

HOST CONTROL OF SYMBIONT TRANSMISSION: THE SEPARATION OF SYMBIONTS INTO GERM AND SOMA

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Abstract.—Obligate, vertically transmitted symbionts occur in many species. Hosts often have elaborate developmental processes and specialized organs to control the reproduction and transmission of their symbionts. Control mechanisms divide the symbionts into reproductive, germline lineages and nonreproductive, somatic lineages. This germ-soma distinction favors reduced competition among symbionts and fewer virulent effects on the host. Observations suggest a repeated evolutionary trend toward host control of symbionts and division of symbionts into germ and soma. Theory predicts that host control evolves only in response to particular mechanisms of symbiont competition, for example, when symbionts disrupt host development.

Many symbionts have a beneficial effect on their host. For example, leeches and lice cannot survive on their blood diet without bacterial symbionts, many sea organisms have complex organs to house luminescent bacteria, and insects that feed on cellulose or plant sap rely on a variety of bacterial partners (Buchner 1965). Although the symbionts provide benefits to the host, those benefits may be partly offset by the disruptive, virulent effects of competition among symbionts.

Hosts often appear to control competition by limiting the reproductive opportunities of the symbionts. I argue that these host-imposed limits separate the symbionts into transmissible, germline lineages and nonreproductive, somatic lineages.

I develop the argument in five parts. First, I summarize the theory of host-symbiont conflict. The theory explains why symbiont competition tends to disrupt the host and therefore why the hosts gain from controlling the reproductive opportunities of the symbionts. I relate theories about symbiont competition to Buss's (1987) argument that competition among cell lineages explains the origin of the germ-soma distinction in metazoans.

In the second section, I describe examples of host-symbiont natural history. This section is taken up with biological details of symbiont transmission and host development. The observations support my claim that symbiont lineages are often separated into germ and soma.

I develop new theory about host-symbiont conflict in the third section. One interesting conclusion is that the average fitness of a host population generally increases by imposing germ-soma differentiation on the symbionts, but host al-

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les that cause such traits do not necessarily increase in frequency. This conclusion is similar to a result from the theory of genomic conflict: mean fitness of hosts generally increases by imposing uniparental inheritance of cytoplasmic symbionts, but host alleles that prevent cytoplasmic mixing spread only when they are associated with an immediate reduction in cytoplasmic virulence (Hoekstra 1987 and Frank 1996a discuss uniparental inheritance of symbionts).

This theory focuses attention on host characters that, by controlling symbionts, cause an immediate increase in host fitness. I argue that limiting symbiont disruption of host development may be particularly important. In the fourth section, I summarize observations showing that symbionts often disrupt host development.

In the final section, I consider the types of comparative and experimental data that could be gathered to learn more about host-symbiont interactions and the evolutionary processes that favor differentiation of germ and soma.

THEORETICAL BACKGROUND

The evolution of within-host competition among symbionts is an example of the general problems of kin selection and group selection (Hamilton 1972; Bremermann and Pickering 1983; Frank 1996b). Each genotype gains within the group by outcompeting its neighbors but loses as the overall efficiency of the group declines (virulence increases). The tension is resolved according to the distribution of genetic variance. Relatively more variance within groups of symbionts favors more intense competition, whereas relatively more variance among groups favors greater cooperation within groups and lower virulence. The problem can be described equivalently in terms of kin selection. The coefficient of relatedness is the ratio of variance among groups to total variance in the population. As the relative variance among groups rises, relatedness and competition increase among neighboring symbionts.

Any problem of within-group competition among genotypes can be viewed as a problem of the evolution of virulence. For example, Buss (1987) used a group selection argument to suggest that cell lineages within a metazoan compete in ways that lower individual fitness. In this case an "individual" is really a group of cell lineages. A lineage gains a reproductive benefit relative to its neighbors by increasing its probability of contributing to the germ tissue. The intensity of competition and the potential for reducing individual fitness depend on the genetic variation among cell lineages.

