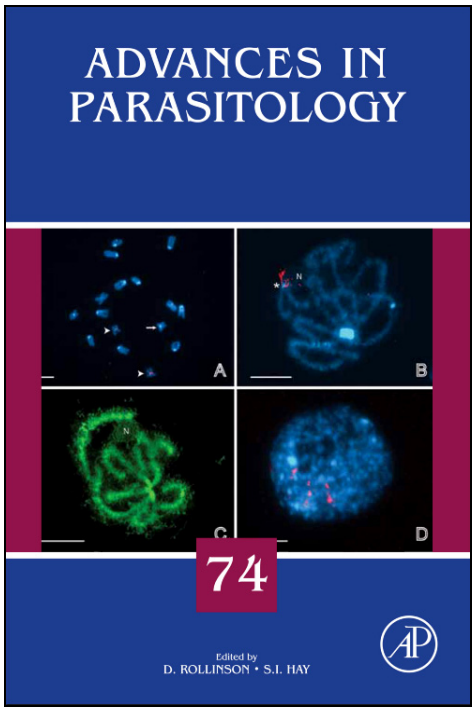

From: Robert Poulin, The Many Roads to Parasitism: A Tale of Convergence.
In D. Rollinson & S. I. Hay, editors: Advances in Parasitology, Vol. 74,
Burlington: Academic Press, 2011, pp. 1-40.
ISBN: 978-0-12-385897-9

CHAPTER 1

The Many Roads to Parasitism: A Tale of Convergence

Robert Poulin

Contents

Abstract

Parasitic organisms account for a large portion of living species. They have arisen on multiple independent occasions in many phyla, and thus encompass a huge biological diversity. This review uses several lines of evidence to argue that this vast diversity can be reduced to a few evolutionary end points that transcend

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Advances in Parasitology, Volume 74
ISSN 0065-308X, DOI: 10.1016/B978-0-12-385897-9.00001-X

phylogenetic boundaries. These represent peaks in the adaptive landscape reached independently by different lineages undergoing convergent evolution. Among eukaryotic parasites living in or on animals, six basic parasitic strategies are identified based on the number of hosts used per parasite generation, the fitness loss incurred by the host, and the transmission routes used by the parasites. They are parasitoids, parasitic castrators, directly transmitted parasites, trophically transmitted parasites, vector-transmitted parasites and micropredators. These show evidence of convergence in morphology, physiology, reproduction, life cycles and transmission patterns. Parasite–host body size ratios, and the relationship between virulence and intensity of infection, are also associated with the different parasitic strategies, but not consistently so. At the population level, patterns of parasite distribution among hosts are not uniform across all parasitic strategies, but are distinctly different for parasitoids and castrators than for other parasites. To demonstrate that the above six strategies defined for animal parasites are universal, comparisons are made with parasites of plants, in particular, plant–parasitic nematodes and parasitic angiosperms; these are shown to follow the same evolutionary trajectories seen among animal parasites, despite huge physiological and ecological differences between animals and plants. Beyond demonstrating the inevitable convergence of disparate lineages across biological hyperspace towards a limited set of adaptive strategies, this synthesis also provides a unifying framework for the study of parasitism.

1.1. INTRODUCTION

Most textbooks of parasitology emphasise the great diversity of parasitic organisms, which come in a range of sizes and shapes, occupy vastly different microhabitats within their hosts, and follow a wide range of routes for their transmission from one host to the next. The very organisation of these textbooks, with each chapter devoted to a different taxonomic group of parasites, underlines the unique features of different groups of parasites that distinguish them from each other. In sharp contrast, the goal of this review is to draw attention to the striking similarities between phylogenetically unrelated parasites that have resulted from the convergent evolution of different parasite lineages down parallel paths.

The concept of convergent evolution, or the acquisition of the same biological traits in unrelated lineages, is one of the cornerstones of evolutionary biology. Phenotypic similarities arising from convergence among unrelated organisms living under similar selection regimes are extremely common in nature. Parallel evolution towards similar phenotypes may

occur simply because genetic and developmental constraints limit the number of possible phenotypes (Orr, 2005). Alternatively, parallel evolution may be the signature of adaptation if different organisms attain analogous phenotypes via different genetic changes (Arendt and Reznick, 2008). Whatever the underlying genetic architecture, as a rule, only a limited number of phenotypic solutions are reached by different lineages evolving under similar evolutionary pressures. Biological hyperspace, the multidimensional volume corresponding to all possible combinations of phenotypic traits, can only be crossed by a few roads leading to a constrained number of viable destinations. Although other morphological or functional states are theoretically possible (see McGhee, 2007), they are maladaptive and would yield lower fitness than the small number of realised alternatives. Thus, from many different starting points, organisms generally only evolve along a few trajectories, towards stable 'attractor' points of optimal functionality, leaving most of the vast volume of biological hyperspace empty of living organisms. This is essentially what Wright (1984) meant by adaptive peaks: one or a few high points on an adaptive landscape corresponding to combinations of genes and phenotypic traits that confer on organisms a high probability of surviving and reproducing. Even slight departures from these peaks lead to lower fitness, and whatever their starting point on the adaptive landscape, selection tends to drive the genotypic composition of populations away from valleys and towards the peaks.

The central argument of this review is that parasites, despite their broad phylogenetic diversity, have converged on a limited number of adaptive peaks as reflected by analogies in morphological, ecological and epidemiological traits. For certain sets of traits, the convergence is superficial and far from universal, whereas for others it is inescapable and fundamental to the success of the various parasitic lineages. The evidence summarised here comes mostly from eukaryotic parasites, including metazoans as well as 'protozoans' or 'protists' (in the traditional sense), living in or on animals, though mention of prokaryotic parasites will be made where appropriate. Here I define parasites as organisms that have an obligate physical association with a host, and that must obtain a critical resource, usually nutrients, from the host resulting in a fitness cost to the latter. Therefore, I exclude various forms of behavioural parasitism such as brood parasitism or kleptoparasitism, although the same arguments regarding convergent evolutionary strategies could be made for them, too.

I first show how the numerous independent transitions to a parasitic mode of life by vastly different organisms have eventually led to a very limited set of outcomes in terms of general parasitic strategies. Second, I proceed to examine the evidence for convergence at both morphological and functional levels, as well as that emerging from a look at the life cycles

and transmission modes of parasites. I then discuss how parasite-to-host body size ratios show a discontinuous distribution roughly matching the divisions into parasitic strategies better than phylogenetic clades, and how the virulence associated with infection also corresponds to those strategies more-or-less independently of the parasites' phylogenetic origins. This leads to an overview of patterns in abundance and host use which suggest that parasite lineages adopting the same parasitic strategy also display convergence at population and perhaps even community levels. In order to challenge some of the patterns derived from an examination of animal parasites, the review ends with a brief comparison with selected groups of plant parasites for which good ecological information is available. Throughout, this review emphasises the commonalities among consumers belonging to many disparate phyla by showing that there are only a few stable end points for organisms adopting a parasitic mode of life, and that their phenotypic evolution is inexorably canalised towards these end points.

1.2. ORIGINS OF PARASITES AND EVOLUTIONARY TRAJECTORIES

The evolution of parasitism itself appears inevitable in any ecosystem. This is true even in cyberspace, where Ray (1994) has shown that among reproducing and mutating programs competing with each other for resources (memory space), shorter parasitic programs invariably evolve by dropping part of their code. These then borrow the missing instructions they need to copy themselves from other 'host' programs, at the latter's expense. All living organisms represent concentrated sources of nutrients and energy compared to what is available in the non-living part of the environment. Selection has favoured organisms capable of consuming smaller ones, and of course it should also favour those capable of feeding on larger ones whenever this is a possibility, given that larger organisms provide larger amounts of resources. Parasitism has therefore evolved repeatedly during the history of life, and the ensuing diversification of parasitic lineages has led to parasites *sensu lato* accounting for a substantial portion of extant biodiversity (Poulin and Morand, 2000, 2004; Windsor 1998). Transitions to a parasitic mode of life and the evolutionary paths subsequently followed by parasites are examined in this section.

1.2.1. Phylogenetic transitions to parasitism

Some taxonomic groups of parasites appear to be monophyletic, that is, the product of a single transition to a parasitic mode of life followed by diversification of the ancestral lineage. These include myxozoans

(Canning and Okamura, 2004), acanthocephalans (Herlyn et al., 2003), pentastomids (Zrzavy, 2001), nematomorphs (Hanelt et al., 2005) and the flatworm lineage that gave rise to monogeneans, trematodes and cestodes (Littlewood et al., 1999). In contrast, in many other groups of parasites, extant parasite species are the product of several independent transitions, and are therefore polyphyletic. Turbellarian flatworms include many parasitic species descended from unrelated ancestors (Rohde, 1994). Among nematodes, parasitism of animals has evolved on many independent occasions (Clarck, 1994), at least four times according to molecular evidence (Blaxter et al., 1998). Some groups of parasitic insects are monophyletic, such as strepsipterans (Whiting et al., 1997), fleas (Krasnov, 2008) and parasitoid hymenopterans (Whitfield, 1998), whereas in other groups, like blowflies (Stevens, 2003) and lice (Johnson et al., 2004; Murrell and Barker, 2005), parasitism arose independently a few to several times. Parasitism has also arisen numerous times among crustaceans, including several separate transitions within large lineages like copepods (Boxshall and Halsey, 2004; Poulin, 1995a), isopods (Dreyer and Wagele, 2001; Poulin, 1995b) and amphipods (Poulin and Hamilton, 1995). To this list could be added many other groups, such as entomopathogenic fungi (Humber, 2008) or gastropods (Ponder and Lindberg, 2008), or of course protists as varied as ciliates, flagellates and apicomplexans (i.e. *Plasmodium*, *Toxoplasma*), in which parasitism has been adopted repeatedly by independent lineages.

