OFFPRINTS FROM COEVOLUTION OF PARASITIC ARTHROPODS AND MAMMALS
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Chapter 2

Coevolution as a Process

What Parasites of Animals and Plants Do Not Have in Common

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INTRODUCTION

I detest reading definitions, but we must begin with one. As used here, *coevolution* is the event where the members of one species select for a change in another species successfully and then in turn evolutionarily respond to that change. This is what was meant by Erlich and Raven's (1965) seminal paper on the subject, although their example of butterflies radiating onto host plant families is largely not an example of coevolution

(Janzen 1980, 1981). *Diffuse coevolution* is the same process, except that one or both species in the foregoing definition are replaced by a suite of species. Defined in this manner, the evolution of elaborate tarsi for holding onto the host's hairs is in itself explicitly *not* an example of coevolution (unless one wishes to argue that hair shape is an evolutionary attempt to make life difficult for the parasite). Likewise, a cataloging of the host specificity of Mallophaga is not in itself a coevolutionary study. In addition to requiring such a hardening up of the word *coevolution* to make it operational, my intent is to draw attention to the obvious fact that many of the tightly evolved relationships between parasites and hosts clearly display no evidence of complementary evolutionary change by the host, and furthermore, in many cases there is no theoretical reason to expect such change. The same applies to mutualisms such as seed dispersal and pollination by animals.

The assignment for this chapter was to discuss coevolution as a process with respect to mammalian ectoparasites. To examine the coevolutionary relationships between a pair of groups usefully, one needs to know who interacts with whom, what group 1 does to group 2, and how group 2 responds to group 1. There is already a very competent and respectable review literature telling us where we stand on these three points with mammalian ectoparasites (in addition to the chapters in the present volume, see Barbehenn 1978; Schad 1963; Catts 1982; Valdivieso and Tamsitt 1970; Freeland 1976; Mansour 1979; Kennedy 1975, 1976; Price 1975, 1977, 1980; Holmes and Price 1980; Brooks 1980; Kuris 1974; Wakelin 1979; Wikel and Allen 1976; Randolf 1979; Jackson 1969; Marshall 1981; Rothschild and Clay 1952; Nelson et al. 1975, 1977; Fain 1979; Johnston 1975). Perhaps the most distinctive trait of this literature is the imbalance in favor of knowing who parasitizes whom: more emphasis has been on the effect of parasites than on how hosts have evolutionarily responded to parasites. Likewise, there is a very large body of information on the ectoparasites of plants (e.g., see reviews in Rosenthal and Janzen 1979; Janzen 1973; Levin 1971, 1973, 1976a, b; Edmunds and Alstad 1978; Price 1975; Jermy 1976; Atsatt 1977; Strong and Levin 1979; Strong 1979; Dugdale 1977; Sutherland 1977; Russell 1977; Farrell 1977; Grandison 1977; Friend and Threlfall 1976; Vinson 1976; Clausen 1976; Niemela and Haukioja 1980; Bryant 1981; Gilbert 1977; van Emden and Way 1973; Southwood 1973; Haukioja and Niemela 1979; Powell 1980; Mound and Waloff 1978; Wallace and Mansell 1976; Atsatt and O'Dowd 1976). Price (1980) offers the first general review that is based on these two literatures, which have evolved at least 100 years with virtually no cross-fertilization.

Rather than to attempt a superficial regurgitation of the literature of mammalian ectoparasitology, my goal is here better served by comparing a caricature of the ectoparasites of mammals with the ectoparasites of plants, and in this manner underlining some of the ways parasites and hosts do and do not evolve or coevolve. Table 2.1 lists a few of the more glaring ways in which these two large groups of ectoparasites differ, emphasizing

