Chapter 17

Do Populations

Self-Regulate?

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Voles probably exemplify a general law that all species are capable of limiting their own population densities without either destroying the food resources to which they are adapted, or depending upon enemies or climatic accidents to prevent them from doing so. If this is true, self-regulatory mechanisms have presumably been evolved through natural selection, and arguments in support of this view can certainly be advanced.

<div align="right">CHITTY 1960, p. 111</div>

This sort of stabilization is often described in such terms as "the population adjusts itself to its food supply" or even "the population regulates its reproduction so as not to produce numbers in excess of what the environment can support." Such expressions imply that the density regulation is an evolved adaptation of the population as a whole, and that without such adaptations there would be no numerical stability.

These interpretations are utterly without justification. . . . the adjustment of fecundity to food supply is adaptive from the individual standpoint.

<div align="right">WILLIAMS 1966, p. 236</div>

Whether insect populations "self-regulate" may well depend on the meaning assigned to self-regulation. In the broadest sense, *self-regulation* is a label for instances in which the actions of members of a population prevent it from exceeding its resources. Specifically individuals emigrate, die, or reduce the numbers or quality of their offspring at high densities, and their doing so is not the direct result of extrinsic factors other than conspecifics. For example, deaths from predators, pathogens, or starvation are not labeled self-regulating, but deaths from causes within the population (e.g., cannibalism, senescence, and density-induced hormonal imbalance) have been. Similarly, emigration upon exhaustion of food or cover at a site is not termed self-regulating, but emigration from a site having all needed resources has been.

Thus *self-regulation* (broad sense*) refers to density-regulating actions by members of the regulated population. The evolutionary origins of such actions are important to understanding them, and the reproductive consequences of the actions are the key to their evolution.

On the one hand the population regulating actions of individuals may be to their own reproductive advantage—for example, emigration from a habitat that is likely to become unsuitable to one that is likely to be bountiful; or death as a consequence of all-out reproductive effort at a time when later reproduction would be precluded. In these instances the evolutionary origin of the action need be no more complicated than differential reproductive success of genetically different individuals. The population-regu-

*A more restrictive definition of population self-regulation is that it involves *self-sacrificing* density-regulating actions by population members; such a definition may be of a null set.

lating consequence of the action may be an incidental effect of selection at the individual level; parsimony dictates that it be so considered in the absence of evidence to the contrary. To reiterate, an advantage or benefit to a population (or species, or ecosystem) does not require that the phenomenon evolved in that context; it need not be a population (or species, or ecosystem) adaptation. Adaptation at the population level or higher is a difficult concept and should not be invoked lightly. In the past it has been used as an easy way to avoid difficult questions (Williams 1966).

On the other hand the population-regulating actions of individuals may be detrimental to individual reproductive success—the individuals may be reducing their genetic contribution to future generations but benefitting the population. If this be so, natural selection at the level of the individual would tend to eliminate the behavior. Individuals that acted selfishly would leave more descendents than those that acted in the interests of the population. Within populations, genes that promoted selfishness would increase in frequency at the expense of their alleles, which promoted altruistic reproductive restraint, even if the population ultimately became extinct as a result. Extinction of populations with selfish individuals suggests that differential survival and perpetuation of groups of individuals (demes in this case) could effect adaptation at the population level (or higher). Such a process has been termed *group selection.*

The hypothesis of evolutionary change by group selection has a long history and many forms. V. C. Wynne-Edwards (1962) stimulated widespread discussion of its significance by proposing that much of the social behavior of animals concerned their regulating their own numbers and that the behaviors had evolved through group (i.e., interdemic) selection. Since 1962 group selection has been carefully analyzed, reviewed, and rereviewed (e.g., Maynard Smith 1964, 1976, 1978, Williams 1966, 1971, Ghiselin 1974, Gilpin 1975, E. O. Wilson 1975, D. S. Wilson 1975, 1980, Alexander & Borgia 1978, Wade 1978, Dawkins 1982).

