Hundreds of animal infections were still reported in 2025, and dogs eating fish or discarded fish entrails may help maintain transmission in some places. Eradication now depends on surveillance across species, water sources and behaviour. A programme that nearly solved the human cycle had to expand its map of the host system.

Schistosomiasis control faces a different network. Treatment reduces worm burden and disease, but people can be reinfected where freshwater snails, contaminated water and repeated contact persist. Safe water, sanitation, changes in exposure and selected snail-control measures address different arrows. Focusing on one stage can lower harm while leaving the cycle intact.

Malaria offers still more links: preventing mosquito bites, reducing vector survival, clearing human infections, protecting high-risk groups and limiting parasite development. Every pressure can provoke response. Mosquito populations evolve insecticide resistance. Parasite populations evolve drug resistance. Vectors may shift feeding time or location. Human behaviour changes when interventions are inconvenient or trust fails. The route is

biological and social, so durable control needs both.

Eradication is a stricter goal than control. It requires no continuing transmission anywhere and confidence that surveillance would detect a return. Parasites with animal reservoirs, durable environmental stages, silent infections or many vectors are poor candidates. Guinea worm has no vaccine, yet its narrow water-based route made eradication conceivable. The discovery of substantial animal transmission made the finish harder without erasing the logic: the more compulsory links a route has, the more opportunities control may have, but only if the map includes every host that matters.

Intervention therefore begins with a diagram and ends with evolution. Identify every host and stage. Find the link whose removal is feasible, safe and hard to replace. Measure what happens after pressure is applied. The parasite does not know it is being controlled. Populations carrying variants that survive the new conditions leave more descendants, and the system changes around the intervention.

Surveillance must therefore ask more than whether cases fell. It needs to detect altered species composition, new reservoirs, resistant variants, changing seasonality and shifts in where exposure occurs. A falling average can conceal a persistent pocket capable of reseeding transmission. The final phase of elimination is expensive because every remaining infection matters and because the system becomes hardest to observe when it is nearly gone.

How we know

Parasites preserve badly because many are small and soft-bodied. Their history is reconstructed from several records. Eggs and cysts survive in coprolites, latrines and mummified tissue. Amber can preserve arthropods and fungi. Bone lesions sometimes reveal chronic infection, though similar damage can have other causes. Ancient DNA and proteins can identify organisms that morphology alone cannot separate.

Living cycles are established through microscopy, dissection, molecular

matching, field observation and carefully controlled infection studies. Phylogenies show repeated origins of parasitism and connect radically different life stages. Food-web surveys expose interactions missed by ordinary species lists. Drug, vector and deworming interventions can function as experiments when designed and interpreted cautiously.

The weakest evidence often concerns intent-like claims. A behavioural change may be parasite adaptation, host defence or collateral damage. Demonstrating manipulation requires linking infection to a specific change and that change to greater parasite transmission. Human experiments are limited by ethics, and laboratory hosts may not reproduce field ecology. The broad mechanisms are secure. The exact number of parasite species, the oldest origins and many complete life cycles remain unknown.

What People Get Wrong

"Parasites are a small group of primitive worms"

Worms dominate the public image because they are visible, memorable and often large enough to remove dramatically. They are one part of a strategy scattered across the tree of life. Parasitic animals include insects, mites, crustaceans and several worm lineages. Single-celled eukaryotes include *Plasmodium*, trypanosomes and many parasites of wildlife. Fungi invade living hosts. Thousands of flowering plants draw resources through haustoria.

Primitive is wrong for a second reason. Reduction often follows specialisation and dependence. Tapeworms lost a gut after their ancestors entered an environment full of digested nutrients. Some parasitic plants have tiny leaves because the host supplies carbon. Such organisms can possess elaborate attachment, sensing, reproductive and immune-evasion systems while lacking structures that a free-living relative needs.

The correction matters because a worm-centred view makes parasitism look like one ancient oddity. Repeated independent origins show the opposite. Living bodies repeatedly create opportunities for exploitation, which helps

explain why parasitism has evolved so many times independently. The diversity of parasites follows the diversity of hosts, which means undiscovered hosts and poorly studied ecosystems almost certainly contain undiscovered parasitic lineages.

"A successful parasite makes its host as sick as possible"

Maximum sickness is useful only when it improves onward transmission. A parasite that immobilises a host may lose contact opportunities. One that kills before producing transmissible stages has destroyed its route. Natural selection therefore acts on the relation between replication, damage, duration and exit.

That relation varies. Vector-borne transmission can continue from a bedbound host. Durable eggs can survive after death. A trophically transmitted parasite may benefit when the host becomes easy prey. A parasite passed from parent to offspring depends more closely on host reproduction. Coinfections can reward faster exploitation because competing strains share the future cost.

The opposite slogan, that parasites always evolve towards harmlessness, fails for the same reason. Virulence can rise, fall or stabilise depending on ecology. It may also be produced partly by host immunity rather than parasite extraction alone.