One way to control renegade cell lineages is with "policing" traits that enforce a germ-soma split early in development. This split prevents reproductive bias among lineages during subsequent development. Once the potential for bias has been restricted, a cell lineage can improve its own fitness only by increasing the fitness of the individual.

Maynard Smith (1988) agreed with Buss's (1987) logic about the potential for cell lineage competition, but he argued that metazoans solved their problems of cell lineage competition by passing through a single-celled stage in each generation. When an individual develops from a single cell, all variation among subsequent cell lineages must arise by de novo mutation. In Maynard Smith's view,

such mutations must be sufficiently rare that the genetic relatedness among cells is essentially perfect. Thus, the soma sacrifice reproduction as a natural, altruistic act in favor of their genetically identical germline neighbors. Buss recognized the importance of de novo mutations within an individual but argued that these would be sufficiently common to favor significant cell lineage competition and policing.

Cell lineages of symbionts face a similar tension between competition within hosts and the overall success of their host. However, two additional factors influence symbionts when compared with metazoan cell lineages. First, when symbionts are transmitted vertically, from parent to offspring of the host, hundreds or thousands of symbiont cells typically infect each offspring. This large founding population contrasts with the single-celled bottleneck typical of a newborn metazoan. Thus, a host develops with a potentially diverse set of symbionts. Second, symbionts may infect a host horizontally, from another host individual or from the environment. Such mixing of symbiont lineages greatly decreases relatedness within hosts and favors within-host competition. Selection will always favor the symbionts to transmit partly by horizontal routes to avoid competition against relatives within the host (the dispersal effect of Hamilton and May 1977, applied to symbionts in Frank 1994).

In summary, metazoans appear to maintain cooperation among cell lineages by passing through a single-celled bottleneck in each generation. By contrast, symbiont lineages have greater potential for diversity within hosts and require other mechanisms of control. This leads to my argument that hosts often impose a germ-soma split on their vertically transmitted symbiotic lineages. This split prevents reproductive bias and reduces opportunities for virulent competition among symbionts.

BIOLOGICAL EXAMPLES

Buchner (1965) describes hundreds of cases in which the interwoven lives of two organisms proceed through embryogenesis, metabolism, and transmission. A few examples must serve to illustrate aspects of host control, opportunities for symbiont competition, the number of symbionts transmitted, and germ-soma differentiation among symbionts. (All descriptions and page numbers that follow are from Buchner 1965.)

The logic of host-symbiont conflict is sufficiently clear that some of the following details may seem unnecessary for the general argument. Indeed, the theoretically inclined reader may wish to skip ahead to the next section in which the theoretical argument is extended in new ways. However, progress on this question will ultimately begin with a careful reading of the huge and confusing literature on symbiosis. The main value of the present article is to bring some order to this mass of observational detail.

Vertical Transmission of Symbionts

The examples in this section illustrate mechanisms of symbiont transmission. In the second example, the hosts appear to separate symbionts into transmissible, germline lineages and nontransmissible, somatic lineages.

Many lice require bacterial symbionts to provide essential vitamins and supplement the normal diet of vertebrate blood (pp. 482–507, 771–772). Mycetocytes, host cells that house populations of intracellular bacteria, group together into a mycetome that sharply constricts the stomach. These symbionts are transmitted to progeny via infection of special organs between the ovaries and the oviducts, the ovariole ampullae. The method of infection differs widely among species. In *Pediculus*, changes in the gut mycetome begin with the approach of the third molt. “The symbionts swarm in thick clumps . . . and glide backward on the ventral side of the stomach. It is not clear just how this movement takes place, for motility has never been observed in the symbionts. At any rate, the latter are not scattered throughout the body cavity but advance toward the ampullar rudiment only” (pp. 487–488).

When the eggs develop to a certain stage, some of the symbionts, joined in vacuoles, transfer from the nearby ampulla to the base of the egg. “From here the bacteria glide into a depression in the oocytes and, when the latter are sufficiently enriched, they close behind the symbionts” (p. 489). This migration happens only in the female. In males the gut mycetome begins to degenerate at the same stage. Although the mycetome and symbionts generally do not disappear altogether, the remaining structure and bacterial population are usually much reduced.