This chapter is not the place for another detailed review of group selection, but some general ideas need to be stated before pursuing further the subject of self-regulation in insect populations. Group selection—in the sense of differential survival and perpetuation of genetically different groups—does occur. The debate over group selection concerns whether it has a significant role in determining the speed with which a trait changes and, in particular and of greatest importance, whether it can cause a trait to change in a direction opposite that produced by individual selection. The focal issue is whether traits detrimental to an individual and its close kin can become established because of their beneficial effect on the survival and propagation of the group. This issue has been met in two ways. First, modelers have postulated combinations of population phenomena that overcome the principal theoretical difficulties with selection among demes overpowering selection among individuals—namely, how to make extinction and reformation of demes common enough to act within the same

time scale as individual selection, how to produce demes that differ significantly in the traits in question, and how to combine the isolation needed for demes to be different with the vagility needed for the successful groups to spread to the sites vacated by unsuccessful ones. The general nature of such models is that they work, mathematically, provided the population structure remains within the proposed narrow limits. Modelers have thus far had little success in identifying real populations that have the required characteristics, but so little is known of demographic structure under natural conditions and the particular requirements vary so much among models that there may be room for optimism. [cf. Maynard Smith (1976) and Wade (1978) for reviews of group selection models.]

The second approach to evaluating the importance of group selection in determining direction of evolutionary change is to study critically the traits that are supposed to owe their existence to it. To the extent that their existence accords with the simpler model of individual selection, the likelihood of group selection being important is diminished. With the possible exception of sexual reproduction (Williams 1966, 1975, Maynard Smith 1978) no trait has continued to receive strong support as being good for the population but detrimental to individual reproductive success.

Present evolutionary theory suggests that most, perhaps all, adaptation originated through selection at levels no higher than the individual and its close kin. If this is so, self-regulation in insect populations should be a by-product of individual adaptation and not itself an adaptation.

The remainder of this chapter will analyze seven phenomena that result in, or have been reputed to result in, population self-regulation. Emigration and sex ratio will be discussed at greater length than other phenomena because they are of general interest and importance and are especially likely to repay further study. The phenomena associated with population self-regulation are among the least understood in insect ecology; to some extent our ignorance results from their having been explained away as traits good for the population or species.

17.1 CANNIBALISM

Intraspecific predation is frequent among insects and usually has a density-dependent component that makes it population regulating (Fox 1975a, Polis 1981). In most cases older, larger individuals attack younger, smaller ones, but cannibalism within cohorts occurs (e.g., Fox 1975b, Duelli 1981), and pupation and molting sometimes reverse the vulnerabilities of age classes (e.g., Corbet & Griffiths 1963, Tschinkel 1981). When the cannibal is a predator and the conspecific prey is no kin, the origin and adaptiveness of the behavior are apparent—a predator that dines on a competitor has aided its own reproduction indirectly as well as directly. The double benefits of cannibalism reduce the surprise in finding that herbivorous insects are at times avid intraspecific predators. Hatchling butterflies and leafbeetles

eat unhatched eggs or other hatchlings, and *Tribolium* larvae feed on eggs, pupae, and callow adults (Brower 1961, Eickwort 1973, King & Dawson 1972).

The population effects of cannibalism under natural conditions are difficult to isolate from other mortality factors, but studies by Fox (1975b) on the backswimmer *Notonecta hoffmanni* and by Polis (1980) on the scorpion *Pauroctonus mesaensis* establish that cannibalism can be a major mortality factor (more than 50% of first-instar *N. hoffmanni*) and a major source of nourishment (28% of diet biomass observed for *P. mesaensis*).

In some instances cannibalism is in defense of a specific space that has the food required for the cannibal's development (see Section 17.2 and Table 17.1). In grain-feeding insects, the first larva to occupy a grain may attack and kill any that follow (Crombie 1944). Similarly, the earliest parasitoid larva in a host may kill subsequent ones (Askew 1971). In the corn earworm (*Heliothis zea*) a larva occupying an ear tolerates no companions even though the food supply is sufficient for several, and cannibalism limits the population well below the limits of food resources (Barber 1936). Stinner et al. (1977) reported cannibal-caused earworm mortalities in excess of 75% as "not unusual" in North Carolina corn fields.

Cannibalism has self-evident population stabilizing effects and has never been mistaken for self-sacrifice. The remaining six phenomena are more problematical both in their effects and in their evolutionary origins.