Treating gentleness as evolution's destination creates bad predictions. The right question is which level of harm leaves the most successful descendants under the current route. That answer can change when vectors, treatment, host density or behaviour change, so virulence is a moving ecological property rather than a fixed personality.

"A complicated life cycle is bad design"

A cycle through snail, fish and bird looks absurd if every stage is expected to perform the same task. It makes more sense when the tasks are separated. One host may be abundant and ideal for multiplication. Another may provide growth. A predator may offer a long-lived intestine, mates and wide dispersal.

Complexity also arises from history. Evolution modifies available routes rather than planning from a blank page. A lineage that once used one host can add another when predation creates reliable transport. Later adaptation makes each stage dependent on the arrangement. The result can be highly effective and fragile at the same time.

Every extra host adds risk. Hosts must meet, stages must mature at the right time, and most descendants fail. Parasites answer with resistant stages, enormous reproductive output and precise cues. Complexity is therefore neither evidence of foresight nor evidence of stupidity. It is a trade: more specialised opportunities in exchange for more points of failure.

That trade is why life cycles are so useful for control. A route with several compulsory links offers several places to break it. It is also why interventions aimed at the wrong host can produce impressive local effort and little change in transmission.

"Parasites control minds like puppets"

Some parasites alter host behaviour in ways that increase transmission. The killifish and *Euhaplorchis* case has field evidence linking conspicuous behaviour to bird predation. *Ophiocordyceps* fungi coordinate striking changes in ants before spore release. These are serious evolutionary phenomena.

Puppet language converts them into a mechanism that the evidence rarely supports. The fungus need not issue commands to an ant's brain. Imaging shows extensive growth around muscle fibres while the brain remains uninvaded during the manipulated stage. Chemical signals, altered

metabolism, tissue damage and host circuitry may combine. Coordination can emerge without a miniature pilot.

Other famous claims are weaker. *Toxoplasma* infection has altered rodent responses to cat odour in some studies, but results vary and direct increases in cat predation are hard to demonstrate. Claims about human personality or psychiatric disease add further causal gaps.

A proper manipulation claim needs a changed trait, stage-specific timing, a plausible mechanism and evidence of greater parasite fitness. Strange behaviour alone proves infection has consequences. It does not prove adaptive control. The distinction protects the science from becoming a collection of cinematic anecdotes in which every damaged host is credited to parasite strategy.

"Parasites sit outside the food web"

A parasite eats living tissue, is eaten inside its host and can move when one host consumes another. It belongs in the food web several times over.

Leaving parasites out once made ecological diagrams cleaner and less accurate. Detailed studies found that parasite-host links could outnumber ordinary predator-prey links. Adding the predators that ingest parasites inside prey creates still more connections. Some of those meals kill the parasite. Others deliver it to the host where it reproduces.

Parasites can also change interaction strength. Infected prey may be easier to catch. Castrated hosts divert energy away from reproduction. A parasite can suppress a dominant competitor or drive host population cycles. Its effect can pass through species it never infects.

The correction matters because stability, energy flow and extinction risk depend on links. An organism hidden inside another body remains an ecological participant. Its links may be weaker or harder to measure than a kill by a predator, but invisibility is a sampling problem, not ecological absence.

"People in wealthy countries no longer have parasites"

Improved sanitation, safe water, vector control, food inspection, housing and access to treatment have removed or reduced many routes. They have not abolished parasitism.

Ticks transmit parasites in temperate landscapes. *Toxoplasma* circulates through cats, prey, meat and soil. *Giardia* can spread through contaminated water. Lice, pinworms and scabies mites still occur in wealthy countries. Travel, migration, changing land use, imported food, wildlife contact and immunosuppression create further routes.

The larger error is geographical morality. Heavy burdens of malaria, schistosomiasis and other neglected diseases are concentrated where ecological exposure meets weak infrastructure, conflict, poverty and limited care. That distribution does not mean parasites belong naturally to poor places. It means transmission routes have been left open and the people carrying the cost have had less power to close them.

Wealth changes exposure and control. It does not create a biological border. The useful comparison is therefore between routes left open and routes successfully closed, not between supposedly parasitic and parasite-free societies.

"Every parasite should be eradicated"

Eradication is an appropriate ambition for selected parasites causing severe human disease when transmission can be ended safely and globally. Guinea worm is a compelling target because the route is narrow, the suffering is clear and there is no ecological case for preserving human infection.

That judgement cannot be copied across all parasitic life. Most parasites do not infect humans. Native parasites regulate wildlife, connect food webs and carry distinct evolutionary history. Some are restricted to one endangered host and face coextinction. Removing them can alter competition or expose

relocated hosts to later infections without familiar immune experience.

Even disease control requires precision. Eliminating a parasite from one host species may fail if reservoirs remain. Killing vectors can damage other species. Broad treatment can select resistance. A campaign that reduces illness may be wiser than eradication when surveillance, biology or social conditions make zero transmission unattainable.

The choice is not sentiment against hygiene. It is a decision about a specific relationship. Ask who is harmed, whether transmission can be stopped, what other hosts are involved and what ecological effects removal will produce. Parasite is not a conservation verdict or a death sentence. It names the interaction that must be judged case by case, and the same scientific clarity is needed both to eradicate Guinea worm and to avoid erasing harmless native symbionts.