Table 2.1 General Differences between Mammalian and Plant Ectoparasites

Mammal Ectoparasites	Plant Ectoparasites
1. Aside from host defenses, subject to virtually no predation and parasitization while on the host.	1. Subject to severe predation and parasitization while on the host.
2. Chemical content of food has relatively low variation among all mammal species.	2. Chemical content of food is enormously variable among all plant species.
3. If more than a small fraction of the living tissues of the host is removed, the host dies.	3. Very large fractions of host tissue may be removed without killing host.
4. Feeding on virtually any member of the host population is likely to generate selective pressure favoring traits that deter the parasite.	4. Many members of the population are genetically dead even if they continue to live and be food for parasites.
5. Close body contact with host is commonplace, such that the host can chemically identify the parasite.	5. Body contact with host is minimal, and where it occurs, opportunity to identify parasite is minimal.
6. Hosts contribute strongly to interhost movements by parasites.	6. At best, hosts play only a passive role (unavoidably produce distinctive chemical/odor fingerprints) in interhost movements by parasites.
7. Allospecific and conspecific individuals compete largely through the medium of the resource budget and immunological system.	7. Allospecific and conspecific individuals often compete directly by eating that portion of the plant some other parasite would have.
8. Commonly occur in large numbers on a host.	8. Rarely occur in large numbers on a host.
9. Spend most if not all their life on or very close to the host.	9. Spend major portions of their life at long distances from the host.

those that I suspect have resulted in substantial differences in evolution or coevolution.

PARASITES (PARASITOIDS) AND PREDATORS OF ECTOPARASITES

There appears to be virtually no predation or parasitoidization of mammalian ectoparasites while they are on the host other than that of the host itself as part of its own defense. To be sure there is the odd case of allo-

grooming by such things as oxpeckers on zebras, but by and large the fleas, for example, on rodents are not sought after by fur-gleaning birds, ants, or monkeys. Ectoparasites of mammals are not generally sought by ichneumonids, tachinids, braconids, chalcids, and so forth. This is in strong contrast to the very severe mortality bestowed on foliage-feeding insects by whole communities of birds, lizards, monkeys, parasitic wasps, ants, parasitic flies, viruses, fungi, bacteria, and other carnivores.

Surely then, one of the generally unappreciated advantages of evolutionarily moving from a scavenger, omnivore, or free-living carnivore diet to an ectoparasitic mode of life is increased freedom from generalist and many kinds of specialist carnivores. The close proximity to the host must often result in increased mortality, however, from a particular carnivore (the host). Such mortality has been responded to through the evolution of a variety of behavioral and morphological traits that are functional in avoiding predation from a specific animal. Since this evolution occurs on a specific animal with a vested interest in being able to prey on a very particular ectoparasite morphotype, there have been a few cases of what appear to be diffuse coevolution: grooming toe morphologies, social grooming, scratching behavior, itching physiology, and so forth. However, each of these traits is functional in other aspects of cleaning behavior as well, and, therefore, the selective pressure is extremely diffuse and certainly generated by more than just selection by the ectoparasites.

It is likewise conspicuous that mammalian ectoparasites are totally lacking in the complex of aposematic and mimicking insects so prominent among plant ectoparasites. There are several suspect causes. The diets of mammalian ectoparasites are not such as to preadapt the insect to an aposematic mode of existence, in contrast to plant tissue which may fill the gut of the most edible insect with the most inedible materials. Second, the ectoparasite is not under selection so much by visually orienting as tactile predators. Third, the predator of the ectoparasite is not so much after a meal as it wants to be lethal; even when a flea or tick is distasteful, it can be spit out, once killed.

It can be argued with respect to plants that one of the selective pressures for the production of odoriferously conspicuous secondary compounds, which are for the most part defensive chemicals, is that it makes the plant more olfactorily conspicuous to the hymenopterous and dipterous parasitoids that will search it for caterpillars. The possibility for this kind of coevolution simply does not appear to exist in the case of mammalian ectoparasites, unless various I-am-ready-to-be-groomed displays are analogous to such plant chemical signals.

The demography of animal populations such as ectoparasites that spend major portions of their lives in an environment with no threat of general predators or any kind of parasites is bound to be very different from that of free-living animals: hence, much tighter coevolutionary interactions between parasite and host should be possible in mammals and their ectopara-

sites than in plants and their ectoparasites. Mammalian ectoparasites that are developing coevolutionary (as well as evolutionary) interactions do not have a large variety of other carnivores selectively tugging in other directions. The distorted morphologies of mammalian ectoparasites, as well as those of other vertebrate ectoparasites, are undoubtedly in part the result of this relatively monolithic direction of selection. A caterpillar has to deal not only with the traits of its host, but also with avoiding birds, wasps, and ants—each of which calls for quite different escape behaviors and morphologies.