A different cycle occurs in *Haematopinus*, in which the symbionts are separated into transmissible and somatic forms during early development. In the female embryos the mycetocytes separate into two groups. One set migrates to the midgut and forms the gut organ that presumably contributes essential vitamins to the host. The other set moves under the dorsal (upper) surface and forms storage aggregations that hang down into the body cavity. As the third larval molt begins, these storage mycetomes degenerate, releasing their symbionts into the molting fluid between the old and new cuticle. “Now the only invasion place in this stage of development takes form in a wide opening of the genital rudiment. Strange, fibrous, conducting cells sprouting at the distal end of the rudiment are set off sharply from their more deeply staining surroundings; the symbionts glide with the molting fluid to that opening, apparently very rapidly, pass into it, rise to the fiber cells, and are soon appropriated by them. . . . [E]ach of the ovarioles differentiates a second group of paler prehensile cells which are extended like hands toward the newcomers” (pp. 494–496). The symbionts are moved into the ovariole ampullae, an infected cushion of cells that is pushed between the ovarioles and the oviduct. Each egg is then infected as it develops.

Number of Symbionts Transferred to Offspring

The tendency for competitiveness and virulence increases as relatedness among genetic elements declines within a host. This may be the reason the metazoans pass through a single-celled stage in each generation within the formation of a fertilized egg (Maynard Smith 1988). The genetic bottleneck caused by single-celled reproduction means that differences among cells arise from de novo mutations during development. The high relatedness among cells promotes cooperation.

A similar problem arises when we consider the number of vertically transmitted

symbionts that colonize an offspring. If only one symbiont is transmitted, then relatedness among symbionts will be high, with all differences arising from de novo mutations within the host. In general, the relatedness among vertically transmitted symbionts decreases with a rise in the number of colonists in each generation and an increase in the mutation rate of the relevant life-history characters (Szathmáry and Demeter 1987; Frank 1994). There can be a surprisingly strong tendency for conflict even in vertically transmitted symbionts.

It is difficult to find data for the number of symbionts transferred. A few anecdotes from Buchner (1965) will at least suggest the kinds of transmission patterns that can occur.

In *Pediculus*, discussed earlier, the ovarial ampullae contain 3,000–6,000 symbionts. Approximately five eggs are produced daily, with 150–250 symbionts transferred to each egg (p. 689). The number of symbionts in the ampullae apparently does not decline through this process, suggesting active division.

In the drugstore beetle *Sitodrepa panicea*, symbiotic yeasts are smeared on the eggs during oviposition. The hatching larva devours much of the egg shell and ingests many of the yeast cells. The yeast reproduce rapidly in the gut, but only a few are admitted into the specialized gut organs that house the symbionts during the larval period. The few successful colonists are probably the only yeasts that will survive in the host. Thus, only a limited number of yeasts are transmitted to the offspring (pp. 119–124).

Symbionts are frequently kept in organs located near the gut. This is probably for metabolic reasons. Some of the symbionts must also be transferred to offspring. One pathway between gut and reproductive tissue is provided by migrating host cells that contain symbionts (mycetocytes). The reproductive organs develop uninfected. At a later stage some symbionts are moved to the receptive eggs. For example, in aleurodids (homopteran whiteflies), whole mycetocytes, situated at normal sites between fat cells, transfer to the ovarioles after the third molt. The mycetocytes degenerate, releasing numerous symbionts that gather in a groove at the posterior egg pole (p. 649). In some cases whole mycetocytes are transferred to the egg—a sort of somatic fertilization (pp. 268, 649, 656). These fertilizing mycetocytes often originate within larger mycetomes. A limited subset of the symbiont population is loaded into the mycetocyte. The remaining symbionts are not transmitted to eggs but often continue to appear healthy, perhaps continuing with somatic duties.

Germ-Soma Distinction among Symbionts

The distinction between reproductive and nonreproductive symbionts happens rather late, when transmitting mycetocytes load some symbionts for movement to the egg. In many cases this distinction between “somatic” symbionts and “germline” symbionts happens earlier. The giant x-symbionts of the fulgoroid planthoppers provide a striking example (pp. 386–432). (Planthoppers harbor several symbiont species; “x” is the name of a particular species.)