In addition to the above examples and others well covered in parasitology textbooks, parasitism has also evolved in numerous other taxa that are often completely overlooked in reviews of evolutionary transitions to parasitism. These include foraminiferans (Cedhagen, 1994), cnidarians (Raikova, 1994), polychaetes (Hernández-Alcántara and Solis-Weiss, 1998; Poulin, 2001), pearlfishes (Parmentier and Das, 2004) or the infamous candirus, a large group of Neotropical catfishes that live within the gills of larger fish and feed on their blood or flesh, rarely penetrating the urethra of humans or other mammals (Fernández and Schaefer, 2009).

When one starts adding up all the known independent transitions to a parasitic mode of life, the total rises rapidly. Besides incompletely resolved phylogenies, two factors can lead to an underestimation of the true number of times that parasitism has arisen during the evolutionary history of life on Earth. First, some parasitic lineages may have gone extinct without leaving a trace. Occasional fossil evidence indicates that host–parasite associations that existed millions of years ago have no present-day counterparts (e.g. Poinar et al., 1997), suggesting that at least certain parasite lineages no longer exist. Second, some transitions to parasitism may be reversible, especially in parasites that show little morphological specialisation for parasitism compared to their free-living counterparts. Returns to a free-living life style have been either postulated

or documented in protists (Siddall et al., 1993), nematodes (Bert et al., 2006), mites (Radovsky et al., 1997; Smith, 1998), leeches (Borda and Siddall, 2004; Light and Siddall, 1999) and lampreys (Salewski, 2003). These reversals could in some cases eliminate all traces of earlier transitions to parasitism. With these caveats in mind, a reasonable estimate still amounts to at least 100 independent transitions among eukaryotes from a free-living existence to one of obligate parasitism on animals.

Given the many independent origins of parasitism across numerous phyla, one might expect a diverse array of ecological attributes and host exploitation strategies among extant parasites. This is not really the case, however. Living parasites fall within a limited set of more-or-less distinct parasitic strategies; these represent the signature of convergent evolution, and they are defined in Section 1.2.2.

1.2.2. Observed evolutionary trajectories of parasites

There have been earlier categorisations of parasites based on shared traits rather than phylogeny. For example, it is common in ecological parasitology to distinguish between internal or endoparasites, and external or ectoparasites. This simple classification, however, although useful in certain circumstances, breaks down in many cases, such as 'mesoparasitic' copepods that are partially embedded in host tissues but still exposed to the external aquatic habitat (Boxshall and Halsey, 2004). Parasites are also sometimes classified based on their life cycle patterns, for example, parasites with simple versus complex life cycles. Another distinction is often made between typical parasites that do not kill their host and parasitoid insects that do. All these attempts at categorising parasite strategies fail because they each consider a single trait, rather than taking a more holistic view of all key parasite features and their associations.

The most influential division of parasites has been Anderson and May's dichotomy between microparasites and macroparasites (Anderson and May, 1979; May and Anderson, 1979). Their proposal for this fundamental division was accompanied by two general sets of mathematical models, one for each type of parasite, designed to analyze host–parasite population dynamics. These models have underpinned the work of an entire generation of epidemiologists and played an important role in our understanding of the dynamics of parasites of medical, veterinary and conservation importance (Diekmann and Heesterbeek, 2000). The essential feature of microparasites is that their impact on host survival or fitness, that is, their virulence, is not dependent on the number of separate infection events, since they multiply directly within the host. In contrast, the virulence of macroparasites is intensity dependent: because macroparasites do not multiply within the host, their impact is proportional to the number of separate individual parasites that infect the

host. Parasites capable of multiplying within their host tend to be unicellular organisms such as protozoans, bacteria and viruses, whereas those not capable of multiplication within the host are generally metazoans like helminths and arthropods, and thus the names micro- and macroparasites are a reflexion of the body sizes of these parasites. Perhaps unfortunately, the terms micro- and macroparasites have become widely associated with those taxa, although in principle their definitions transcend any taxonomic boundaries.

More recently, Kuris and Lafferty (2000) have recognised a suite of distinct parasite strategies derived from dichotomies in (i) the number of hosts exploited by an individual parasite at a particular stage in its life history, (ii) whether the effect of the parasite is to reduce host fitness to zero or only to reduce it a little, (iii) whether this effect is intensity dependent and (iv) whether host death is necessary for parasite transmission (see also Lafferty and Kuris, 2002). By applying these dichotomies in a factorial design, they define a set of possible strategies for parasitism, most of which are realised in nature. Kuris and Lafferty's (2000) scheme, as opposed to earlier ones, uses several life history traits of parasites to identify a limited number of convergent evolutionary trajectories leading to a few adaptive peaks, and it thus serves as a great starting point for the present synthesis.

Having said that, the proposed categories of Kuris and Lafferty (2000) do not always fully capture the strong convergence that has marked the evolution of parasitism. First, some essential aspects of parasite biology, such as life cycle patterns and transmission mode, are only partially incorporated in the Kuris and Lafferty (2000) classification, and yet they provide some of the strongest signals of convergence. Second, the rules they followed to divide parasites into strategies are sometimes based on properties determined by phylogeny rather than features that are consequences of a transition to parasitism. For instance, the 'pathogen' and 'trophically transmitted pathogen' categories of Kuris and Lafferty (2000) have been erected solely because they induce fitness reductions in their hosts that are not dependent on intensity, since these parasites multiply within the host. With few exceptions (e.g. the monogeneans *Gyrodactylus* spp.), these parasites are mostly protozoans, bacteria or viruses; free-living protozoans and bacteria also readily multiply asexually, and therefore this ability and its consequences for host fitness are properties of the taxa involved and not adaptations evolved for parasitism. Finally, Kuris and Lafferty (2000) considered strategies as specific to a particular life history phase of the parasite. It is true that the relationship of a helminth parasite with its intermediate host when it is a juvenile can be very different from that with its definitive host when it is an adult. The exploitation strategies of parasites are stage specific and not species specific. However, selection also shapes an optimal, integrated set of

ways of dealing with different hosts as a global strategy for a particular genotype, in addition to acting separately on different life stages. Some convergent patterns are only apparent when the entire life history of parasites is considered. Therefore, I opted to depart slightly from the Kuris and Lafferty (2000) scheme. Unlike theirs, my goal is not to define strict categories, but rather to describe general evolutionary end points towards which most parasite lineages tend to converge.

Figure 1.1 summarises the six parasite strategies recognised here and illustrates the fundamental differences between them; other characteristics of these strategies are mentioned below and in later sections. In an attempt to define a small number of distinct parasitic strategies, I do not dwell on minor exceptions. There are indeed taxa that do not sit comfortably under any label; typically, they share many traits with one of the strategies defined below, but depart from it with respect to one or two characteristics. Within the framework of adaptive peaks, these exceptions can be seen as standing on the slope of a peak, but not really in the valleys between peaks. They are therefore slightly off the ideal 'type' for a particular strategy, as opposed to distinct alternative strategies. They will be mentioned where relevant in what follows; the important fact

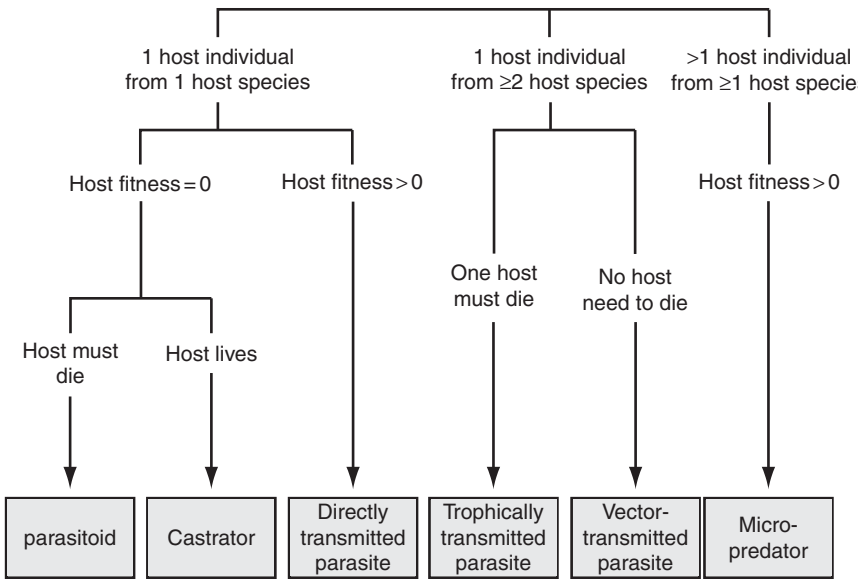

FIGURE 1.1 Classification tree of the six parasitic strategies considered here, and encompassing the vast majority of known parasite taxa. The first division is based on the number of hosts used, both in terms of species and individuals, by one full parasite generation; subsequent divisions are based on fitness impact on hosts.

here is that the vast majority of parasites fit very well within one of the following six strategies.

Parasitoids grow inside a single host and kill that host as a normal and necessary part of their development. Typically, from infection of the host until the latter's death, parasitoids increase their own mass by several orders of magnitude, and achieve final sizes only slightly smaller than that of the host. The best-known parasitoids are braconid wasps and their caterpillar hosts, as well as tachinid or conopid flies and their insect hosts (Godfray, 1994). The term parasitoid has been coined almost a century ago specifically to assign a name to the strategy of these insects. Other parasitoids include nematomorphs (hairworms) and mermithid nematodes in their arthropod hosts, *Cordyceps* fungi in their insect hosts and oenonid polychaetes in their polychaete hosts. Host death at the end of the infection is in principle not necessary for parasite transmission; if the host somehow survived when the parasitoid is finished with it, there would be no negative fitness consequences for the parasitoid. Indeed, crickets can occasionally survive following the emergence of nematomorphs from their body cavity (Biron et al., 2005). Although host death is not favoured by selection acting on parasitoid genes, it is nonetheless an almost inevitable outcome of the considerable damage caused by the parasitoid during its development.