Use It

Draw the route before naming the cure

When a parasite appears, resist beginning with the name. Draw the route. What leaves the infected host? How long can that stage survive? What must touch, bite, eat or drink what? Is another species required? Where does sexual reproduction occur? Which host keeps the parasite circulating when human cases fall?

This lens turns a frightening organism into a system with dependencies. Guinea worm becomes contaminated water, copepods, ingestion, emergence and renewed water contact. Malaria becomes human blood stages, mosquito feeding, development in the mosquito and another bite. Schistosomiasis becomes eggs, freshwater, snails, skin contact and return of eggs to water.

The route also shows why one successful intervention may be insufficient. Treating infected people can reduce burden while unsafe water sustains reinfection. Killing one vector species may leave another. If the arrow

remains, the parasite can return.

Add time to the map. Some stages appear only in a season, mature after a fixed delay or depend on a vector surviving long enough. Rainfall can create breeding sites; migration can bring definitive hosts; temperature can speed or block development. A route drawn without timing may identify the correct species and still miss the period when transmission can be interrupted most efficiently.

Name the stage

The same parasite can behave like several different organisms during one life. A sporozoite enters. A feeding stage grows. A gametocyte waits for a mosquito. An egg survives outside. A larva seeks a snail. Calling them all the parasite can hide which form is present and what it can do.

Ask which stage causes symptoms, which stage is detected, which stage is killed by an intervention and which stage transmits. These are often different. A blood test can miss a parasite confined to tissue. A drug aimed at active growth may spare a dormant stage. An adult worm can be less damaging than eggs trapped in organs.

Stage thinking also disciplines dramatic claims. Behavioural manipulation should appear when transmission is possible, not at any convenient moment. A change that occurs before the parasite is infective may be collateral damage unless it improves later development.

Separate parasite damage from defence damage

Injury can come from direct feeding, destroyed cells, blocked vessels, toxins, physical pressure or stolen nutrients. It can also come from inflammation, fibrosis, fever and other host responses. Most disease combines several routes.

This distinction changes what should be measured. A falling parasite count may not produce immediate recovery if immune damage continues. Severe symptoms can occur with modest burdens when the host response is intense. Conversely, high burdens can persist with limited outward illness if

the host tolerates them.

The distinction also prevents a moralised model of immunity. A stronger response is not always better. Resistance lowers parasite abundance. Tolerance limits the cost of a given abundance. Both can be adaptive, and either can fail. When evaluating an infection, ask what the parasite is doing, what the host is doing in reply and which process is producing the observed harm.

Look for the missing host

A parasite found in one species may depend on another species that is absent from the immediate scene. The obvious host may be an intermediate station. The definitive host could be a predator, vector or scavenger. A reservoir may maintain infection without showing much disease.

This lens is useful beyond medicine. An infected fish can point towards a bird that eats it. A trematode in a bird can imply a snail stage in local water. A parasite disappearing from a restored habitat may reveal that one host in the cycle has not returned. An outbreak after wildlife movement may reflect a reservoir rather than repeated transmission among the sick animals being noticed.

Missing-host thinking also guards against blame. The species carrying the adult parasite may not be the species spreading the infective stage to people. Effective control follows the cycle, not the most visible victim.

Expect evolution after control

Every sustained intervention changes which parasite variants reproduce. Drugs favour resistance mutations or combinations that survive treatment. Insecticides favour resistant vectors. Vaccines can shift the value of antigens. Shortening infections can reward earlier transmission. Removing common genotypes can release rare ones.

This does not make control futile. It makes monitoring part of control. Use combinations where appropriate, preserve multiple barriers, measure treatment failure, watch vectors and update the model. The strongest

intervention is often one that attacks several independent dependencies rather than placing the whole programme on one molecule.

Evolution also occurs in hosts and behaviour. People change how they use nets, water or medicines. Vectors may feed outdoors or at different times. Domestic animals can become more important reservoirs as human transmission falls. A successful programme alters the ecology that made its first strategy successful.

Add the organisms inside the organisms

When looking at an ecosystem, ask what lives inside the visible species. Parasites add species, biomass, energy flow and links. They may regulate dominant hosts, alter predation, move through food chains or disappear with threatened hosts.

This lens changes conservation questions. A translocation does not move one species; it moves a host and some fraction of its associated community. Quarantine can protect the destination while stripping native symbionts. Reintroduction into parasite-free conditions may create short-term success and long-term vulnerability. None of these outcomes is automatic, which is why inventories and risk assessment matter.

It also changes ordinary observation. A distorted leaf, swollen snail, altered insect or unusual animal movement may be evidence of another organism's life cycle. The visible body is often shared space.

The limits

Parasitism is a biological relation, not a ready-made moral analogy. Calling a person, company or social group parasitic usually imports disgust while discarding the precision that makes the concept useful. Human institutions involve consent, law, power and choice in ways a tapeworm does not.