17.2 TERRITORIAL BEHAVIOR

Some instances of territorial behavior in insects are population regulating but most are not. In either case, territoriality is understandable as an individual adaptation. Individuals defending an area—a *territory*—generally are maintaining exclusive use of certain resources within it. Territoriality has been reviewed by Wilson (1975) and Davies (1978b) and, relative to insects, by Otte and Joern (1975), Price (1975), and Burk (1979).

Territorial behavior in insects can be subdivided on the basis of the context in which it occurs, the most frequent being a male defending an area where he may mate with a female (Table 17.1).

Mating territories are of small consequence to total population and probably none at all to population growth. Nonterritorial males do not necessarily perish prematurely and females are not denied matings. Nonterritorial males may even secure some matings by adapting an alternative, "satellite" behavior (Alcock 1979, Cade 1979). The chief effect of mating territories is an increase in the variance of matings per male; when the area defended is merely a mating site, the result can be viewed as discrimination of male quality by females (Blum & Blum 1979, Bradbury 1981). Insect mating territories have been experimentally verified in the field in dragonflies (Moore 1964) (Fig. 17.1), grasshoppers (Otte & Joern 1975, Schowalter & Whitford 1979), and butterflies (Davies 1978a).

Table 17.1 Insect territoriality—three modes and some examples of each[a]

Principle function of territory—insect examples (References)

1. Mating—Male defends area from other males
    (a) Defended area includes one or more soon-to-be-receptive females:
        Solitary bees and wasps (Lin 1963, Alcock et al. 1978).
        Parasitoid wasps (Wilson 1961, King et al. 1969).
    (b) Defended area includes scarce resource required by female:
        Dragonflies—oviposition sites (Campanella & Wolfe 1974, Uéda 1979).
        Grasshopper—low-resin feeding site? (Schowalter & Whitford 1979).
        Big-legged bug—feeding site (?) (Mitchell 1980).
        Fruit fly—oviposition site (Bush 1969).
        Butterfly—oviposition sites (Baker 1972).
        Horned beetle—feeding site (Eberhard 1979).
        Bark beetle—feeding and oviposition site (Rundinsky et al. 1978).
        Solitary bee—feeding site (Alcock 1980).
    (c) Defended area merely a potential mating site:
        Damselfly (Bick & Bick 1965).
        Roaches (Bell et al. 1978, Gautier 1978).
        Meadow katydids (Feaver 1983).
        Field cricket (Cade 1979).
        Butterflies (Davies 1978a, Bitzer & Shaw 1980).
        Hawaiian *Drosophila* (Ringo 1976, Spieth 1978).
        Fruit flies (Tychsen 1977, Burk 1983).
        Solitary bees and wasps (Alcock et al. 1978, Gwynne 1980).
2. Progeny rearing—one or both parents defend area that has scarce resources required by progeny
    (a) Parent(s) defends site directly:
        Cricket (Walker 1980).
        Aphid (Whitham 1979).
        Burying beetles (Milne & Milne 1976).
        Dung beetle (Palmer 1978).
        Passallid beetle (Schuster 1975).
        Digger wasps (Steiner 1975, Brockman & Dawkins 1979).
    (b) Female marks site—other females avoid because resource is preempted.
        Bean weevil (Oshima et al. 1973).
        Butterflies (Rothschild & Schoonhoven 1977).
        Fruit flies (Prokopy 1981).
        Parasitoid wasps (Vinson 1976).
3. Feeding—juvenile or nonreproductive adult defends its food or feeding site
    (a) Rivals destroyed:
        Antlions (Wheeler 1930).
        Granary weevil (Assem 1971).
        Corn earworm (Barber 1936).
        Parasitoid wasps (Askew 1971).

Table 17.1 *(Continued)*

Principle function of territory—insect examples (References)

   (b) Nonlethal defense:
        Mantids (Edmunds 1976).
        Antlion (McClure 1976).
        Soldier beetle (Rausher & Fowler 1979).
        Mosquito (Gillett et al. 1977).

*Eusocial species excluded, see Chapter 18.