Parasitic castrators show many parallels with parasitoids (Kuris, 1974). Instead of killing their host, castrators block host reproduction and use the host's reproductive investments for their own reproduction. Here I am talking not just of moderate to large reductions in host reproductive output, but of an almost total suppression of host reproduction. Like parasitoids, castrators also attain relatively large sizes compared to their hosts. However, castrators do not reduce host lifespan, and can obtain a high transmission rate without trading off longevity. Obrebski (1975), Jaenike (1996) and Bonds (2006) have modelled the benefits of castration, and found that castration should be advantageous under many conditions. Castrators include ascothoracican barnacles in echinoderms, entoniscid isopods and rhizocephalan barnacles in their crustacean hosts and strepsipteran insects in their hymenopteran hosts. Of course, the larval stages of several helminths in their intermediate hosts also act as castrators; for example, the sporocysts and rediae of trematodes completely castrate their snail hosts, and the larval stages of cestodes or acanthocephalans can induce almost complete castration in their arthropod or fish intermediate hosts (Lafferty and Kuris, 2009). As adults, these parasites adopt a different strategy (*Trophically transmitted parasites*; see below) since castration is only one part of their lifetime bag of tricks. Castration by these various taxa is achieved either directly, by feeding on the gonads of the host, or indirectly by either diverting energy away from gonad development or by the secretion of ''castrating'' hormones (Baudoin,

1975; Coustau et al., 1991; Høeg, 1995; Lafferty and Kuris, 2009; Webb and Hurd, 1999).

Directly transmitted parasites infect only one host individual in their lifetime, and generally induce little or moderate pathology; however, some directly transmitted parasites can be quite virulent. This strategy is used by a wide range of parasites, including many endoparasitic metazoans like nematodes or turbellarians; ectoparasitic metazoans such as monogeneans, copepods, cyamid amphipods; most cymothoid isopods, lice and mites; unicellular parasites like microsporidians and many kinds of 'protozoans' (e.g. *Giardia*, *Eimeria*); and of course numerous fungal, bacterial and viral pathogens. Often, they are parasitic for almost their entire life, with only infective stages (eggs, spores, etc.) living off the host. Some, though, are parasitic for only part of their life. For example, in unionid bivalves, only the juvenile stages, or glochidia, are parasitic on fish; adults are typical free-living bivalves. Most directly transmitted parasites that are unicellular tend to multiply within the host, whereas this is very rare among the metazoan parasites. Entomopathogenic nematodes (families Steinernematidae and Heterorhabditidae) are unusual in that they multiply within their insect host after the latter is killed by bacteria carried symbiotically by the nematodes (Gaugler, 2002). Nevertheless, the ability to multiply within the host is generally a property of the higher taxa to which these parasites belong, and not a specific adaptation forming a part of this parasitic strategy. From an evolutionary perspective, most transitions to a parasitic mode of life probably resulted in directly transmitted parasites, given their basic nature; from this starting point, many lineages subsequently evolved more intense exploitative strategies, such as parasitoidism or castration, or added extra hosts to their life cycle.

Trophically transmitted parasites must infect two or more host species in a given sequence, and must be transmitted from an intermediate host to their definitive host, in which they mature, through predation of the former by the latter. The definitive host is almost invariably a vertebrate, whereas a broad range of invertebrates and vertebrates can serve as intermediate hosts, depending on the parasite taxon. These parasites include all trematodes (schistosomes have secondarily lost the transmission-by-predation stage of their life cycle) and cestodes, all acanthocephalans, all pentastomids, many nematodes and several 'protozoan' taxa (e.g. *Toxoplasma*, *Sarcocystis*). In a few cases, there are two instances of transmission by predation in the same life cycle, with the other one occurring at an earlier stage between two different intermediate hosts, as in the well-studied cestodes *Schistocephalus* and *Ligula*. In their intermediate hosts, trophically transmitted parasites can sometimes be quite virulent. Trematode larval stages in their snail first intermediate hosts almost invariably act as parasitic castrators (Lafferty and Kuris, 2009). Juvenile stages in

other taxa, like cystacanths of acanthocephalans and plerocercoids or cysticercoids of cestodes, can also cause very pronounced reductions in the reproductive output of their intermediate hosts (Heins et al., 1999; Rauque and Semenas, 2009; Webb and Hurd, 1999). In contrast, the virulence of the adult stages of trophically transmitted parasites in their definitive host is usually very low, and often fitness impacts on the host are too small to be quantified.

Vector-transmitted parasites must infect two hosts to complete their life cycle: the first is almost always a vertebrate, and the second is a micropredator (see below) that acts as a vector between vertebrate hosts to ensure the passage of new parasite generations to new hosts. Vector-transmitted parasites tend to be very small, in both absolute and relative terms, and live in the blood of their vertebrate definitive host, from where they are acquired by blood-sucking micropredators. They include filaroid nematodes, many well-known disease-causing 'protozoans' (e.g. *Plasmodium*, *Babesia*, *Leishmania*, *Trypanosoma*), as well as numerous bacteria and viruses. These parasites generally multiply within their vertebrate host, and induce significant pathology and morbidity (Ewald, 1983).

Micropredators feed on multiple host individuals per generation; depending on their host specificity, these hosts may belong to one or many species. Micropredators do not form durable associations with one host individual of a particular species, as seen in the other types of parasites. Each micropredator attack on one host is generally brief, lasting from several seconds to several days, and is followed by a period spent off the host, often used for moulting and growing, or for reproduction and egg deposition. On each visit to a host, a relatively small amount of tissue, usually blood, is removed from the host. Micropredators include leeches, branchiuran crustaceans, gnathiid isopods, mosquitos and other blood-sucking dipterans (Simuliidae, Streblidae, etc.), fleas, ticks, lampreys and vampire bats; one could even count cookie-cutter sharks as micropredators, since they take a single bite of flesh from a host and move on. As a rule, the direct impact of blood-sucking micropredators on host fitness is minimal, except when they are relatively large compared to host size (e.g. lampreys). However, they can indirectly cause significant losses of host fitness when they carry vector-transmitted parasites that induce disease, or when they open wounds that serve as entry points for pathogenic bacteria or viruses.

None of these six strategies has a single evolutionary origin; within each strategy, representative species are spread across many phyla (Fig. 1.2). Each of them is the common end point of several independent lineages that meet on an adaptive peak. The sections that follow discuss many other convergent features displayed by parasites in general, and by unrelated parasite taxa that ended up on the same peak.

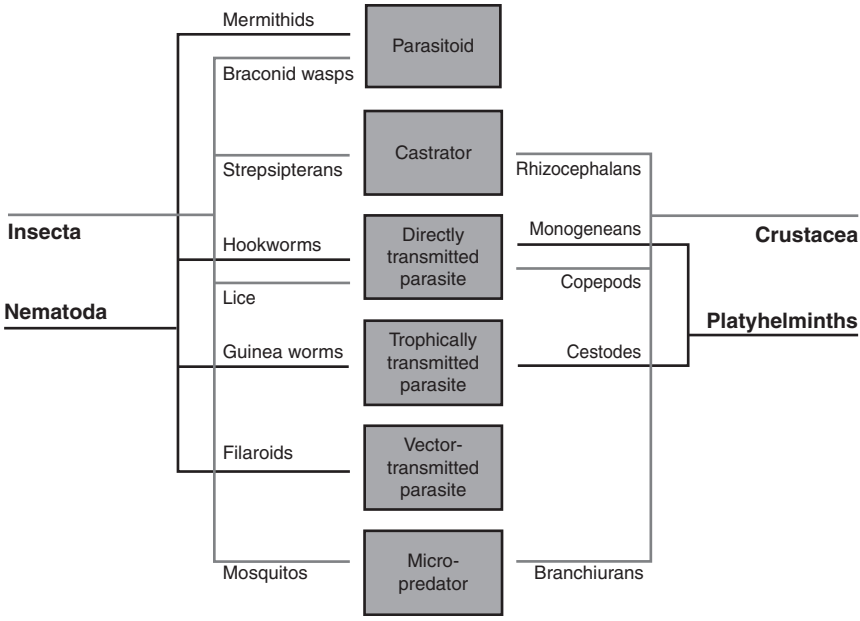

FIGURE 1.2 Representation of some higher taxa of parasites in each of the six parasitic strategies. Members of the same higher taxon may adopt different strategies, and each strategy consists of members of different higher taxa. Note that the trees are not meant to reflect actual phylogeny.

1.3. CONVERGENCE IN MORPHOLOGY AND FUNCTION

It is tempting to say that, at a superficial morphological level, the most obvious case of convergence among parasitic lineages is their apparent loss of structural complexity when compared to either their closest free-living relatives or their hosts. Words like retrogressive and degenerative have been associated with parasite evolution in the scientific literature (see Brooks and McLennan, 1993, for review), and loss of structural complexity has even been coined sacculinisation, after a parasitic castrator, the rhizocephalan *Sacculina*. Apart from most parasitoids and micropredators, it is true that parasites from many lineages have lost sense organs and other structures. In many families of parasitic copepods, the adults show no obvious signs of the segmentation, cephalic sensory structures or articulated appendages that are the hallmarks of their phylum (Boxshall and Halsey, 2004). Many parasitic gastropods have lost their molluscan shell and general body plan (Combes, 2005; Ponder and Lindberg, 2008). There are cases of extreme morphological simplification, as in myxozoans or the curious dicyemid mesozoans parasitic in the

kidneys of cephalopods, which have effectively regressed from a multicellular to a protistan state (Canning and Okamura, 2004; Noto and Endoh, 2004; Okamura and Canning, 2003). However, modern electron microscopy has revealed a wide array of new sensory receptors in parasitic flatworms (Rohde, 1989, 1994), and rigorous assessments of rates of character changes in these parasites indicate that the majority of evolutionary changes in morphological characters were innovations rather than losses (Brooks and McLennan, 1993). Nevertheless, it remains true that many parasitic lineages have convergently evolved towards a simpler morphology following their transition to a parasitic mode of life.