Evolutionary explanation is not justification. A trait can spread because it benefits parasite transmission and still cause suffering that humans should prevent. Natural does not mean desirable, and ecological importance does not require preserving infection in people.

The category also has blurred edges. Pathogenic microbes, parasitoids, brood parasites, micropredators and exploitative mutualists cross boundaries depending on the definition. Use the term to clarify a relationship, not to win an argument about labels.

Finally, this book is not a guide to diagnosis or treatment. Parasite symptoms overlap with many other conditions, tests have limits and therapies differ by species, stage, geography and patient. The transferable model is the route, host and stage. Clinical decisions require current professional evidence.

The one thing to keep

Keep the host-as-habitat reversal.

From inside, a body feels like an individual with a boundary. From outside, it is warm tissue, flowing nutrients, chemical gradients, shelter and transport. The parasite sees opportunity where the host sees self. That reversal explains why parasitism has evolved so many times and why defence can never be finished.

It also changes what counts as an organism's design. Skin is shaped partly by what tries to cross it. Immune variation reflects enemies that keep changing. Behaviour includes avoidance, grooming and social responses to infection. Sex may persist partly because parasites make genetic sameness costly. Population cycles and food webs carry hidden links. A haemoglobin variant can bear the signature of an ancient contest that remains active in living children.

The lesson is not that everything is secretly infected or that cooperation is false. Mutualism, competition, predation and parasitism coexist, and relationships can move along that range. The lesson is that life creates exploitable structure. Any system that captures energy, stores resources and maintains an interior becomes valuable to another lineage.

That pressure is permanent because neither side receives a final design. Hosts close routes. Parasites find others. Hosts recognise common

disguises. Rare disguises spread. Control succeeds, ecology shifts and selection begins from the new conditions.

To see a parasite clearly is to see two organisms and the route between them. Once that route is visible, the apparent freeloader becomes a force that has helped build the host, the ecosystem and the evolutionary world both inhabit.

Terms

Parasite. An organism that lives in or on a host, draws resources from the association and reduces host fitness on average. The term describes a relationship, not one evolutionary lineage, and its boundaries vary with the ecological definition being used.

Host. The organism that supplies a parasite with habitat, resources or transport. One parasite may use several hosts, and the most visibly harmed host need not be definitive, a reservoir or the main source of infection.

Ectoparasite. A parasite living mainly on the host's outer surface, such as a louse, flea or tick. Surface residence changes exposure, feeding, transmission and opportunities for grooming.

Endoparasite. A parasite living inside the host, whether in the gut, blood, tissue or cells. Internal residence provides stability while creating barriers to exit and immune evasion.

Obligate parasite. A lineage that must use a host to complete its normal life cycle. Its dependence often produces extreme specialisation and makes the required host a potential control point.

Facultative parasite. An organism capable of free living but able to exploit a host under suitable conditions. Facultative lifestyles show that parasitism can be an ecological option rather than permanent identity.

Definitive host. The host in which a parasite reaches sexual maturity or carries out sexual reproduction. It is defined by the life cycle, not by size, symptoms or human importance.

Intermediate host. A required host carrying larval development or asexual multiplication rather than sexual maturity. Several intermediate hosts may occur in sequence before the definitive host is reached.

Reservoir host. A species or population that maintains a parasite and can seed infection elsewhere. Reservoirs can sustain transmission even when disease in the target host becomes rare.

Vector. An organism that carries a parasite between hosts. Biological vectors also support development or reproduction; mechanical vectors transport infective material without an essential internal stage.

Parasitoid. Usually an insect whose larva develops in or on one host and eventually kills it. Parasitoids occupy the conceptual border between sustained parasitism and one-kill predation.

Helminth. A practical collective term for parasitic worms, mainly nematodes, cestodes and trematodes. These groups are not one close lineage, despite sharing elongated bodies and medical textbooks.

Nematode. A roundworm from a vast animal phylum containing free-living and parasitic species. Parasitic nematodes use direct or complex cycles and infect plants as well as animals.

Cestode. A tapeworm, usually with a scolex for attachment and a segmented reproductive body. Adults absorb nutrients through their surface and commonly rely on trophic transmission.

Trematode. A fluke, typically a flatworm with suckers and a life cycle involving molluscan and vertebrate hosts. Their stages can multiply, encyst, manipulate and cause disease differently.

Protist. A convenient, non-formal label for diverse eukaryotes that are not animals, plants or fungi. Many single-celled parasites, including trypanosomes and *Plasmodium*, fall within this broad category.

Apicomplexan. A group of mostly parasitic protists with specialised machinery for entering host cells. It includes *Plasmodium*, *Toxoplasma* and

coccidian parasites of many animal species.

Haustorium. A feeding and attachment organ produced by parasitic plants or some fungi. It penetrates host tissue and connects the parasite to water, minerals, carbon or cellular contents.

Trophic transmission. Passage to the next host when an infected organism is eaten. The route can favour encystment, altered host behaviour and maturation only inside the predator.

Zoonosis. An infection naturally transmitted between vertebrate animals and humans. Zoonotic cycles complicate control because removing human transmission may leave a wildlife or domestic-animal reservoir intact.