FOOD DIVERSITY FOR ECTOPARASITES

If a flea were to sample the blood of the 30 species of mammals in its habitat, the diversity of potentially dangerous chemicals it encounters in no way would equal the diversity of secondary compounds that would be encountered by a caterpillar sampling a random selection of the leaves of 30 species of plants. To be sure, there are parts of the plant (apical meristems, cambial cells, some phloem and xylem saps, ovules, seeds encased in very hard nuts) that may be relatively bland, and of course some species of plants have similar chemical defense characteristics (especially closely related ones). Likewise, hormone differences, antibody differences, sugar-titers, and other blood traits render each species of mammal's blood a different diet. However, as a general rule, a louse, tick, or flea making an evolutionary hop from one species to another, especially if within families or genera of mammals, will have proportionately fewer diet composition problems than will a caterpillar or sucking bug making the analogous hop across plant taxa.

A major aspect of the coevolution of host–parasite relationships in both mammals and plants is what the parasite does when a host mutant appears that drastically lowers the parasite's fitness. The more easily a mutant parasite can hop to another species of host on which it has a fitness even a bit higher than on the mutant host, the less likely the appearance of the initial mutant host is to generate an act of coevolution (once the parasite has made the hop to a new host, it no longer can coevolve with the old one). The more similar are hosts, the easier the hop. The question then becomes whether the mammalian barrier of mild food differences and strong behavioral/morphological differences, preening, itching, fur texture, and immune responses, are on the average equal to strong food chemical differences and weak morphological differences of plants (problems in caterpillar crypticity on the foliage of the new host, etc.). Although there are many cases in mammalian ectoparasites where a particular group seems to have been locked evolutionarily into one taxon, there are also many plant–insect pairs. Further, the widely held view that restriction of a distinctive parasite group to a particular host taxon means a long evolu-

tionary history is logically quite indefensible. Ectoparasites of plants have quite clearly explosively radiated across large sets of genera or species of hosts, and have probably done it over a time period so short that little or no change in the hosts need have taken place; there is no reason why the same could not have occurred with mammals and their hosts. It should be mentioned that such a radiation is certainly not evidence for coevolution, and in fact is unlikely to represent it. If a species of louse makes the evolutionary hop onto one member of a species-rich genus of rodents and then radiates onto all of them, generating a number of louse species along the way, it is precisely the failure of a host response that allows the louse mutant genotype to hop from mouse species to mouse species—presuming that the closely related mice have similar defenses. Were the mice to start coevolving with the lice, one very possible result would be the elimination of the lice or their restriction to only a few species of mice, the very case that is often thought of as not suggesting *coevolution*.

BITE SIZES

Plants wear their stomachs on the outside, have an enormous surface area to volume ratio for living tissue, and have enormous regenerative power when compared with mammals. Associated with these three traits in concert, plant ectoparasites can, and often do, periodically harvest enormous pulses of food from their hosts, followed by a period when the host recovers while being subjected to little or no parasitism. Plants can sustain ectoparasite species that for seasonal, predatory, or other reasons require periodic peaks of density far greater than could be sustained by the host on a full-time basis. When a caterpillar eats all the leaves off its host plant and then disappears for a year, it has been allowed a life-style equivalent to a population of fleas that thoroughly exsanguinated most or all of the rabbits in a habitat at 12-month intervals. Of course, there are occasional events where a mammalian ectoparasite builds up to a level where it kills the host, but even here the amount of tissue that has been removed is quite small compared with what a population of defoliating caterpillars removes. Furthermore, such an event is generally not viewed as the normal form of the parasite–host interaction.

It is tempting to suspect that the amount of tissue grazed off by mammalian ectoparasites has been evolutionarily or coevolutionarily adjusted downward because these ectoparasites are much more dependent on the host for habitat than are plant ectoparasites. However, an examination of that hypothesis is greatly confounded by the much superior repair and regeneration properties of plants. It is also confounded by the fact that a gram of material removed by a mammalian ectoparasite is of considerably greater usable value than is a gram of material removed by a plant ecto-

parasite, with the obvious exceptions of mallophaga on the one hand, and specialists on cambial tissue and ovaries on the other hand.