It is at a functional level that the more interesting instances of convergence occur. For instance, many independent lineages have adopted the same site of infection. Filaroid nematodes, schistosome trematodes and numerous protozoans live in the blood of vertebrate hosts. The host gastrointestinal tract is also a common site of infection, having been invaded independently by acanthocephalans, several lineages of nematodes, the common ancestor of cestodes and trematodes and numerous protozoans. It comes with an obvious way in for infective stages, and an obvious way out for their propagules after they reproduce. In a further case of convergence, among parasitic nematodes, gut-dwelling lineages have made repeated and independent transitions from the gut to living in other tissues (Nadler et al., 2007). Evidence of parallel evolution comes not only from the actual site of infection adopted by different parasites, but also from their methods of within-host migration between their point of entry in the host and the final site of infection, with remarkable similarities between lineages as different as nematodes and trematodes (Haas et al., 2007; Sukhdeo and Sukhdeo, 1994, 2002).

The adoption of a particular site of infection has an impact on the subsequent evolution of parasites. Among helminths residing in the host gut, attachment structures are necessary to avoid being dislodged by passing gut contents and peristaltic movements; in response, different lineages have all evolved some form of attachment structures. Hooks and suckers are recurring themes, though the exact mechanism used by a particular taxon varies widely. Kearn (2005) provides a very clear comparison of the attachment structures of monogeneans, copepods and other ectoparasites of fish. Although these do not provide cases of convergence at the level of anatomical details, they still show the inevitable appearance of structures with similar functions in completely unrelated lineages. The site of infection imposes other selective pressures that lead to convergent evolution. For example, all parasite lineages living in the host gastrointestinal tract have evolved an anaerobic metabolism and resistance against enzymatic attack (Cox, 1993). Also, since digested food is readily available thanks to the host, two unrelated groups of gut-dwelling parasites, acanthocephalans and

cestodes, have lost their mouth and digestive system, and simply absorb nutrients through their integument.

Some parasites that live within host cells have convergently evolved the ability to alter the cell environment to suit their needs. These include the nematode *Trichinella spiralis* as well as many protozoans (Despommier, 1993; James and Green, 2004; Lüder et al., 2001). They can induce profound structural alterations in the cell, or delay and even prevent apoptosis, or programmed cell death, of the host cells they inhabit. All this is presumably accomplished by altering host gene expression in parasitised cells.

For parasites as for any other organism, life is all about successful reproduction. The modes of reproduction used by different parasites bear legacies from their ancestors, and are to some extent dictated or constrained by phylogeny. Nevertheless, there are some examples of convergent evolution in the way parasites reproduce and achieve transmission. In particular, certain combinations of traits related to reproduction appear much more likely to evolve than other combinations. In an analysis of species traits in 21 independent lineages of ectoparasitic arthropods, consisting of micropredators and directly transmitted parasites, Poulin (2009) found that although most pairwise combinations of character states have been adopted at least once, some combinations are much more frequent than others, suggesting they evolve in tandem. For instance, laying eggs off the host is associated more frequently with the micropredator strategy and laying them on the host is associated more frequently with the direct-transmission strategy (Fig. 1.3). Also, if the host is an ectotherm, the parasite is more likely to produce mobile infective stages capable of active host finding, whereas if the host is an endotherm, the infective stages are more likely not to be mobile and depend on host-to-host contacts for transmission (Fig. 1.3). This last association is probably not independent from the fact that ectothermic hosts often inhabit aquatic environments, where free-swimming infective larvae can be utilised. Nevertheless, the analysis considered 194 possible pairwise combinations of character states, and revealed that arthropod parasites have tended to converge on some parts of the multidimensional character space more than on others (Poulin, 2009).

Several earlier textbooks emphasised the very high fecundity of many parasitic lineages, particularly some cestodes and nematodes. However, phylogenetic analyses cast some doubt on the generality of any evolutionary trend towards huge reproductive output associated with any form of parasitism (see Poulin, 1995c, 1996). Indeed, *all* organisms should be under strong selection to maximise their reproductive output, and not just parasites. A common problem faced by many directly and trophically transmitted parasites is that the co-occurrence and physical encounter in the same host at the same time of a male and a female parasite of the same

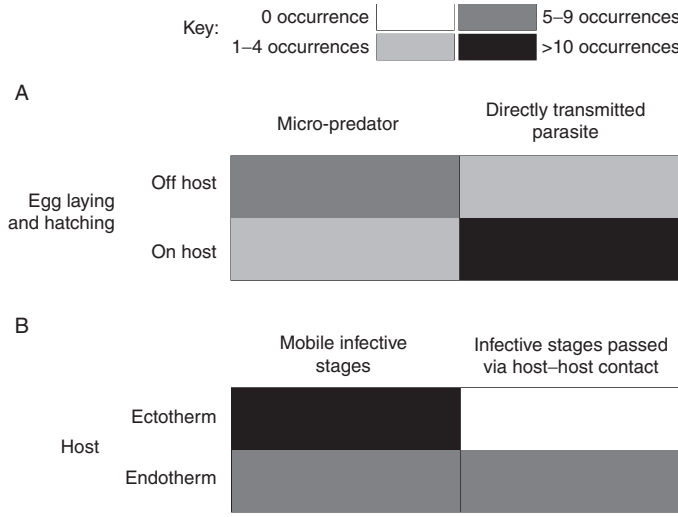

FIGURE 1.3 Two simple 2 × 2 matrices of combinations of character states seen among 21 independent lineages of ectoparasitic arthropods. These are excerpts of a larger matrix of 194 possible pairwise combinations of nine characters, each existing in two to four states. (A) Association between parasitic strategies and egg-laying habits; (B) association between host type and mobility of infective stages. The darker the shading of the cell, the more frequent the trait combination. Modified from Poulin (2009).

species are unlikely in many cases because of low population density. This is possibly less of an issue for hermaphroditic cestodes and trematodes, but in both hermaphroditic and dioecious taxa, there are apparently convergent cases of two individuals mating for life. The lifelong, although not always strict, monogamy of schistosome trematodes achieved by the male worm permanently holding the female is one example (Basch, 1990). Perhaps more striking, in the monogenean family Diplozoidae and in some members of the trematode family Didymozoidae, two pre-adult worms become physically fused for life in the days following their first meeting (Kearn, 1998). The fusion of two individuals also occurs in several families of parasitic copepods, such as the Chondracanthidae, where the small male attaches permanently to the female's genital region upon their first encounter (Raibaut and Trilles, 1993). Rare mating opportunities have thus driven similar adaptations in different lineages.

The above discussion illustrates that at a functional level, that is, with respect to site selection, attachment, feeding or reproduction, there are numerous examples of convergent evolution among unrelated parasite lineages. Section 1.4 takes this one step further, by looking at one of the defining characteristics of parasites: their life cycle.

1.4. CONVERGENT LIFE CYCLES AND TRANSMISSION MODES

Several specific aspects of parasite transmission show clear similarities among unrelated taxa suggestive of convergent evolution. For example, many taxa have long-lived spores or eggs that are very resistant to environmental conditions, whereas many others, particularly in aquatic habitats, use mobile, non-feeding infective larvae well equipped for finding suitable hosts. Particular modes of entry into the host have also evolved in completely unrelated lineages; for instance, the cercariae of schistosome trematodes and the infective juveniles of hookworm nematodes use remarkably similar mechanisms to penetrate human skin. However, it is the entire life cycle, and not any particular infection or transmission event, that is associated with each of the six parasitic strategies defined earlier (Fig. 1.4). The fact that practically all parasitic lineages use one of the general life cycles shown in Fig. 1.4 suggests that other possibilities, whether they have been explored or not over evolutionary time by failed lineages, do not correspond to adaptive peaks.

1.4.1. Evolution of complex life cycles

Vertical transmission, that is, transmission from parent to offspring occurring via ovaries, milk, or through the placental wall, is relatively rare among parasites, and generally limited to small-bodied parasite taxa. When present, vertical transmission can add some twists to the life cycle, such as the alternation of host species between parasite generations seen in some microsporidians (Smith, 2009). If we exclude these unusual cases, there are only two strategies, trophic transmission and vector transmission, that necessitate one individual of at least two host species for completion of the cycle. In the case of trophic transmission, we can imagine an ancestral parasite with a simple one-host life cycle and how changing external conditions may have exerted selective pressures favouring adjustments to the life cycle. For instance, the parasite's host may have found itself becoming part of new food chains because either new prey or new predators arrived on the scene. If the host became the frequent prey of a new predator, selection would have favoured any parasite capable of surviving ingestion by the predator, and capable of surviving inside and exploiting this new resource. If the host started to feed regularly on a new prey, selection would have favoured parasites capable of exploiting the prey as a conduit towards the host, especially if the traditional mode of transmission was associated with low infection success. In the former scenario (upward incorporation of a new host), a new definitive host would be added to the cycle, whereas in the second