Life cycle. The ordered sequence of stages, hosts, reproduction and transmission through which a parasite lineage persists. Every compulsory transition is both an adaptation and a possible failure point, so the cycle is the basic map for explanation and control.

Prevalence. The proportion of hosts infected in a defined population at a stated time. Prevalence measures distribution, not how many parasites each infected host carries or how ill it is.

Intensity. The number of individual parasites in an infected host. Intensity can vary enormously among hosts and may predict damage or transmission better than infection status alone.

Virulence. The degree of harm associated with infection, often measured through reduced survival or reproduction. Virulence evolves through transmission ecology and can include damage caused by host defence.

Resistance. Host traits that prevent infection or reduce parasite burden. Resistance changes parasite abundance and can drive counter-adaptation, including antigenic change, immune suppression or altered host use.

Tolerance. Host traits that reduce fitness loss at a given parasite burden without necessarily killing the parasite. Tolerance can preserve function while exerting different evolutionary pressure from resistance.

Immune evasion. Any parasite strategy that avoids, redirects or survives host immune attack. Hiding inside cells, molecular mimicry, suppression and changing exposed antigens are common routes.

Antigenic variation. Programmed or selected change in molecules recognised by host immunity. African trypanosomes switch surface coats, allowing rare variants to expand after antibodies clear the previous form.

Host manipulation. A parasite-induced change in host phenotype that increases parasite fitness. Demonstrating it requires more than altered behaviour: timing, mechanism and transmission benefit must align.

Coevolution. Reciprocal evolutionary change between interacting lineages. Host defence alters parasite selection, parasite countermeasures alter host selection, and common genotypes can lose advantage through Red Queen dynamics.

Go Deeper

The inviting tour. Carl Zimmer, *Parasite Rex: Inside the Bizarre World of Nature's Most Dangerous Creatures* (Free Press, 2000). Zimmer moves from individual life cycles to immune evasion, manipulation and ecosystem effects without turning the subject into a catalogue of horrors. The science has advanced since publication, and some famous manipulation stories now need more caution than popular accounts once gave them. It remains an unusually good next step for a reader who wants strangeness, research stories and conceptual scale in energetic narrative form. Read the later scientific claims alongside current sources, but keep Zimmer's gift for making an invisible life cycle feel inhabited.

The broad synthesis. Claude Combes, *Parasitism: The Ecology and Evolution of Intimate Interactions*, translated by Isaure de Buron and Vincent A. Connors (University of Chicago Press, 2001). Combes treats hosts as environments and follows parasitism from entry and life cycles through genetics, communities and coevolution. It is long, systematic and richer in examples than a standard textbook. Read it when the central model in this

book feels useful enough to deserve a full scholarly architecture. Its age shows in some examples and terminology, yet the movement from organism to community remains unusually coherent.

The behaviour test. Janice Moore, *Parasites and the Behavior of Animals* (Oxford University Press, 2002). Moore examines when altered behaviour supports adaptive manipulation and when pathology, host response or weak evidence offers a better explanation. The book is older than much molecular work on the subject, but its sceptical framework remains valuable. Read it to replace the phrase mind control with better questions about phenotype, timing, mechanism and transmission. The reward is methodological discipline rather than a parade of spectacular cases.

The evolutionary toolkit. Robert Poulin, *Evolutionary Ecology of Parasites*, second edition (Princeton University Press, 2007). This is the demanding option. Poulin develops the theory behind host specificity, virulence, life-history evolution, parasite diversity, aggregation and community structure, testing broad claims against comparative data. It assumes comfort with evolutionary and ecological reasoning and sometimes with quantitative evidence. Read it for the field's machinery rather than its most spectacular organisms, and expect a reference work rather than a leisurely narrative. It is particularly strong when a broad claim about parasites needs to be converted into a testable evolutionary question, compared across species and separated from the anecdote that first made the claim attractive. Keep it beside recent papers because the field has continued to move.

Notes and Sources

Scientific claims, current burden figures and publication details were checked on 9 August 2026. The notes follow the book's order and identify the main evidence behind claims that carry unusual numerical, causal or interpretive risk. Routine biological descriptions are supported by the standard syntheses listed in the bibliography.

The Whole Thing in One Page and Why You Should Care

What counts as a parasite. The book uses an ecological definition: a parasite obtains habitat or resources through an intimate association that reduces host fitness on average. That covers lineages far beyond medically important worms while keeping predation, parasitoidism and mutualism available as neighbouring categories rather than pretending that every interaction has a sharp permanent boundary. Claude Combes and Robert Poulin provide the main theoretical foundations for this treatment.

Independent origins. The statement that animal parasitism arose at least 223 times in fifteen phyla comes from Weinstein and Kuris's comparative survey. The figure is a documented minimum, not an estimate of every origin that ever occurred. Daniel Nickrent's review supports the estimate of about 4,750 parasitic flowering-plant species and twelve independent origins among angiosperms. Both counts depend on classification and phylogenetic resolution, so the book uses them to establish repeated evolution rather than exact final totals.