These bacterial x-symbionts differentiate into huge, strangely shaped forms that often contain dark lumps or intensely staining granules. These degenerate forms appear incapable of further division or reproduction.

Both males and females contain differentiated, giant x-symbionts. Only the

females have a separate rectal organ that is used as a storage depot for transmissible forms of the undifferentiated symbiont. These transmissible rectal forms are separated from the terminal giant x-forms by remarkable developmental processes (pp. 424–430).

In *Fulgora europaea*, there are four different symbiont species in the egg. These are gathered into a special embryonic organ, the collecting mycetome. Within this mycetome the different species are separated, and the organs that will house each species begin to develop. As the rudimentary x-organ differentiates, collecting arms from the midgut chord extend deep into the syncytial x-mass and surround some symbionts. Well-defined mycetocytes form containing x-symbionts. These symbionts have already begun to swell on the pathway to giant forms, but within the mycetocytes they shrink into smaller units. Eventually these mycetocytes become migratory cells, forcing their way through a narrow canal and into the intestinal rudiment. Shortly before hatching, these mycetocytes degenerate, releasing the symbionts into the intestine. With the first larval meal, peristaltic movements propel the symbionts to the rectum.

Intestinal cells in the rectum sprout long, protoplasmic, branched processes that capture the symbionts. The symbionts are then moved to special cells that differentiate into the rectal organ (p. 426). Later the eggs are infected with several symbiont species, including those from the rectal organ. Interestingly, the symbionts from the rectal organs are transmitted to the eggs in “exceptionally small numbers” (p. 407).

The x-symbionts separate into somatic and germline forms early in development. Many symbionts split into reproductive and somatic cells even earlier. For example, the symbionts of ticks often infect the primordial germ cells of females very early in embryonic differentiation. Not all of the symbionts end up in the germ tissue; some infect the Malpighian tubules and are excluded from vertical transmission (pp. 447, 451, 655).

The germ-soma separation is a process that limits the opportunities for transmission to a subset of cell lineages. Hosts commonly impose such limits on the reproductive opportunities of their symbionts. Buchner (1965, p. 707) reviews an extensive body of literature in the following way:

For some reason it is apparently to the advantage of host animals to localize the creation of transmission forms more narrowly, for a steady progression toward increased localization can be traced among the test animals. In some cases only isolated occupants of but one cell or one syncytium are converted into transmission forms; in other cases all the occupants of a cell are affected and transformation may occur anywhere in the mycetome; in still other cases a certain degree of local delimitation may be noted, for instance, to the marginal areas of the organs; and finally there are cases where the transformation is clearly limited to narrowly defined areas.

HOST CONTROL OF SYMBIONT COMPETITION

A traditional gene of population genetics is locked into the rigid mendelian system, with little opportunity to increase its success within the host. Biases of within-host success sometimes occur and are called “segregation distortion” or “meiotic drive” (Lyttle 1991). However, such biases appear to be rare, which

suggests that the Mendelian mechanism is generally successful in suppressing within-host competition of genes.

Consider a vertical symbiont that infects the eggs early in host development. Each symbiont individual is like a single "allele," so the host has a high ploidy for that "locus." Only a few alleles will be transmitted to the next generation. Any symbiont trait that causes a positive bias in transmission probability will tend to increase in frequency if the cost to overall host fitness is not too high. Thus, the symbiont population is subject to the selective pressures of segregation distortion, but without the evolutionary history of the Mendelian controls. With each new vertical symbiosis, the conflicts that favor Mendelian reproductive fairness arise once again.

Suppose the host could randomize the chances of transmission for each symbiont. The ensuing reproductive fairness would remove any potential for bias within the symbiont population. This would be favorable for the host because a symbiont could only increase its own fitness by increasing the fitness of the host. Although average host fitness would eventually improve, there is not always an immediate advantage for a host modifier that influences symbiont transmission patterns (Hoekstra 1987). Thus, it is useful to study the selective consequences of a variety of specific scenarios. (For general discussions of reproductive fairness, see Alexander 1987; Wilson and Sober 1994; Frank 1995; Maynard Smith and Szathmáry 1995.)