It is easy to suspect that the evolution of strong regenerative powers in plants came about as one of a variety of defensive responses to herbivores. It is hard to imagine how herbivores have evolved into taking amounts of food that do not push the plant out of the habitat; in fact, there is considerable suspicion that herbivores do push this or that species of plant out of a habitat, or substantially alter its density. Coevolution does not readily fall out of the interaction. With mammalian ectoparasites, however, it is easy to see how selection could have resulted in moderation of the amount of tissue removed since so much of the life of the ectoparasite is spent on the host. If there are microdemes of parasites on each individual host, there may even have been a form of group selection possible. However, it is much less clear whether mammals have evolved regenerative powers in direct response to the usual damage done by an ectoparasite. When a warble fly larva exits from the usual host, the wound normally closes cleanly, whereas it may be purulent when the larva exits from an artificial host. But is this because the usual host has evolved such a response or because the antibiotics, anesthetics, and other tissue-altering drugs released by the fly larva were simply not evolutionarily fine-tuned to the biochemistry of the artificial host?

STRUCTURED HOST POPULATION

If a mammalian ectoparasite depresses its host's fitness in any way, it is not hard to imagine that there will be selection for traits to eliminate the parasite, but whether the selection will be manifest in a change in the genotype is quite another question. But what about the hosts that would have died, for example, before reaching reproductive status through mortality factors quite unrelated to the presence of the ectoparasite? If a usual level of fleas does not influence a vole's chances of being taken by a fox before the age at which the vole would have been reproductive were it not to have fleas, have the fleas on that vole gotten a selective "free ride" from each fox-eaten vole? In other words, is the vole population, when under heavy predation by foxes, supporting a major flea population that is then not selectively felt by the voles? This weird-looking question derives directly from a consideration of how plant ectoparasites may interact with their hosts.

The preceding question can be answered in the affirmative *if* the ectoparasites distribute themselves on the host population in partial or total response to the eventual fate of the host. The members of a plant population, excluding seeds for the moment, commonly can be partitioned into two groups. There are those seedlings and saplings that are growing in

sites where they have some chance of becoming adults. There are also those seedlings and saplings that are growing in sites in which they are guaranteed never to become reproductive; that is to say, they are evolutionarily dead, irrespective of how green they are. The most extreme case of this would be a tree seedling growing in the dim light of a cave where its seed was dropped by a frugivorous bat. A more commonplace case is that of seedlings and saplings growing directly below the crown of a middle-aged parent tree, where the young plants must attain the canopy or die in a period much shorter than the usual remaining life span of the parent. Here, if the ecological herbivore is a species that specializes on such plants, it becomes an evolutionary detritivore.

The key point is whether the ectoparasite is wholely or largely restricted to the plants that are doomed to die. The evolution of such a feeding preference is easy to visualize. A herbivore immigrates into a new habitat. Its traits cause it quite serendipitously to use that portion of the plant population growing in very heavy shade rather than the portion of the plant population growing in tree falls, edges, creek banks, and so forth. Such a herbivore is much less likely to select for an evolved defense response by the plant population than is the herbivore that initially moves onto the members of the plant population with a high chance of surviving to reproductive status. Additionally, the herbivore need not have come from some other habitat but could also have appeared through an evolutionary move from some other host species. The final outcome of such a process should be the accumulation of a higher equilibrium density of species on those portions of the plant population that are doomed than on those that are not. That is to say, it is the *lack* of a coevolutionary response by the plant that may result in this pattern.

The question now becomes one of whether structure such as that described for plants can be recognized in the interactions of mammals with their ectoparasites. The key data are those telling us how ectoparasites are distributed among the members of a mammalian population and the environmental processes that maintain that distribution. Since most mammals can move about, it may well be that *all* members of the population have some chance of survival to reproductive status, and therefore the scenario for plants cannot be reasonably applied to animals. We need studies of how parasite individuals are distributed over the various fate-classes of their hosts.