Current human burden. The World Health Organization estimated 282 million malaria cases and 610,000 deaths in 2024. Its February 2026 schistosomiasis fact sheet estimated that at least 253.7 million people required preventive treatment in 2024 and reported transmission from 79 countries. These are modelled and reported public-health figures, not direct censuses. Their purpose here is scale and distribution, not clinical guidance.

Sickle haemoglobin and malaria. Allison's 1954 study established the association between sickle-cell trait and protection from malaria. Later work has clarified mechanisms and limits. Band and colleagues showed that the degree of protection depends partly on *Plasmodium falciparum* genotype. The association supports a cautious claim that host and parasite genetic variation interact and may reflect reciprocal selection. The manuscript distinguishes one-copy protection from the serious disease risk associated with inheriting two sickle variants and does not present the trait as an uncomplicated benefit.

Guinea worm. The Carter Center's provisional 2025 figures reported ten human cases, compared with an estimated 3.5 million annual cases when the eradication programme began in 1986. Animal infections, especially in dogs, are now central to the final eradication problem. The case is used to show how mapping a narrow life cycle can create several physical interruption points without a vaccine or curative medicine.

Core Ideas evidence

Relationship and dependence. Combes's treatment of hosts as environments informed the organising model, but the final manuscript gives equal weight to dependency, specialisation and host defence. It clarifies why internal residence creates resources, stability, hazards and routes in much the same way that any habitat does, without implying that all parasites face the same conditions. The contrast between ectoparasites, intracellular protists, helminths, fungi and parasitic plants is retained throughout to prevent one human-disease model from standing for the field.

Virulence and transmission. The trade-off account is presented as a framework, not a universal law. Alizon and colleagues review its history and empirical problems; Cressler and colleagues compare theoretical predictions with tests. De Roode, Yates and Altizer provide a strong system in monarch butterflies, where more harmful parasite genotypes can produce more transmission stages while host debilitation creates a cost. Host immune responses can themselves cause damage, which is why the manuscript separates parasite growth from total pathology.

Complex life cycles. Auld and Tinsley review the evolutionary ecology of complex cycles and the conditions under which added hosts can improve growth, reproduction or transmission while adding points of failure. The malaria sequence follows the Centers for Disease Control and Prevention's life-cycle account. The wording definitive host is applied by reproductive stage: sexual reproduction occurs in the mosquito, so human narrative importance does not determine the label.

Immune evasion. McCulloch and colleagues support the description of

antigenic variation in African trypanosomes. Johnston and colleagues identified a structurally distinct TGF-beta mimic secreted by *Heligmosomoides polygyrus* that binds host receptors and induces regulatory T cells in experimental systems. This is used as one concrete mechanism, not as a claim that all helminths suppress immunity in the same way.

Manipulation and wider ecological effects. Lafferty and Morris linked *Euhaplorchis californiensis* infection with conspicuous killifish behaviour and increased predation by birds. Shaw and colleagues later connected infection with altered brain monoamine systems. Fredericksen and colleagues used three-dimensional imaging to show extensive fungal networks around muscles in ants manipulated by *Ophiocordyceps*, while the brain remained uninvaded during the examined stage. Doherty's critique supports the repeated caution that infection-induced change is not enough: adaptive manipulation requires evidence connecting phenotype, timing, mechanism and parasite fitness.

Food webs, biomass and population effects. Lafferty, Dobson and Kuris found that including parasites added more parasite-host links than predator-prey links on average in their resolved webs. Dunne and colleagues later clarified how the result changes when classic parasitism, predation on parasites and concomitant links are counted separately, so the book retains the correction without claiming one universal percentage. Kuris and colleagues measured parasite biomass in three estuaries and found it exceeded top-predator biomass in each. Hudson, Dobson and Newborn experimentally reduced *Trichostrongylus tenuis* in red grouse and prevented the expected population crashes, providing stronger causal evidence than a simple correlation between worms and declining hosts.

Coevolution and sex. Lively's field study of New Zealand snails and sterilising trematodes supports the parasite-mediated advantage of sexual reproduction in a natural system. The manuscript treats this as one strong contribution to the Red Queen account rather than the sole explanation for sex. Papkou and colleagues provide genomic and phenotypic evidence of

rapid reciprocal adaptation in an experimental host-parasite system. The book's broader claim is limited to repeated feedback: common host defences favour parasite countermeasures, which in turn alter selection on hosts.

Operating sequence and evidence

The malaria route. The life-cycle sequence follows the CDC's account of sporozoites, liver stages, blood stages, gametocytes and mosquito development. Cox's historical review supports the sequence from Laveran's observation of blood parasites in 1880, through Ross's mosquito work on avian malaria in 1897 and the Italian demonstration of human malaria transmission in 1898, to discovery of the liver stage in 1948. These discoveries are separated because each revealed a different location and therefore a different point of intervention.