A second context of insect territoriality is one or both parents defending an area that has resources required for development of progeny (Table 17.1). Such defense prevents "scramble" or exploitation competition (Chapter 15) in which the developing juveniles may use up available resources and perish for lack of more. Blowflies, for example, may lay so many eggs on a piece of carrion that the maggots run out of food prior to obtaining enough to pupate (Nicholson 1954). On the other hand, a pair of burying beetles may not only defend a small carcass from other burying beetles
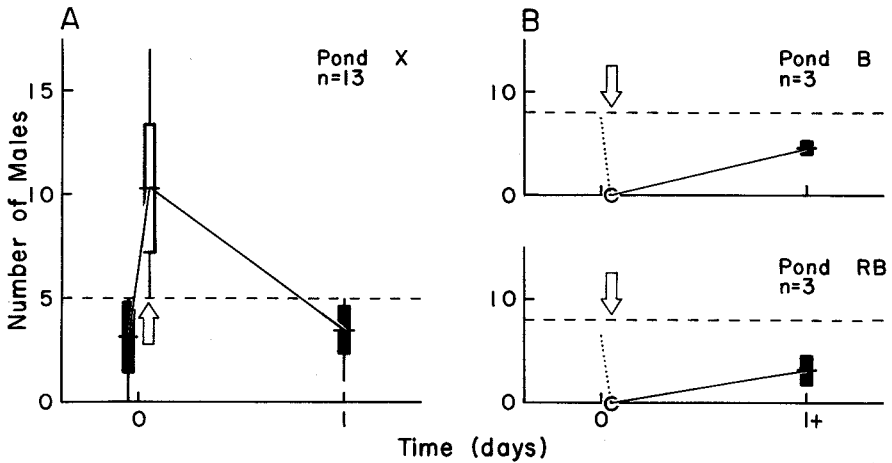

Fig. 17.1   Results of adding and removing territorial male odonates around bomb-hole ponds, Arne, England (Moore 1964). (A) Results of 13 addition experiments, *Cerigrion tenellum*, 1955 (from Table 9, Moore 1964; two cloudy days omitted); arrow indicates addition of 5–10 males to resident population. Horizontal dotted line is maximum number of males at pond under natural conditions. Vertical symbols show range (vertical line), mean (cross line), and ±1 standard deviation (vertical bar): left, prior to adding males; center, males added; right, 1 day later. By marking the males prior to release, Moore determined that they were only half as likely to remain as those already in residence, but this excludes the return to low level being a result of all introduced males leaving. (B) Results of six subtraction experiments, *Pyrrhosoma nymphula* (from Tables 4 and 8, Moore 1964); arrows indicate removal of all resident males. Other symbols as in A. Specific starting population not recorded; new "experimental" steady population (left symbol) noted after "a day or more."

(Milne & Milne 1976), but also introduce mites that consume blowfly eggs and small larvae (Springett 1968).

A more usual method of defense of resources for progeny is less direct and fails to satisfy some definitions of territoriality: the ovipositing female deposits a chemical that warns other females that added progeny will meet rigorous or overwhelming competition. This behavior is best known in parasitoids (Vinson 1976) (Fig. 17.2) and fruit flies (Prokopy 1981) and is diverse in detail: (1) females may leave chemical trails as they search for hosts (DeBach 1944, Price 1970, Greany & Oatman 1972); (2) females may chemically mark the host after ovipositing (Prokopy et al. 1977, Prokopy 1981); (3) females may chemically mark the host internally during oviposition (Greany & Oatman 1972, Guillot & Vinson 1972, van Lenteren 1976). In each case chemicals enable the depositing female and others to direct their searches or their eggs elsewhere. The advantage that a marking female gains by obeying her own marks is evident; the advantage to other females of refraining from ovipositing in a marked host are not as certain but two considerations probably contribute in varying proportions: (1) an offspring of a subsequent female will be younger than that of the marking female and will therefore have little chance of winning in competition with it; (2) even if later progeny are competitively equivalent to earlier ones, the offspring of a subsequent female will have no greater than 50% chance of