HOST RECOGNITION OF ECTOPARASITES

Although the array of host-generated facultative immune responses does not seem to be as spectacular with mammalian ectoparasites as with mammal endoparasites, the long periods of intimate body contact and the feed-

ing mode of mammal ectoparasites make it possible quite often for the host to identify the ectoparasite chemically and therefore respond specifically to it. The chances for coevolution are great. Inflammation from chiggers on humans and the lack of inflammation on usual hosts provide a familiar example. How long a mosquito can feed without itching is no accident, although one wonders to what degree the mammal host has evolved, if at all, toward adjusting its sensitivity to mosquito fluids. It is not surprising to find that the chemical defenses of mammals against ectoparasites are often quite parasite specific; the specificity of the immune system is perhaps its greatest structural difference from the chemical defense systems of plants, be they standing or facultative.

Ectoparasites of plants, however, by and large feed in such a manner that there is only a minimal chance of the plant knowing much more than that it had its leaf eaten off. When the ectoparasite is very sedentary (e.g., scale insects, mealybugs), then there is the chance of internal chemical changes that begin to approximate mammalian immune responses. However, such cases are both rare in species and rare in individual cases when compared with the great amount of foliage browsing and sapsucking done by more mobile herbivores. The only way a plant can "know" who is eating its leaves is through the evolutionary form of learning whereby most of the herbivory committed during previous generations was carried out by that set of specialist herbivores, as well as generalists, that have to some degree breached the chemical defenses of the plant. That is to say, whoever is eating you today is likely to be who was eating you yesterday. When one considers that most plant species have very large suites of herbivore species that feed on them, the possibility of species-level recognition is even more distant.

It should be cautioned that plants do have a variety of facultative defenses that are turned on when tissues are eaten and damaged. And in some cases, the level or kind of chemistry may vary depending on whether it is a scissors or a cow or beetle that dripped its saliva on the leaf when eating that leaf. Nevertheless, there appears to be nothing that approximates the very chemical specificity invented by the mammalian immune response. Why has this system not diffusely coevolved in plants? I suspect that a major reason is that caterpillars, as well as many other kinds of herbivores, feed on a plant for a few weeks or months and then leave for a variety of reasons quite independent of the plant–ectoparasite interaction, for example, bad weather, lack of time for another generation, predator/parasite buildup, and so forth. Furthermore, a plant that is heavily damaged at one time is quite likely to be heavily damaged again by a quite different species of insect, and it may be many years before the first species again commits severe herbivory to that species. Finally, there is enormous heterogeneity in a plant population as to which individuals are fed on in a given year, a heterogeneity that is driven by many more factors than just

the chemistry of the foliage. In strong contrast, a mite population that is not depressed or slowed by some sort of active immune or other response by its host mammal is likely to increase to lethal or severely debilitating levels and to spread thoroughly through the remainder of the mammalian population as well. The very traits that make a mammal different from a plant, such as endothermy, high edibility, and active microenvironment modifiers, create a high-quality parasite/predator-free microenvironment for the exploding mite population. In short, mammals have the environment much less on their side than do plants when it comes to dealing with ectoparasites, and they make up for it with their immune system and behavior. It is hard to coevolve a fine watch if God keeps pouring sand into the works.

HOSTS DISPERSE ECTOPARASITES OF MAMMALS

Although a mammalian ectoparasite is capable of movements between hosts of a few centimeters to meters, or of sitting in one place until a host passes by, quite analogous to a caterpillar, it generally lacks the movement abilities of the winged phase of plant ectoparasites. Of course, some parasitic Diptera have wings and plant mites lack them, but, in general, mammalian ectoparasites depend on their hosts for geographic displacement, and plant ectoparasites both search actively through the habitat and move readily across a wide variety of nonhabitats. Clearly, plant ectoparasites have a much higher chance of being panmictic within the general habitat they occupy while mammalian ectoparasites have a much higher chance of existing in microdemes at the level of nests, individual animals, family groups, and so forth. On the other hand, if there are detriments to decreased outcrossing, mammalian ectoparasites are more likely to suffer them than are plant ectoparasites. One wonders why gravid ticks are so eager to abandon house and home!