Schistosomiasis. The description of skin-penetrating larvae, snail intermediate hosts, adult worms in blood vessels and tissue-trapped eggs follows the WHO account. Much chronic damage comes from immune reactions to retained eggs rather than direct feeding by the adults. The book uses that distinction to show why parasite burden and disease severity cannot always be treated as the same variable.

Plant parasitism. Nickrent supplies the diversity and origin framework for parasitic angiosperms. Haustoria are described functionally as attachment and transfer organs connecting parasite and host vascular systems. The book does not attempt a botanical classification of every form, since the intended job is to show that parasitism is an evolutionary strategy across kingdoms.

Breaking transmission. The Guinea worm sequence follows the Carter Center's programme documentation. Malaria control examples are drawn from WHO guidance and are included to show that one route often requires several interventions: mosquito control, prevention, diagnosis, treatment and surveillance. Treatment regimens are outside this book's scope and no medical instructions are offered.

How we know. Conway Morris reviews the sparse and uneven fossil record of parasitism. Most direct evidence comes from hard structures, traces, pathologies, inclusions and exceptionally preserved material; soft internal parasites are easily lost. Modern reconstruction therefore combines morphology, life cycles, host distributions, experiments, pathology, microscopy, molecules and phylogenies. The absence of a fossil cannot by itself date the origin of a soft-bodied parasitic lineage.

What People Get Wrong and Use It

Mind control. Janice Moore's synthesis and Doherty's later critique informed the evidential threshold. The book accepts strong cases while resisting a narrative in which every behavioural change is an adaptation of the parasite. Sickness, inflammation, neural damage, host defence and altered energy use remain alternatives until transmission benefit is demonstrated.

Parasites in wealthy countries. Lower burdens of several major parasitic diseases reflect sanitation, housing, vector control, food systems, veterinary care and access to diagnosis and treatment. They do not indicate biological absence. The correction is ecological and infrastructural, not an attempt to create a clinical list of infections.

Conservation. Carlson and colleagues' global plan argues that parasites are part of biodiversity and need research, risk assessment and conservation where appropriate. This does not conflict with eradication of selected parasites causing severe disease. The manuscript separates the relationship being targeted from the category as a whole: eliminating human Guinea worm transmission and preserving a host-specific native parasite are different decisions with different evidence.

The transferable lenses. Route, stage, damage mechanism, missing host, evolutionary response and hidden relationships are analytical prompts, not diagnostic rules. They are intended to improve questions about biological systems. Health decisions require local clinical and public-health guidance.

Terms and Go Deeper

Terminology follows standard parasitological usage, with warnings where a practical label is not a single evolutionary group. Edition, translator and publisher details for the four recommended books were checked against publisher and library records. Older works are recommended for their conceptual or narrative strengths, with their age and limits stated in the annotations.

Bibliography

Books

Combes, Claude. *Parasitism: The Ecology and Evolution of Intimate Interactions*. Translated by Isaure de Buron and Vincent A. Connors. Chicago: University of Chicago Press, 2001.

Moore, Janice. *Parasites and the Behavior of Animals*. Oxford: Oxford University Press, 2002.

Poulin, Robert. *Evolutionary Ecology of Parasites*. 2nd ed. Princeton: Princeton University Press, 2007.

Zimmer, Carl. *Parasite Rex: Inside the Bizarre World of Nature's Most Dangerous Creatures*. New York: Free Press, 2000.

Research and institutional sources

Allison, A. C. "Protection Afforded by Sickle-cell Trait against Subtertian Malarial Infection." *British Medical Journal* 1, no. 4857 (1954): 290-294. DOI: 10.1136/bmj.1.4857.290.

Alizon, Samuel, Andrew Hurford, Nicole Mideo, and Minus van Baalen. "Virulence Evolution and the Trade-off Hypothesis: History, Current State of Affairs and the Future." *Journal of Evolutionary Biology* 22, no. 2 (2009): 245-259. DOI: 10.1111/j.1420-9101.2008.01658.x.

Auld, Stuart K. J. R., and Matthew C. Tinsley. "The Evolutionary Ecology of

Complex Lifecycle Parasites: Linking Phenomena with Mechanisms." *Heredity* 114, no. 2 (2015): 125-132. DOI: 10.1038/hdy.2014.84.

Band, Gavin, Ellen M. Leffler, Muminatou Jallow, et al. "Malaria Protection due to Sickle Haemoglobin Depends on Parasite Genotype." *Nature* 602, no. 7895 (2022): 106-111. DOI: 10.1038/s41586-021-04288-3.

Carlson, Colin J., Skylar Hopkins, Kayce C. Bell, et al. "A Global Parasite Conservation Plan." *Biological Conservation* 250 (2020): 108596. DOI: 10.1016/j.biocon.2020.108596.

Centers for Disease Control and Prevention. "DPDx: Malaria." Accessed 9 August 2026.

Conway Morris, S. "Parasites and the Fossil Record." *Parasitology* 82, no. 3 (1981): 489-509. DOI: 10.1017/S0031182000067020.

Cox, Francis E. G. "History of the Discovery of the Malaria Parasites and Their Vectors." *Parasites & Vectors* 3 (2010): 5. DOI: 10.1186/1756-3305-3-5.