Host Modifiers That Repress Reproductive Bias among Symbionts Cannot Spread Simply by Randomizing Symbiont Success

If the host imposed reproductive fairness by randomizing symbiont success, the symbionts would, over time, evolve to be less competitive and less virulent. But the host modifier can only gain an advantage by direct association with relatively less virulent symbionts. The immediate benefits of the modifier depend on the details of symbiont competition and transmission.

Reproductive bias among symbionts could occur by preferential movement into the germ tissue during embryogenesis. A symbiont trait that enhanced the probability of germ-tissue colonization would spread even if that trait imposed a cost on overall host fitness. A cost could arise because the symbiont reduced allocation to beneficial metabolic traits or because the symbiont disrupted development during its attempts at embryonic migration to germ tissue. If reproductive bias among the symbionts occurred, the hosts could reduce the symbiont advantages of competition by randomizing the probability of germ-line infection and thus imposing reproductive fairness among the symbionts.

Host traits that imposed reproductive fairness on the symbionts would not spread without directly reducing symbiont virulence. For example, suppose the symbionts were diverting a share of their resources to compete for access to the germ tissue. If a host modifier were able to randomize the symbionts by physically mixing them, then reproductive fairness would be imposed. However, the host modifier does not gain in this case because the symbionts still have a portion of their resources allocated to competition rather than beneficial metabolism. There is no immediate benefit to the host.

Host Modifiers of Symbiont Reproductive Fairness Spread Only When They Directly Reduce the Competitive and Virulent Traits of Symbionts

Suppose that virulence arises from the damage competing symbionts cause to each other and the consequently reduced symbiont population available for beneficial metabolism. For example, symbionts may compete by producing specific allelopathic toxins. A host modifier could potentially reduce such competition by cleaving all symbiont toxins. The modifier would gain an immediate benefit by its association with a more robust population of symbionts.

Other host mechanisms that impose reproductive fairness may also have immediate beneficial effects. For example, hosts often sequester symbionts into specialized cells early during development and thereby control symbiont movement during embryogenesis (Buchner 1965, chap. 13). This type of physical control by the host may prevent the symbionts from competing for access to germ tissue and simultaneously prevent damaging disruption during early, sensitive phases of development.

Symbiont Modifiers of Symbiont Reproductive Fairness Spread Only When They Directly Reduce Within-Host Competition and Virulence

In the previous cases the host imposed reproductive fairness on its symbiont population. Under some circumstances, a symbiont modifier can spread that causes "self-policing" by imposing reproductive fairness among its neighbors (Frank 1995). The conditions for the success of the symbiont modifier are similar to those for the host modifier: success requires direct control and reduction of competitive and virulent traits of neighboring symbionts. A modifier would not spread if it imposed reproductive fairness without directly reducing virulence.

Summary

A host modifier that represses competition will eventually cause a decline in symbiont allocation to competitive traits. Thus, the modifier increases the average fitness of the host and symbiont population. However, the evolutionary dynamics of the modifier depend on immediate costs and benefits. A costly modifier can spread only when it directly reduces virulent traits. The equilibrium level of virulence by uncontrolled symbionts is roughly $l - r$, where r is the coefficient of relatedness among symbionts within hosts (Frank 1994, 1996b). Virulence is measured here by the proportional reduction in group (host) fitness caused by competitive traits of symbionts. Thus, a host modifier that represses the virulent tendencies of symbionts and has a cost c will spread when $c < (l - r)$.

A rare symbiont modifier promoting self-policing has different dynamics because the modifier will occur in a fraction r of the symbionts within a host. However, the net condition for increase of a symbiont modifier is also $c < (l - r)$, where c is the fitness reduction for a bearer of the modifier that reduces the destructive tendencies of local symbionts (Frank 1995).