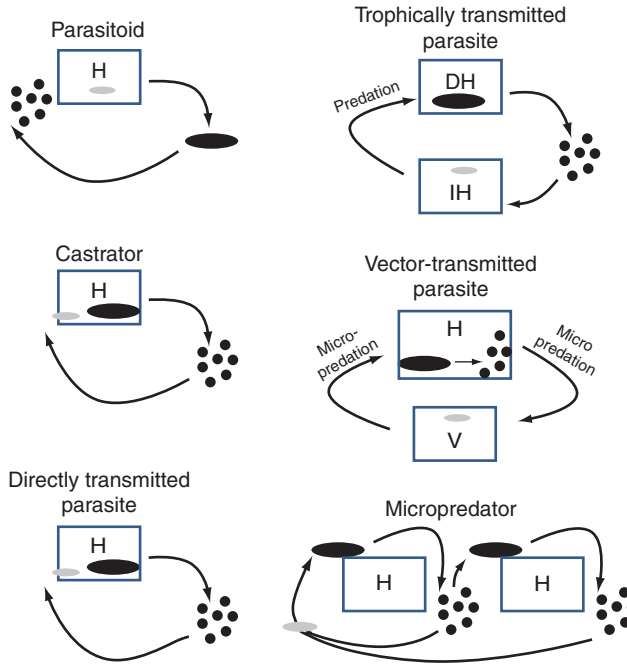

FIGURE 1.4 Characteristic life cycle patterns of the six parasitic strategies. These are meant to represent the most typical patterns, and particular taxa may show slight departures from the patterns shown here. In particular, protozoan parasites with either direct or vector transmission can mature and reproduce within their host or their vector, trophically transmitted parasites can have two or even three intermediate hosts, and not just one, and micropredators can sequentially attack much more hosts than shown in the figure. In each cycle, the developmental sequence proceeds clockwise through a single generation; adult parasites are indicated by black ellipses, juvenile stages by smaller grey ellipses, and eggs or other kinds of propagules by clusters of black dots. H, host; DH, definitive host; IH, intermediate host; V, vector.

scenario (downward incorporation) it would be a new intermediate host. These situations must have occurred regularly often over evolutionary time, and mathematical models show clearly that adopting a longer cycle with trophic transmission would benefit parasites by allowing prolonged growth and greater lifetime fecundity, or by concentrating mature parasites in a few hosts in a way that facilitates mating and cross-fertilisation (Brown et al., 2001; Choisy et al., 2003; Parker et al., 2003).

Elongation of life cycles involving one stage of trophic transmission has been a recurrent theme in the evolutionary history of parasites. In acanthocephalans, a two-host life cycle with transmission by predation is an ancestral trait shared by all extant species, and thus it evolved only

once in that group (Herlyn et al., 2003). The same is true for pentastomids (Riley, 1986). However, trophic transmission and complex life cycles evolved on a few independent occasions among nematodes (Adamson, 1986; Blaxter et al., 1998). Similarly, among flatworms, multi-host life cycles with trophic transmission evolved repeatedly among both cestodes and trematodes (Cribb et al., 2003; Littlewood et al., 1999; Olson et al., 2001). The same is undoubtedly true for protozoans, although this remains to be confirmed by phylogenetic analysis. In any event, overall there is solid evidence that the life cycles of trophically transmitted parasites result from numerous independent origins followed by convergent evolution towards the same adaptive peak.

In the case of vector transmission, a similar overall conclusion is unavoidable, although here too detailed phylogenetic evidence is often lacking. This strategy originated once among nematodes in the ancestor of filaroids, and several times among protozoans since it is used by unrelated taxa. And of course it appeared in different groups of bacteria and viruses. There are no explicit mathematical models of the evolution of vector transmission as a parasitic strategy, but the benefits of hitching a ride on a vector must have exerted strong pressures on a range of small-bodied parasites, here too leading to convergence of disparate lineages towards a shared strategy.

It is not entirely true that all multi-host life cycles involve either trophic or vector transmission. A very small number of exceptions exist, in which mobile juvenile parasites leave their intermediate host to seek their definitive host. Schistosome trematodes do this, of course, but in their case this transmission strategy is most likely derived from ancestral trophic transmission following a drastic decrease in rates of predation by the former definitive host (Shoop, 1988). The exceptions I have in mind involve three other lineages. First, in a group of myxozoans, infective stages leave an oligochaete to seek a fish host (Canning and Okamura, 2004). Second, many ticks require two or three individual hosts, usually of different species, to complete their life cycle (Oliver, 1989). Third, some ectoparasitic copepod genera (e.g. *Lernaeocera*) of the family Pennellidae also require two hosts. After developing into adults on one fish, they detach from it and the females must then attach to a new fish host of a different species, on which they usually undergo drastic morphological changes and massive increases in size prior to egg laying (Boxshall and Halsey, 2004; Whitfield et al., 1988). The fact that these exceptions involve species-poor clades is telling: a complex life cycle outside the bounds of trophic or vector transmission cannot be adaptive if the few lineages adopting this transmission route have failed to radiate even a little.

1.4.2. Adaptations to transmission challenges

The adoption of a multi-host life cycle in turn exerted selective pressure on parasites to adapt to some of the demands of a longer and more intricate transmission route (Poulin, 2007a). Many textbooks emphasise the great losses associated with each transmission event (e.g. Esch and Fernández, 1993). One solution to this attrition over the course of a long cycle with many transmission events would be to evolve the ability to multiply asexually within an intermediate host, to start the next leg of the cycle with many offspring, just as adult parasites each produce many offspring released from the definitive host. Perhaps not surprisingly, trematodes have evolved this very ability. A single larva infecting a snail first intermediate host causes the castration of the host, thereby freeing energy that can be channelled towards the parasite's reproduction (Lafferty and Kuris, 2009); over the course of its life within the snail, that trematode larva will produce, depending on the species, hundreds, thousands, or even tens of thousands clonal copies of itself that will continue the life cycle. Remarkably, trematodes are not the only trophically transmitted parasites capable of asexual multiplication in intermediate hosts: the trait has evolved independently in some cestode lineages (Moore, 1981; Moore and Brooks, 1987). It is therefore likely that this amplification of numbers is a trait strongly favoured by selection, but possibly constrained by a lack of sufficient genetic variation, or by the nature of the intermediate host. For instance, the arthropods used as intermediate hosts by many cestodes and acanthocephalans may not provide sufficient resources for asexual multiplication, or may not be able to survive it.

Another recurrent adaptation among trophically transmitted parasites is a truncation, either facultative or obligate, of the original multi-host cycle. Several lineages of trophically transmitted helminths drop one host, along with the trophic transmission stage, from their life cycle, presumably in response to the temporary or long-term shortage of a required host. Abbreviated life cycles have been reported in over 30 trematode families, and appear to be the product of at least 20 independent evolutionary events (Lefebvre and Poulin, 2005; Poulin and Cribb, 2002). Most often, they involve progenesis, or the precocious maturation of a juvenile stage inside the intermediate host that eliminates the need for trophic transmission to the usual definitive host (Lefebvre and Poulin, 2005; Poulin, 2003). Truncated life cycles have also been reported in cestodes (Andreassen et al., 2004; Poddubnaya et al., 2003) and in nematodes (Jackson et al., 1997; Levsen and Jakobsen, 2002), making it another good example of parallel adaptations in independent lineages.

By far, the best example of convergent adaptations among trophically transmitted parasites, however, is the evolution of host phenotype manipulation by these parasites. A large number of parasites transmitted trophically induce behavioural, physiological or morphological alterations in their intermediate host that render the latter more visible or otherwise susceptible to predation by definitive hosts (Moore, 2002; Poulin, 1995d, 2010; Thomas et al., 2005). The fitness benefits for the parasites are obvious: those that use manipulation achieve higher transmission rates and are thus favoured over those that do not. Theoretical analyses of the phenomenon indicate clearly that under most realistic circumstances, even if induction of changes in host phenotype is physiologically costly for the parasite, host manipulation should be favoured by selection (Parker et al., 2009; Poulin, 1994, 2007a). And, not surprisingly, it has emerged as a prominent adaptation in most major lineages of trophically transmitted parasites. Among acanthocephalans, the majority of, if not all, species tested to date appear capable of altering either the behaviour or coloration of their arthropod intermediate host in ways that have been shown or seem likely to increase predation by definitive hosts; this universal ability across members of the phylum indicates a unique ancestral origin inherited by all extant species (Moore, 1984). However, the patchy distribution of documented cases in cestodes, trematodes and nematodes across their phylogenetic trees strongly suggests multiple independent origins of manipulative abilities in these groups (Moore, 2002). Examples of host manipulation have also been reported in trophically transmitted protozoans (Berdoy et al., 2000; Hoogenboom and Dijkstra, 1987), to complete the list of major taxa using this parasitic strategy. Host manipulation is clearly a hallmark of trophic transmission, irrespective of the parasite taxon involved, and certainly one of its best-studied facets (Poulin, 2010). The physiological mechanisms through which parasites induce alterations in host phenotype, however, remain unknown in many cases (Lefèvre et al., 2009; Poulin, 2010). Nevertheless, since unrelated parasites often manipulate their hosts in the same way, that is, they induce the same behavioural changes in their respective host, they are likely to rely on the same physiological pathways since there are only a finite number of ways to induce photophilia or hyperactivity (Ponton et al., 2006). Convergence in manipulation of host phenotype may therefore be apparent at both proximate and ultimate levels.

The ability to manipulate host phenotype is not restricted to trophic transmission: it has also evolved repeatedly among vector-transmitted parasites. Although some of these parasites appear capable of manipulating the behaviour of their main host beyond mere pathology, that is, in ways likely to improve transmission (Day and Edman, 1983), it is normally the vector that is targeted. Typically, infected vectors display aberrant foraging behaviour, for instance high rates of probing on the host

skin or shorter feeding bouts per host visited (Moore, 1993). Once again convergence manifests itself since vector manipulation is known from all major groups of vector-transmitted parasites, that is, filaroid nematodes, protozoans and viruses (Moore, 1993).