Cressler, Clayton E., David V. McLeod, Carly Rozins, Josée van den Hoogen, and Troy Day. "The Adaptive Evolution of Virulence: A Review of Theoretical Predictions and Empirical Tests." *Parasitology* 143, no. 7 (2016): 915-930. DOI: 10.1017/S003118201500092X.

de Roode, Jacobus C., Andrew J. Yates, and Sonia Altizer. "Virulence-Transmission Trade-offs and Population Divergence in Virulence in a Naturally Occurring Butterfly Parasite." *Proceedings of the National Academy of Sciences* 105, no. 21 (2008): 7489-7494. DOI: 10.1073/pnas.0710909105.

Doherty, Jean-François. "When Fiction Becomes Fact: Exaggerating Host Manipulation by Parasites." *Proceedings of the Royal Society B* 287, no. 1936 (2020): 20201081. DOI: 10.1098/rspb.2020.1081.

Dunne, Jennifer A., Kevin D. Lafferty, Andrew P. Dobson, et al. "Parasites Affect Food Web Structure Primarily through Increased Diversity and

Complexity." *PLOS Biology* 11, no. 6 (2013): e1001579. DOI: 10.1371/journal.pbio.1001579.

Fredericksen, Maridel A., Yizhe Zhang, Missy L. Hazen, et al. "Three-dimensional Visualization and a Deep-learning Model Reveal Complex Fungal Parasite Networks in Behaviorally Manipulated Ants." *Proceedings of the National Academy of Sciences* 114, no. 47 (2017): 12590-12595. DOI: 10.1073/pnas.1711673114.

Hudson, Peter J., Andrew P. Dobson, and Dave Newborn. "Prevention of Population Cycles by Parasite Removal." *Science* 282, no. 5397 (1998): 2256-2258. DOI: 10.1126/science.282.5397.2256.

Johnston, C. J. C., D. J. Smyth, R. B. Kodali, et al. "A Structurally Distinct TGF-β Mimic from an Intestinal Helminth Parasite Potently Induces Regulatory T Cells." *Nature Communications* 8 (2017): 1741. DOI: 10.1038/s41467-017-01886-6.

Kuris, Armand M., Ryan F. Hechinger, Jenny C. Shaw, et al. "Ecosystem Energetic Implications of Parasite and Free-living Biomass in Three Estuaries." *Nature* 454, no. 7203 (2008): 515-518. DOI: 10.1038/nature06970.

Lafferty, Kevin D., Andrew P. Dobson, and Armand M. Kuris. "Parasites Dominate Food Web Links." *Proceedings of the National Academy of Sciences* 103, no. 30 (2006): 11211-11216. DOI: 10.1073/pnas.0604755103.

Lafferty, Kevin D., and A. Kimo Morris. "Altered Behavior of Parasitized Killifish Increases Susceptibility to Predation by Bird Final Hosts." *Ecology* 77, no. 5 (1996): 1390-1397. DOI: 10.2307/2265536.

Lively, Curtis M. "Evidence from a New Zealand Snail for the Maintenance of Sex by Parasitism." *Nature* 328 (1987): 519-521. DOI: 10.1038/328519a0.

McCulloch, Richard, Christina A. Cobbold, Luisa Figueiredo, et al. "Emerging Challenges in Understanding Trypanosome Antigenic Variation."

Emerging Topics in Life Sciences 1, no. 6 (2017): 585-592. DOI: 10.1042/ETLS20170104.

Nickrent, Daniel L. "Parasitic Angiosperms: How Often and How Many?" *Taxon* 69, no. 1 (2020): 5-27. DOI: 10.1002/tax.12195.

Papkou, Andrei, Thiago Guzella, Wentao Yang, et al. "The Genomic Basis of Red Queen Dynamics during Rapid Reciprocal Host-Pathogen Coevolution." *Proceedings of the National Academy of Sciences* 116, no. 3 (2019): 923-928. DOI: 10.1073/pnas.1810402116.

Shaw, J. C., W. J. Korzan, R. E. Carpenter, A. M. Kuris, K. D. Lafferty, C. H. Summers, and Ø. Øverli. "Parasite Manipulation of Brain Monoamines in California Killifish (*Fundulus parvipinnis*) by the Trematode *Euhaplorchis californiensis*." *Proceedings of the Royal Society B* 276, no. 1659 (2009): 1137-1146. DOI: 10.1098/rspb.2008.1597.

Weinstein, Sara B., and Armand M. Kuris. "Independent Origins of Parasitism in Animalia." *Biology Letters* 12, no. 7 (2016): 20160324. DOI: 10.1098/rsbl.2016.0324.

World Health Organization. "Malaria." Fact sheet, 4 December 2025. Accessed 9 August 2026.

World Health Organization. "Schistosomiasis." Fact sheet, 23 February 2026. Accessed 9 August 2026.

The Carter Center. "Guinea Worm Disease Reaches All-Time Low: Only 10 Human Cases Reported in 2025." 30 January 2026. Accessed 9 August 2026.