The theory is interesting because it focuses attention on particular mechanisms for the separation of symbionts into germ and soma. Simple randomization mechanisms, although ultimately beneficial, have difficulty during the phase of initial

increase. By contrast, control mechanisms can spread if they directly limit the potential disruption of host development or directly reduce destructive competition.

DISRUPTION OF DEVELOPMENT

Symbionts are often remarkably numerous in the egg and during early embryogenesis. Can symbionts become so well domesticated that they do not reduce the efficiency of host development? That may often be the case, but many observations suggest that symbionts can be disruptive and perhaps impose a cost to the host.

In some tunicates with luminescent bacteria,

the blastomeres of an egg in total cleavage are appropriated by the symbionts, and it is obvious that in [an organism] with determined cleavage and extensive infection this appropriation of the blastomeres must cause severe disturbances in the course of development. The cleavage cells do indeed appear paralyzed; as so often occurs when embryonic cells are laden with symbionts, the tempo of their division is maximally reduced; somatic cells of the maternal body, which generally possess extensive reproductive capacities in tunicates, step into the breach and build a provisory substrate of the future organism. The substrate is not replaced until the embryonic cells are in a position to transmit their inmates to the elements of the blood which constitute their definitive site and thus make it possible for them to assume their actual organ-building tasks. (Buchner 1965, p. 589)

The primordial germ tissue often plays an important role in moving the symbionts during early development. "Above all, we must recall the widespread significance of the invaginating germ band, as a means of transportation, by which not only symbionts but also often primordial germ cells are similarly moved from posterior to anterior poles [of the egg]. This close relationship between symbionts and invagination processes is doubtless of deeper significance and should not be regarded as a chance occurrence resulting from infection [of the egg] at the posterior pole" (p. 673).

Where the germ band is formed at a distance from the symbionts, various strange and complicated developmental steps often arise.

Other untoward consequences are observed with Anoplura [sucking lice] and Mallophaga [chewing lice] where a well-described symbiont aggregation is formed at the posterior end of the eggs but the [germ band] invagination sets in laterally. As with blattids [roaches], the symbionts reach the embryonic midgut without coming in contact with the invagination and, in their later behavior, which is unusual and differs from case to case, apparently again reflect the effort of the host animals to compensate for this "bad situation." Polyphyletic devices represented here are: a refreighting to the midgut epithelium not observable elsewhere, migration of isolated mycetocytes derived from yolk cells to the body cavity and later dispersal in it, or migration of intact, very primitive mycetomes to it. It is only in *Pediculus* that there occurs a meeting of the symbionts with the end of the germ band and therewith their transportation forward, but these symbionts, as in the relatives, nevertheless get to the midgut. The complicated phenomena recently discovered by Baudisch suggest all too clearly that these are emergency measures. (P. 674)

The complications of vertical transmission and permanent housing are, in Buchner's view, necessary costs of guaranteed infection when the symbiont supply is

rare or erratic. Buchner suggests that symbionts are acquired from the environment whenever they are readily available (p. 640). Phylogenetic evidence provides further circumstantial data for the cost of carrying symbionts because, according to Buchner's analysis, symbionts are often lost when a change in diet occurs (Buchner 1965, chap. 15).

Such broad comparative information is difficult to evaluate solely from Buchner's review. But his work shows the importance of separating the different dimensions of host-symbiont interactions when analyzing evolutionary problems. Even purely vertical symbionts may impose significant metabolic and developmental costs. These costs may be offset by various metabolic advantages, of which there is considerable evidence (Buchner 1965, chap. 16). The presence or absence of the symbiont in a lineage may depend on the net effect of such costs and benefits, but focusing only on net fitness tends to obscure the details of symbiont competition within hosts and mechanisms of host control.

In summary, host control of symbiont movements and replication may be favored because the host gains immediate benefits by reducing symbiont disruption of development. The separation of symbiont cell lineages into germ and soma appears to be a common consequence of host control.