Finally, host manipulation is also seen in parasitoids and castrators, and it is the outcome of separate evolutionary events among these kinds of parasites, too. Parasitoid wasps can be vulnerable to predators (or even other parasitoids!) following their emergence from their host. Numerous wasps can modify the behaviour of their host, either prior to emergence (Brodeur and McNeil, 1989; Eberhard, 2000; Fritz, 1982) or for a few days post-emergence before the host dies (Brodeur and Vet, 1994; Grosman et al., 2008), in ways that ensure the parasite's survival. Other types of parasitoids must emerge from their host, or release their propagules, in a specific microhabitat very distinct from the one where the host normally lives. Nematomorphs and mermithid nematodes, for instance, need to emerge from their terrestrial arthropod hosts into water or water-saturated substrate. Remarkably, in a perfect example of convergence, members of these totally unrelated taxa induce hydrophilia in their hosts, and force them to plunge in water or enter water-saturated soil moments before the worms emerge and kill their host (Hanelt et al., 2005; Maeyama et al., 1994; Poulin and Latham, 2002; Thomas et al., 2002; Vance, 1996). Altered microhabitat selection is also a feature of *Cordyceps* fungi and their relative, which cause infected insects to perch at specific heights and in specific positions on vegetation before they die and the fungi grow out of their cadavers (Andersen et al., 2009; Maitland, 1994). Less is known of what castrators do to their hosts, although there is evidence that both rhizocephalans (Høeg, 1995) and entoniscid isopods (e.g. Shimomura et al., 2005) usurp the parental care behaviour of their crustacean host, inducing the latter to 'fan' the parasite's eggs which occupy the space where the host's eggs would be if it were not castrated.

In summary, both life cycle patterns and specific adaptations associated with the life cycle show clear cases of convergence leading to a small number of combinations that correspond well with the six parasite strategies defined in this review. Other aspects of transmission biology could be added to the above list. For example, many parasites are capable of suppressing various components of the host's immune response (Boëte et al., 2004; Rigaud and Moret, 2003; Wikel, 1999). Also, certain parasites including cestodes and nematodes show remarkable plasticity in their adult sizes, in response to either host size or crowding by other parasites within the host; there can be 20- to 90-fold differences between the smallest and largest adult individuals in a population, a level of variation rarely seen among free-living organisms (Poulin, 1996, 2007a). However, not enough information is available at present to determine whether these

adaptations are characteristics of certain parasitic strategies and not others, or whether they occur independently of a parasite's strategy.

1.5. BODY SIZE AND VIRULENCE

Parasite body sizes, both absolute and relative to that of the host, and virulence are associated with the parasite classification scheme proposed by Kuris and Lafferty (2000) and Lafferty and Kuris (2002). Although this scheme is not strictly adhered to here, these features remain fundamental to our understanding of parasite strategies. If any proposed strategy is distinct from others, one would expect these important properties to fit accordingly. Thus, parasite-to-host size ratios for parasite taxa using a given strategy should not fall across the entire spectrum, but instead cluster within a relatively narrow band corresponding to an adaptive peak. The match between the six proposed parasite strategies and both parasite–host size ratios and virulence patterns is examined in this section.

1.5.1. Parasite–host size ratios

Along with their definitions of different types of parasitic strategies, Kuris and Lafferty (2000) and Lafferty and Kuris (2002) proposed that body size, or more precisely the relative size of parasite-to-host, is the key to understanding the evolution of these strategies. The body size of an animal scales with its energetic requirements (Gillooly et al., 2001), and should thus constrain the type of trophic interactions in which a parasite can engage with a host of a particular size. Lafferty and Kuris (2002) used energetic models to determine which parasite strategy would yield the highest conversion rates into offspring in different areas of the two-dimensional, host-size-by-parasite-size space. These models reveal that optimal strategies for a particular combination of host and parasite body mass fall into discrete adaptive peaks that depend on both relative and absolute body masses. For instance, the absolute size of the host can constrain parasitic strategies. Parasitoids rarely infect hosts weighing more than a few grams, perhaps because the slower growth and longer lifespan of larger hosts make the parasitoid strategy relatively inefficient compared to castration. In turn, castrators, although they infect a broader range of host sizes than parasitoids, are not found in hosts weighing over 1 kg (Kuris and Lafferty, 2000; Lafferty and Kuris, 2002). Larger hosts tend to invest relatively little in reproductive tissue (Brown et al., 1993), which would make parasitic castration inefficient.

As done by Kuris and Lafferty (2000), parasite-to-host size ratios for the six parasite strategies defined earlier were mapped along the

continuum of values seen in nature, which span eight orders of magnitude (Fig. 1.5). The data are based on educated guesses and also match those of Kuris and Lafferty (2000). Protozoan parasites were not included; their unicellular nature constrains the maximum sizes they can attain, and all values for these parasites would cluster on the left-hand side of the figure, whatever their parasitic strategy. Their size, like their ability to proliferate within a host, is a phylogenetic attribute and not one associated specifically with parasitism.

Some strategies display narrow distributions of parasite-to-host size ratios (Fig. 1.5). Metazoans transmitted by vectors, that is, filaroid nematodes, tend to be small relative to their host. Adult worms can attain substantial absolute sizes, but their hosts are large mammals, and thus their size relative to that of the host remains small (i.e. *Dirofilaria* in dogs; Nakagaki et al., 2007). Whatever the adult size, the larvae are physically constrained to be very small to allow ingestion by mosquito vectors. At the other end of the spectrum, both parasitoids and parasitic castrators tend to

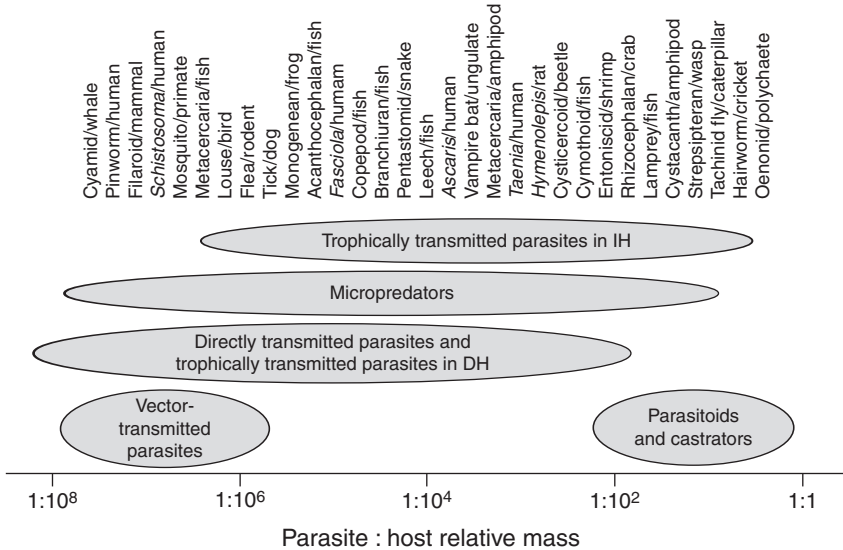

FIGURE 1.5 Approximate distribution (shaded ellipses) of parasite:host mass ratios for each parasitic strategies along the full range of observed values. Ratios of parasite-to-host masses are on a logarithmic scale along the horizontal axis; examples of actual associations between parasites and hosts are given above in their approximate location on the scale. Note that data include metazoan parasites only, that ranges for trophically transmitted parasites are shown separately for the definitive host (DH) and intermediate host (IH), and that the mass of vector-transmitted parasites is relative to their vertebrate host, and not to their micropredatory vector. Modified from Kuris and Lafferty (2000) and Lafferty and Kuris (2002).

be large relative to their host, some almost equalling their host in size. This is also true of the juvenile stages of trophically transmitted parasites that employ a castrating strategy in their intermediate host. Examples of these include the plerocercoids of the cestode *Schistocephalus solidus*, which can individually reach a mass >20% that of their fish host (e.g. Heins et al., 2002), and larval trematodes in their snail host, which as a result of their asexual multiplication, can account for 15–40% of the total tissue mass within the snail shell (Hechinger et al., 2009; Lafferty and Kuris, 2009).

However, directly transmitted parasites, trophically transmitted parasites (both compared to their intermediate and definitive hosts) and micropredators show a wide range of parasite-to-host size ratios, spanning several orders of magnitude (Fig. 1.5). In addition, ranges of parasite-to-host size ratios overlap extensively among these strategies so that relative size alone is a poor predictor of the parasitic strategy used by any given parasite. In many taxa using direct transmission, trophic transmission or micropredation, there is a positive interspecific correlation between parasite body size and host body size (i.e. Kirk, 1991; Morand et al., 1996, 2000; Poulin, 1996). In principle, this should limit variation in parasite-to-host size ratios within these strategies, and yet it does not. There is little doubt that the size of a parasite relative to its host can constrain what type of exploitation and transmission strategy will yield higher fitness, and body size may have tipped the balance one way or the other following a lineage's transition to a parasitic mode of life. However, except for parasitoids and castrators, body size alone cannot be used to distinguish the different types of host–parasite interactions.

1.5.2. Virulence as a function of intensity

From the moment parasites were first separated into micro- and macroparasites (Anderson and May, 1979; May and Anderson, 1979), the relationship between the intensity of infection and virulence has formed the basis of much of epidemiological theory. In this context, intensity refers to the number of individual parasites of the same species that invade and infect a host. In some cases, a single infection event by one parasite will cause the same fitness reduction in the host that would be incurred following multiple infections (intensity-independent virulence). In other cases, each additional infection by a new parasite has an additive effect up to some maximum fitness loss, though the relationship between fitness loss and infection intensity is not necessary linear (intensity-dependent virulence).