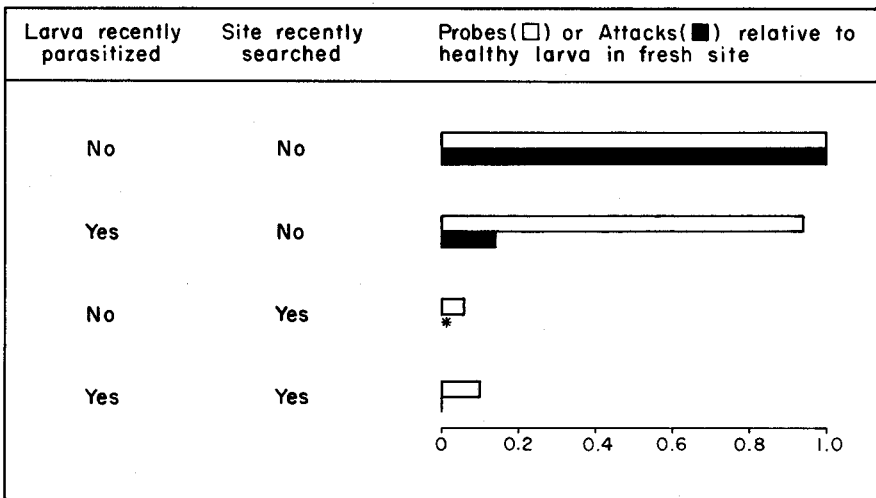| Larva recently parasitized	Site recently searched	Probes(□) or Attacks(■) relative to healthy larva in fresh site
No	No	
Yes	No	
No	Yes	
Yes	Yes	

0   0.2   0.4   0.6   0.8   1.0

Fig. 17.2   Probe and attack rates of the braconid wasp *Orgilus lepidus* on larvae of potato tuberworm *Phthorimaea operculella* as influenced by site having been searched for the previous 2 hr (by other individuals) or by larvae having been parasitized less than 1 hr earlier. *Probes* were thrusts of the ovipositor toward or into a larva; *attacks* were ovipositor insertions of 2 sec or longer (time enough for oviposition). Rates are relative to those with healthy larvae at previously unvisited sites (uppermost bars) (data from Greany and Oatman 1972). Data on attack rate not available.

winning (case where no more than a single larva can mature) and may have none [all larvae in a multiply occupied host may have zero probability of acquiring enough food to mature (e.g., Jackson 1966)].

Whether marking chemicals are deposited continually prior to host finding, on the host, or in the host has important effects on how useful they are to the marking female and how likely it is that ovipositing females of hyperparasites (including parasitoids of fruit-feeding larvae), cleptoparasites, or competitors will use the chemicals to their advantage and to the detriment of the marking female (Vinson 1976, Prokopy & Webster 1978). External marks that last only a few hours may minimize the costs and perhaps not decrease the benefits; internal marks should perhaps last longer (Guillot & Vinson 1972). Previous oviposition may be detected by cues other than specially produced chemicals. For example, Cirio (1971) found that host juices smeared about by ovipositing olive flies, *Dacus oleae,* inhibit further egg laying, and Rothschild and Schoonhoven (1977) found that females of *Pieris brassicae* avoided ovipositing on cabbage leaves that had plastic models of eggs attached (though a volatile chemical deposited with the natural eggs was the greatest deterrent to continued oviposition).

In all of these cases parents act to insure their progeny access to needed resources. The effect may be prevention of excess population and crashes, but no function other than facilitation of individual reproduction need be postulated. In fact, at higher densities the behavior maximizes production of new adults and could then reduce the carrying capacity, causing a crash.

A final context of territorial behavior in insects is the defense of food or feeding sites by developing juveniles or nonreproductive adults (Table 17.1). Again there may be population-regulating effects, but the function is easily placed at the individual level—those maintaining territories have adequate food and probably reduced chances of predation (density-dependent predation, Chapter 12). In many cases attack is direct and the loser becomes food for the winner (Section 17.1). In other cases the defense is not lethal and may be so subtle as to make its function doubtful: The moist eggs of *Aedes aegypti* hatch in response to reduced oxygen, ordinarily a result of metabolism of microorganisms growing on their sculptured surfaces. The first larvae to hatch remain near the unhatched eggs and browse. Gillett et al. (1977) speculate that cropping of bacterial growth on unhatched eggs allows oxygen to increase and reduces the likelihood of additional hatch. Further hatch should occur only when uncropped bacterial growth again reduces dissolved oxygen. Eggs in a container thus hatch at a rate that is compatible with available food.