As briefly mentioned earlier, this means that mammalian ectoparasites have a greater chance to proceed with (co)evolutionary changes through not only the usual kind of selection, but also through some sort of group selection, where the monospecific faunulet of a given host is the unit of selection. This would occur in plants only where the ectoparasites are exceptionally sedentary like some mites, scale insects, and mealybugs, and it would be helped along by having many of the plants in the population so well defended that they were totally unacceptable, thereby rendering the acceptable individuals even more insular. I suspect that any process that tightens up the ecological interaction between host and ectoparasite raises the chance of coevolutionary processes.

In quite a different vein, the adult stage of most herbivores is a specialist at locating hosts or host parts that are widely and often cryptically scat-

tered in space and time. This results in two somewhat different lineages of evolved or coevolved interactions. A plant may have one set of chemical interactions with the caterpillars, once there, and quite a different set of chemical interactions with the ovipositing moth. The very chemicals that render a plant inedible to a large suite of caterpillars may be the unavoidably conspicuous olfactory cues that the ovipositing adult can use to find a rare host plant. Such conflicting effects of the same trait can be very disruptive to the evolution of the one-on-one interactions characteristic of coevolved systems. Perhaps the mammalian analogue would be that the very social proximity that leads to social grooming likewise leads to rapid and thorough spread of ectoparasites among the members of a social group. On the other hand, a mother mammal's ectoparasites are given to offspring about as thoroughly as are the mother's genes; plants start their independent life quite clean. One of the prices paid by juvenile mammals for the milk subsidy is a healthy dose of the mother's ectoparasites, yet they may gain from the opportunity for highly evolved or coevolved interactions that come about through the fidelity and omnipresence of the inoculation.

COMPETING THROUGH THE RESOURCE BUDGET

It is quite evident that mammalian parasites compete with each other through the medium of the resource budget of the host and that they induce immune responses that exclude other parasites. Plant ectoparasites do the same, and likewise in both evolutionary and contemporary time scales, though the facultative chemical responses by plants do not begin to have the specificity and cross-immunity traits of mammals. However, plant ectoparasites also compete inter- and intraspecifically by physically removing the food that would have been eaten by some other herbivore. When a leafcutter ant colony strips the leaves off a tree, it does not spare those with moth eggs or caterpillars and does not leave behind enough food for those larvae to complete their development. When a large number of gypsy moth larvae defoliate a tree, the first to get the leaves do not spare leaves for latecomers. A large caterpillar eating a leaf is likely to consume the young immatures of other insects or physically force them off the leaf.

I suspect that this very direct style of competition is generally missing from mammalian ectoparasite interactions. Feeding fleas and mosquitoes do not generally shoulder each other out of the way or suck the well dry, though physical space may be limiting for mammalian ectoparasites that densely fill small spaces. The consequence should be that three-way interactions between two ectoparasites and a mammalian host are less likely to be focused on the details of the two parasites' traits than is the case with evolved or coevolved three-way interactions between two ectoparasites

and a plant host. For example, the timing and location of oviposition by a moth may well be determined by having to wait to see where some other species of moth is taking its bite out of the plant population before being able to determine the best place to lay its eggs.

LARGE NUMBERS PER HOST

The standing crop of ectoparasites on a mammal is commonly numerically very high. Another way to put it is that mammalian ectoparasite populations tend to be made of very many small individuals rather than a few large ones; this is a way to evolutionarily sneak a lion into a mouse colony, and it can be done by selection for individuals small enough to escape the search behavior of the host. To put it another way, if you are going to be as large as a flea, then you cannot ride around by the hundreds clinging to mouse hairs.