In parasitoids and parasitic castrators, virulence is more-or-less always intensity independent (Kuris, 1974; Kuris and Lafferty, 2000). One parasite is sufficient to cause host fitness to drop to about zero. In contrast, in micropredators, virulence is generally intensity dependent, if we exclude the possibility that the micropredator transmits a vector-

borne parasite to the host: thus, the more attacks on a host, the more blood or other tissues are removed, and the greater the impact on host fitness. However, the situation is not so clear for the other parasitic strategies, because the dependence of virulence on intensity boils down to whether or not the parasite can multiply within the host. With only a few exceptions, the ability to multiply within the host corresponds with the great divide between unicellular and multicellular parasites. Single-celled parasites like protozoans, bacteria and viruses almost invariably proliferate within a host following infection. It does not matter whether one, a few or several individuals of these parasites initially infect a host because the number within the host will quickly rise anyway, with maximum fitness impact. It is possible that genetically mixed infections, which did include more than one individual parasite at the start, can cause greater fitness reductions for the host (Read and Taylor, 2001); however, the additional loss of fitness is generally small compared to that caused by a single infection. In contrast, multicellular parasites like helminths or arthropods are almost invariably incapable of multiplying within their host; the exceptions are limited to a few taxa (hydatid cestodes, the monogeneans *Gyrodactylus* spp., lice). Unless they have very high *per capita* virulence, as a rule the more of these parasites accumulate in a host, the greater the resulting effect on host fitness.

The search for a correspondence between intensity-dependent or -independent virulence and distinct parasitic strategies is therefore confounded by phylogenetic constraints on whether or not parasites can multiply within their host. Certain generalities remain possible when only metazoan parasites are considered. In vector-transmitted filaroid nematodes, virulence is not only intensity dependent but also high compared to that of other nematodes parasitic in mammals. Presumably, the use of vectors uncouples parasite transmission success from host health and has loosened the constraints on the evolution of virulence (Ewald, 1983). In directly transmitted parasites, and in trophically transmitted parasites within their definitive host, virulence is generally low though still intensity dependent. However, in their intermediate host, trophically transmitted parasites are generally more virulent (Ewald, 1995). The slope of the relationship between virulence and intensity appears much steeper in intermediate hosts than in definitive hosts. Sometimes, one or very few parasites within an intermediate host are enough to cause severe reductions in fecundity (e.g. Rauque and Semenas, 2009), or marked alterations in behaviour which, although beneficial to the parasite by increasing the probability of predation by a definitive host, are nonetheless costly to the intermediate host (e.g. Poulin et al., 1992). Trophically transmitted parasites are larger compared to their intermediate host than to their definitive host (Fig. 1.5), and their higher virulence in the former might be the mere consequence of this size difference. Furthermore, because intermediate

hosts are often invertebrates while definitive hosts are vertebrates, differences in host immune mechanisms (e.g. encapsulation by melanising cells vs. antibody-mediated immunity) may also account for the different levels of virulence seen in the two types of hosts. Nevertheless, the possibility remains that parasite virulence is to a large extent the expression of a genetically determined aggressive exploitation of the host. For instance, selection may have favoured low virulence in the definitive host to prevent the evolution of avoidance of infected prey by predatory definitive hosts (Lafferty, 1992).

Therefore, even if the true causes remain unclear, different parasite strategies show some differences in their patterns of virulence. However, as with parasite-to-host size ratios, virulence patterns can only loosely be considered as characteristics of parasitic strategies. They are not inconsistent with the six adaptive peaks defined here, but they are not their strongest correlates.

1.6. CONVERGENCE AT POPULATION AND HIGHER LEVELS

Ecological phenomena at the population or community levels are not directly encoded in a species' genes like phenotypic traits. However, they can still show emergent patterns that are consequences of adaptive convergence in life history traits. Given any particular host population, the reproductive output, dispersal routes, host finding mechanisms, infection processes and other individual-level characteristics of parasites combine with environmental factors, stochastic effects and host defences to determine the overall abundance of the parasite population and its distribution among host individuals. We might expect the different strategies to achieve different distributions among hosts since they differ at so many levels.

Crofton (1971) was the first to suggest that an aggregated distribution among host individuals should be part of the definition of the parasitic mode of life. This type of distribution is characterised by most host individuals harbouring few or no parasites, and a few hosts harbouring high numbers of parasites. The processes that can lead to this sort of distribution include heterogeneity among hosts in both exposure and susceptibility to infection, as well as chance (Poulin, 2007a). If we exclude protozoans or other parasites that can multiply within their host, and if we ignore a few other exceptions, aggregated distributions are overwhelmingly the rule for directly transmitted parasites, trophically transmitted parasites in both the definitive and intermediate hosts, micropredators and vector-transmitted parasites (Shaw and Dobson, 1995). Typically, for these parasitic strategies, a negative binomial distribution provides a good fit to numbers of parasites per host. As these parasites have intensity-dependent effects on their hosts, this is one of the

few distributions that allow the long-term stability of both host and parasite populations via the increased death rate of heavily infected hosts (Anderson and May, 1978). In sharp contrast, the usual distribution of parasitoids and parasitic castrators is one in which most hosts in the population are uninfected, and the few that are almost invariably harbor one or very few parasites (Kuris, 1974). For any parasitic strategy, it is easy to imagine how other distributions could lead to the extinction of either host or parasite. Some form of higher-level selection must have favoured combinations of traits in individuals that are manifested by population parameters allowing persistence: parasite taxa that departed drastically from the distribution best-suited to the strategy they adopted have not survived to be sampled today.

At the community level, there may also be emergent features associated with the strategies of parasites, if whole community patterns reflect the sum of their parts. Following in the footsteps of studies on plant–pollinator interaction networks, recent analyses of host–parasite interaction networks have indicated that a regular and repeatable structure characterises systems involving different kinds of parasites. In an interaction network, a number of parasite species, p, exploits a number of host species, h, such that the total number of possible host–parasite associations is equal to ph; the actual number of observed associations, expressed as a proportion of ph, is the connectance of the network. In networks of fleas (micropredators) parasitic on mammals, and helminths (both directly and trophically transmitted parasites) parasitic on fish, connectance decreases identically and exponentially with the number of host species in the network (Mouillot et al., 2008; Poulin, 2007b). Some species are involved in many associations, others in few; the distributions of associations among host and parasite species in both the helminth-fish and flea-mammal networks show the same asymmetrical patterns, with locally abundant parasite species associated with proportionally more host species, and vice versa (Vázquez et al., 2005, 2007). The patterns obtained for helminth-fish networks are virtually indistinguishable from those obtained for flea-mammal networks, hinting at general features of host–parasite interaction webs that go beyond the parasites' strategy. Perhaps, properties of individual species do not translate into detectable features of entire communities. Alternatively, other facets of host–parasite networks may be determined by parasitic strategies, and further work may uncover them.

1.7. PARALLELS WITH PLANT PARASITES

Like animals, plants offer concentrated sources of nutrients and energy for parasites, but they also present distinct challenges and, as hosts, they are a completely different proposition. From an energetic perspective,

plants are very different kinds of resources than multicellular animals. For any given body mass, the metabolic rate of plants is lower than that of animals, even after correction for temperature effects (Brown et al., 2004; Gillooly et al., 2001). This should mean that less energy can be extracted per gram of host tissue and per unit time from a plant than from an animal. Also, plants are sessile, with tough cell walls as well as thick outer surfaces, and they have no easy point of entry like a mouth for internal parasites. Nevertheless, if the parasitic strategies defined above are indeed adaptive peaks reached by convergent evolutionary trajectories, we should expect to find roughly equivalent strategies among plant parasites.

Excluding a few carnivorous taxa, plants are autotrophs that do not feed on other organisms, which rules out parasites using trophic transmission (unless the plant is used as intermediate host). However, other strategies are well represented among parasites of plants. Many large groups within the insect order Hemiptera, such as aphids and leafhoppers, qualify as micropredators. These phytophagous insects feed on small amounts of sap or other tissue on one individual plant, and then move on to other plants (Kuris and Lafferty, 2000); their virulence is clearly intensity dependent, as one would expect from micropredators. They also serve as carriers of several vector-transmitted parasites including fungi, viruses and bacteria (Agrios, 1997; Dixon, 1998); these can multiply on a plant host, and have the same kind of intensity-independent effects on their host as unicellular parasites of animals transmitted by vectors. Other hemipterans behave as directly transmitted parasites. Scale insects, for instance, feed on sap drawn from a plant's vascular system, inducing intensity-dependent effects on the plant; females are permanently attached to the plant host, while the young they produce disperse to other plants (Edwards and Wratten, 1980). Other insects plaguing plants are reminiscent of parasitoids or castrators. Consider lepidopterans: an adult female lays eggs on a plant, these hatch into caterpillars that feed on leaf tissue while increasing several-fold in body size, and then pupate as they metamorphose into adults. This resembles the behaviour of several parasitoid wasps and flies that attack insects, except that in the case of lepidopterans, the host plant survives. However, in cases involving small plants, the host's survival is greatly at risk when many caterpillars feed on its tissues (Edwards and Wratten, 1980), a very parasitoid-like outcome. Certain fungi can also castrate their plant hosts and usurp the latter's reproductive structures for their own purposes (Roy, 1993), as the castrators described earlier do. These few examples show that the same parasitic strategies emerge, whether the host is a plant or an animal. Plants are not only parasitised by insects or fungi; the rest of this section examines two different groups of plant parasites to determine whether they also adopt the previously described strategies.

1.7.1. Plant–parasitic nematodes

Transitions to parasitism on plants have occurred at least three times in the phylogenetic history of nematodes, if not more (Blaxter et al., 1998). Plant–parasitic nematodes display a few morphological and biochemical adaptations necessary for the exploitation of plant hosts. For instance, all nematodes parasitic on plants have an oral stylet, a sort of hollow protrusible spear that serves to perforate plant cell walls, to inject secretions into the plant cytoplasm and to suck in the cytoplasm (Davis et al., 2004; Jasmer et al., 2003). Also, they possess cellulases, the digestive enzymes needed to break down plant cellulose; these were possibly acquired via lateral gene transfer from bacteria in the distant past (Dieterich and Sommer, 2009; Smant et al., 1998). After they enter the plant, often in the root tissues, some nematodes remain migratory, spending their lives moving through or between plant cells within the host (Moens and Perry, 2009); others, like the root-knot nematodes *Meloidogyne* spp., quickly become sedentary, inducing the drastic transformation of a host cell into an enlarged, multinucleate feeding cell in which the parasite remains for life (Davis et al., 2004; Dieterich and Sommer, 2009).