OTHER PUZZLES AND TESTS

Other puzzles arise when piecing together the fragmentary data available on host-symbiont biology. For example, there is widespread male-female dimorphism in the development and transmission of symbionts. Many symbionts appear to be transmitted primarily from mother to offspring; a male host leads to extinction for a lineage of symbionts. Not surprisingly, symbionts in males often deteriorate in early development and eventually disappear. To give just one example, in the silvanid (flat bark beetle) *Oryzaephilus surinamensis*, "the mycetomes of male imagines undergo far-reaching degenerative processes soon after hatching. The symbionts do not increase as in the females. First assuming round or pleomorphic shapes, they are sometimes fused and often disintegrated" (Buchner 1965, p. 187).

In the fulgoroid planthoppers discussed earlier, both males and females have the giant, somatic x-symbionts, but only the females have the special rectal transmission organs. The x-symbionts in males, which lack opportunity for further transmission, are similar to mitochondria in males with limited or no paternal transmission. Presumably, the efficient functioning of the symbionts in males is simply a correlated response to selection in females. In some cases the males may provide benefits to their sisters, so that functioning symbionts in males are favored by kin selection. But such cases must be relatively rare.

In some blood-sucking hemipteran bugs, both males and females have symbiont-containing mycetomes near the gonads. The females in *Ornithocoris uritui* and *Leptocimex bouteti* have small mycetomes that contain long, interbraided, filamentous symbionts. The transmission of these symbionts to very young oocytes has been described. The males of these species have relatively large, round

mycetomes attached to the seven-chambered testis at the point where the vas deferens has its origins. No information on transmission by males was mentioned (observations from Buchner 1965, p. 480).

Variation in male-female dimorphism is another example of the widespread diversity in host-symbiont interactions. Earlier I discussed observed diversity in host control over symbiont movement and transmission, separation of symbionts into germ and soma, and the disruption of development by symbionts.

My main goal is to order this mass of fascinating but confusing observations into a series of well-defined evolutionary puzzles. For example, the problems of germ-soma distinction among symbionts and the role of host control come into focus only when they are analyzed according to patterns of genetic variation and processes of reproductive competition. Obviously the great diversity of symbiotic interactions cannot be explained fully by a few theorems about when host control would or would not be favored by selection. But the theory does provide a way to begin by suggesting which data are important and what ordering of the data may be fruitful.

For a particular host-symbiont interaction, it would be useful to know the genetic variation of symbionts within hosts, the number of symbionts transmitted to offspring, the frequency and mechanisms of horizontal transmission, the proportion of symbiont lineages that are excluded from transmission (germ-soma differentiation), and the mechanisms of such exclusion. These data for particular host-symbiont pairs must also be placed into a comparative framework. For example, some symbionts appear to be orderly, nondisruptive guests, whereas others cause significant damage and appear more like virulent parasites. Some hosts separate their symbionts into distinct germ and soma very early in development; other hosts select transmissible symbionts later in development after there has been much opportunity for symbiont competition.

Does the age of the host-symbiont interaction explain such variation, as suggested by Buchner (1965) and many earlier authors? Or do patterns of transmission and competition explain variation in host-symbiont relationships, as developed in the ahistorical theories of parasite virulence (Anderson and May 1991; Ewald 1994; Frank 1996b)?

The ideal is, of course, to combine the historical and process-oriented views into a full comparative analysis. Although that will be a difficult task, few problems provide so much interesting comparative material. By contrast, the origins of germ-soma differentiation in metazoans or the origin of meiosis and Mendelian reproductive fairness represent a few evolutionary events hidden deep in the past. To emphasize the opportunities for historical comparisons of symbioses, I close with a final quote from Buchner (1965):

The different methods used by the Anoplura [sucking lice] in housing the symbionts or settling the ovarial ampullae clearly demonstrate that they represent solutions which are found from case to case, and are not derived from one another. . . . [T]he study of the embryonic development affords further proof of such polyphyletic mode of origin. (P. 496)

There is no relationship between the degree of symbiotic organization and [the phylogenetic position] of the host. For example, *Haematopinus* represents the most primitive type

of Anoplura, on the one hand, yet has the most complicated cycle [of symbiont transmission], on the other. (P. 748)

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