With the exception of sometimes being more desirable or conspicuous to foliage-gleaning predators, an increase in size per se is generally not as directly dangerous to a plant ectoparasite. This means that more morphological options are open to the plant ectoparasite in evolving or coevolving its interactions with the host and its environment. In some cases, such as 10–20-g moth caterpillars and leaf-eating beetles, it is clear that selection has pushed the system very far in the a-few-large-individuals direction. On the other hand, there are a few cases where plant ectoparasites have moved in the other direction, with aphids being the most omnipresent, at least in extra-tropical habitats, and conspicuous. A beaver ear stuffed with mites bears a decided resemblance to a cherry inflorescence stuffed with aphids. There are two conspicuous differences, however. First, there are no ants tending the mites in the beaver ear. Second, the mites need not have been produced parthenogenetically, while the aphids were. Aphids are specialists at moving onto a temporarily abundant poorly defended food source and strongly subdividing themselves, with the pieces suffering high carnivory but the whole organism having a very high survivorship once the parthenogenetic beast has had a bit of time to get the initial subdivision well under way. The degree to which they are analogous to the large populations of mammalian ectoparasites sustained by a single mammal depends on the degree to which the mammalian ectoparasites are the parthenogenetically produced offspring of a few initial colonizers. Aphids are well known to have evolved many specific chemical and behavioral adaptations that fine-tune them to the biology of particular species of host plants. However, the degree to which their hosts have responded specifically to aphids with complementary traits is largely unknown. Even gall forming by aphids may be nothing more than a purely one-way manipulation of the host plant's biochemistry by aphid-released hormones and

other signals, and therefore not coevolve at all. Incidentally, it is striking that mammalian ectoparasites are not gall formers, unless warble fly warbles and delayed itching of mosquito bites may be viewed as a very crude beginning, whereas this trait has evolved numerous times in all the major taxa of arthropodian ectoparasites of plants.

ALL OF LIFE TIED TO HOST

As has been obliquely referred to on several previous occasions, the great part of the life cycle of mammalian ectoparasites is spent on or very near to the host. Ticks are the only major exception, but this exception is confounded by the observation that ticks are so willing to leave hosts, only to climb right back on another, that it is as if there is some extrinsic value to exchanging hosts and the time spent off the host is an unavoidable by-product of this exchange. Plant ectoparasites normally spend only one portion of the life cycle on the host. Full-grown caterpillars so commonly actively vacate the area of the host that the location of the pupa is almost guaranteed *not* to be the host individual or species. Adult herbivorous insects more often feed on different hosts or host parts than do juveniles. Adult holometabolous insects commonly have nothing to do with larval hosts except as an oviposition site. For major seasons of the year the feeding stages of a plant ectoparasite may well be totally missing from the habitat, even when the plant is a growing and apparently resource-rich substrate.

The result is that the density of feeding ectoparasites to arrive at a host may be largely or entirely determined by density-dependent and density-independent mortality processes connected in no direct manner with the biology of the host. I am sure that this fact goes a long way toward disrupting parasite–plant evolving or coevolving interactions as they begin. If the number of leaves you lose to a caterpillar is determined not so much by how your leaves taste as by whether there happened to be the right species of nectar-bearing flowers seven months ago in a different habitat, and next year it is determined by whether some other species of caterpillar suffered a disease epidemic to which your caterpillar is also susceptible, then evolutionary, to say nothing of coevolutionary, processes have a tough time maintaining the linkage they need to persist.

CONCLUSION

What we know of mammalian ectoparasites derives from medical and taxonomic interests. What we know of plant ectoparasites derives from agricultural and taxonomic interests. The medical world has by and large

been more fascinated with the biochemistry of the interaction than has the agricultural world, probably because of the relative different net worths to humans of the individual patients. We now need, and see appearing on the horizon, extensive inquiries into the ecology of the interaction itself in the context of the habitats in which it evolved, coupled with detailed observations of what hosts do when ectoparasites are feeding on them. Stating that the host is sick, dead, or debilitated is not a detailed observation. *All* ectoparasitism has a cost, otherwise the ectoparasites are simply detritivores. The question is one of how this cost compares with the budgetary and resource noise in the system and with the cost of eliminating that ectoparasitism, and whether the possible defenses are compatible with other traits of the animal. The identification of the relative role of evolution versus coevolution in such an inquiry is trivial. Of much greater importance is the now well-established understanding that both members of any interaction are potential evolvers, and that neither evolves solely in response to the selective pressures of the other.

SUMMARY

Although ectoparasites of mammals and of plants have a great deal in common, they also differ in many aspects of their ecology, population biology, potential for evolutionary and coevolutionary change, and host interactions. These differences range from the nearly complete lack of predators and parasitoids of mammalian ectoparasites to the possibility that plants may sustain large ectoparasite populations with little or no selective effect owing to the way plant populations are structured.

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