Recently, Dieterich and Sommer (2009) commented that plant–parasitic nematodes ''have almost nothing in common with animal parasites in terms of their parasitic lifestyle''. Jasmer et al. (2003), in contrast, found numerous similarities between the two groups of parasitic nematodes. I agree with the latter authors and feel that any differences lie mostly at a superficial, mechanistic level, and that at a strategic level, plant–parasitic nematodes are essentially directly transmitted parasites not really different from those exploiting animals. An individual plant–parasitic nematode invades one plant individual, stays there for life, reproduces and its progeny leave to infect other plants. Parasite-to-host size ratios for plant–parasitic nematodes clearly fall within the range seen for animal parasites (Fig. 1.5), since nematodes are invariably small whereas their plant hosts range from grasses to trees. The pathology caused to the host plant is clearly intensity dependent. These are clearly all attributes of directly transmitted parasites. Even several infection processes are reminiscent of what is observed in animal–parasitic nematodes. For instance, the penetration of the epidermal cell layer of plants by a combination of thrusting of the stylet combined with enzymatic secretions is not different from what hookworms do to penetrate mammalian skin (Jasmer et al., 2003). Also, the major cell modification induced by plant–parasitic nematodes to create both a home and a feeding structure for themselves is strikingly similar to the cellular rearrangement induced by animal–parasitic nematodes like *Trichiuris* spp. and especially *Trichinella spiralis*, the latter invading a muscle cell in its host and proceeding to restructure it completely (Davis et al., 2004; Despommier, 1993;

Jasmer et al., 2003). Both the animal–parasitic nematodes and their plant counterparts achieve this by manipulating gene expression in their host's cells (Davis et al., 2004; Jasmer et al., 2003; Niebel et al., 1994). Finally, not unlike many animal parasites facing the problem of encountering mates, plant–parasitic nematodes have evolved asexual forms of reproduction like parthenogenesis (Dieterich and Sommer, 2009). Thus, at proximate and ultimate levels, it can be argued that the adaptive peak corresponding to the direct-transmission strategy defined earlier and which was an attractor in the evolutionary landscape of animal parasites has been one too in the evolution of plant parasites.

1.7.2. Parasitic plants

There are over 4000 species of parasitic angiosperms (flowering plants) that derive sustenance from other plants, and these are spread across 19 families (Heide-Jørgensen, 2008; Press and Graves, 1995). Here again, parasitism has evolved repeatedly and independently. Parasitic plants range along a continuum from hemiparasites, like mistletoes, to holoparasites such as dodder, *Cuscuta* spp. Hemiparasites may be facultatively parasitic only; they derive water and minerals from their hosts, as well as nutrients, but have retained some of their photosynthetic ability. Holoparasites are obligate parasites that have lost all chlorophyll, and that cannot assimilate carbon and inorganic nitrogen on their own. Parasitic plants can also be divided based on their site of attachment to the host, which is either the stem or the roots. The distinguishing feature possessed by all parasitic plants is a modified root, the haustorium, which penetrates the host plant and connects to its vascular system; it serves for both attachment and feeding (Heide-Jørgensen, 2008; Stewart and Press, 1990).

Parasitic plants differ from animal parasites in some interesting ways. In both the present parasite classification scheme and in earlier ones (Kuris and Lafferty, 2000), the number of host individuals and host species attacked by an individual parasite during a particular life stage or throughout its life played a key role in the division of parasites into distinct strategies. Many parasitic plants, however, can do something that animal parasites cannot: one individual parasite can simultaneously parasitise several host individuals, not necessarily all of the same species (Pennings and Callaway, 2002). While at first this may appear like a major departure from the strategies outlined earlier, it is in fact a relatively trivial difference. As hosts, plants are sessile and, at canopy level, often in close proximity if not actual physical contact with their neighbours. If similar opportunities existed for animal parasites, surely they too would exploit more than one host at a time, without necessarily using a parasitic strategy different from the six described earlier.

On the whole, parasitic angiosperms fit well within the definition of directly transmitted parasites. An individual attaches to one plant host for life (though it may simultaneously draw nutrients from adjacent hosts starting later in its life). The parasite can manipulate the physiology and morphology of the host plant by interfering with growth hormone production (Pennings and Callaway, 2002; Stewart and Press, 1990), possibly to the parasite's benefit. Its seeds then proceed to infect new hosts, either by breaking their dormancy only in response to chemical cues from suitable hosts (not unlike the eggs of many animal–parasitic nematodes), or following germination, by growing towards a host (Pennings and Callaway, 2002). The impact on host fitness is often minimal when the parasite is small, but increases in an intensity-dependent fashion, except that here 'intensity' means the mass of the parasitic plant instead of the number of parasites (Marvier, 1996; Pennings and Callaway, 2002). Eventually, after it reaches a certain size relative to that of its host, the parasitic plant causes severe reductions in host growth and reproduction, and even occasionally host death (Pennings and Callaway, 2002; Press and Phoenix, 2005; Stewart and Press, 1990). Although these effects are reminiscent of those induced by parasitoids and parasitic castrators, they are truly those of directly transmitted parasites at high infection intensity. Host death is neither necessary for the success of the parasitic plant nor an inevitable outcome (as it is for parasitoids), and the reproductive tissues of the host plants are not specifically targeted (as they would be by a parasitic castrator). The indeterminate growth of parasitic plants just means that eventually they reach a biomass at which the amounts of resources diverted from the host are unsustainable for the latter. Parasitic angiosperms range in size from small herbaceous species to large trees, and so do their host plants (Heide-Jørgensen, 2008; Press and Graves, 1995; Stewart and Press, 1990). Various parasite–host combinations thus span the whole range of parasite-to-host size ratios seen for directly transmitted parasites (Fig. 1.5). The main difference between directly transmitted parasites of animals and parasitic plants is that, in the latter, growth of the parasite will almost invariably cause the severe fitness losses in the host that are only rarely achieved by the former.

There are, however, true parasitoids among plants that parasitise other plants. In many tropical forests, strangler figs (*Ficus* spp.) use a host tree for physical support; although they never draw nutrients from their host, the support they get from the host is an essential resource allowing them to germinate in the canopy, where they receive ample sunlight (Putz and Holbrook, 1989). They extend a network of roots down to the ground from the canopy, encircling the trunk of the host tree. As these roots grow thicker, they squeeze, or 'strangle', the host, cutting off nutrient flow along its vascular system, and eventually killing it. The figs do not benefit from host death, just as parasitoid wasps or

nematomorphs do not actually gain from death of their host; however, host death is an inevitable consequence of the exploitation strategy of all these parasites, uniting them as parasitoids. In the case of the fig-tree interaction, it takes years for the parasite to kill the host (Putz and Holbrook, 1989). This is simply due to the lower growth and metabolic rates of host and parasite compared to their animal counterparts: even though all happens on a different time scale, strangler figs are still parasitoids.

1.8. CONCLUSIONS

In one of his influential books, Gould (1989) proposed that evolution in general, including the rise of humans and their civilisation, is entirely dependent on historical accidents, or contingencies of evolution. Rewind and replay the tape of life on Earth, he said, and you would get a different scenario each time. Conway Morris (2003) countered with a plethora of examples of convergent evolution to support his view that independently of any historical accidents, something very much like humans would have evolved anyway, as a result of inevitable convergent paths of evolution. Conway Morris (2003) argued that convergence is a dominant pattern of evolution, with viable evolutionary paths being few and constrained. Evolution of certain biological properties is not only highly probable, but also highly predictable, even when initial conditions differ.

In this review, I have argued that rampant convergence has shaped parasite evolution. Parasitism has appeared well over 100 separate times, at least once in most phyla, and now accounts for close to half the species on Earth. Yet this vast diversity boils down to only six general parasitic strategies, each characterised by an almost predictable suite of traits. From distinct phylogenetic origins and with vastly different biological properties at the start, separate lineages of parasites have inevitably gravitated across the adaptive landscape towards one of six stable peaks, each characterised by a specific combination of life cycle and transmission mode, impact on host fitness, and a set of other traits ranging from relative body sizes to the ability to manipulate host phenotype. Some trait combinations are just not seen; for instance, reports of directly transmitted parasites that manipulate the behaviour of their host, of parasitoids that are three or four orders of magnitude smaller than their host or of trophically transmitted parasites that are highly virulent to their definitive host are either very rare or non-existent. It seems safe to speculate that if life exists on other planets, as long as alien ecosystems show some basic similarities to ours (i.e. universally shared biochemistry derived from a single origin of life, heterotrophy, finite resources, copying errors during reproduction allowing natural selection), then parasitism

will not only have evolved, but alien parasites would be using exploitation strategies very close to the ones defined here.

The recognition of parasitic strategies that transcend phylogenetic boundaries will hopefully help to tear down the traditional barriers between disciplines that have slowed down progress towards understanding of the fundamental biology of parasitism and diseases. Insect parasitoids have always been the domain of entomologists, plant parasites that of botanists, phytopathologists or agricultural scientists, whereas animal parasites have been the objects of study for ecological, veterinary and medical parasitologists. At a conceptual level, there are no significant differences between these organisms and between the types of interactions they engage in with their hosts. Cross-talk among these scientists is the key to progress in research on parasitism, and accepting the universality of a limited number of parasitic strategies independent of phylogeny should pave the way in that direction.

ACKNOWLEDGEMENTS

I thank Tommy Leung and Frédéric Thomas for comments on an earlier draft, and Juan Timi for his hospitality during my stay in Mar del Plata, Argentina, where this synthesis was written.

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