17.3 EMIGRATION

If a population produces higher proportions of emigrating individuals at higher densities, the effect is regulation; however, there is no need to

consider emigration a population adaptation if it proves adaptive at a lower level (but see Lidicker 1962 and Taylor & Taylor 1979).

General features of insect migration and dispersal were discussed in Chapter 7. The concern here will be with *emigration*—leaving a place of abode or habitat. In the paragraphs below progressively more complicated circumstances will be considered; in each case selection at the individual or genic level can produce the described adaptations.

17.3.1 Transient Habitats

Many insects develop in habitats that remain suitable for no more than one or a few generations—for example, dung, carrion, fruit, logs, early stages of secondary succession. In such habitats permanent residence soon results in total failure. As old habitats vanish, new ones generally appear, and emigrants have unexploited habitats to colonize. Southwood (1962) showed a correlation between level of emigration and degree of impermanence of the habitat; he proposed (p. 172) that the "prime evolutionary advantage" of emigration is to enable "a species to keep pace with changes in the locations of its habitats." Its evolutionary origin is attributable to advantages at lower levels.

17.3.2 Fluctuating Habitats

Long-lasting habitats (e.g., lakes, grassland) may fluctuate in quality. If fluctuations in a region are not in phase, emigrants leaving a deteriorating habitat may find better circumstances nearby. If fluctuations are in phase, as in seasonal changes, only long-distance movement will be of advantage (e.g., monarch butterfly, Urquhart & Urquhart 1978). Insects generally escape seasonally synchronized fluctuations by diapausing (Chapter 6) rather than by emigrating.

17.3.3 Density-dependent Emigration

Emigration is often density dependent, with the proportion of emigrants increasing with population density (e.g., Naylor 1965, Shaw 1973, Hokyo & Kuno 1977, Tshinkel 1978).

The genetic basis for such behavior must be a coadapted set of genes that perpetuates itself more frequently than alternatives because it programs an individual to "emigrate if perceived density $>d$; stay if density $<d$." Density can be perceived directly [e.g., by tactile cues (Lees 1967)] or indirectly (e.g., through excretory products or food quality). Territorially can be considered a special case: "Emigrate if a territory can't be won; stay if one can." The program can be adaptively modified by incorporating previous experience—for example, "increase d if individual has emigrated once before"—or by restricting its use to adults of specific age or sex. The

existence of such specific genetic instructions is easily demonstrated in aphids because the parthenogenetic generations produced from a single overwintering egg or parthenogenetic female are genetically identical—the individuals constitute a clone (Lees 1961). Members of a clone show just the type of responses postulated, and the observed differences in emigration cannot be attributed to genetic differences, since none exist. [R. G. Harrison (1980) recently reviewed dispersal polymorphisms in aphids. Richard Dawkins (1980, 1982) reviewed the evolution of genetically determined behavioral alternatives in populations and included forceful statements of conditional strategies, as used above.]

17.3.4   Bet Hedging

In the previous paragraph the decision to emigrate or not is based on some circumstance or set of circumstances that correlates with reproductive payoff. For the responsible gene complex to increase in frequency, the correlation need not be perfect, but the average benefits must exceed those of competing complexes. Averages are only part of the story, however. What if a decision that is usually favorable is occasionally disastrous? For instance, what if staying home resulted in total reproductive failure once in every 5–20 generations—even though staying home, at low densities, is generally highly favorable? How may an individual (i.e., its relevant gene complex) avoid catastrophe relative to emigration? The most straightforward way for females is by widely spaced oviposition. A female may lay a portion p of her eggs at home and then emigrate, laying the remainder $(1 - p)$ in one or more other places. This is the strategy adopted by the spruce budworm; females lay approximately half of their eggs at their place of origin and the remainder at one or more sites that are generally 50 or more km apart (Fisher & Greenbank 1979). Such a strategy can be enriched, or compromised, by making p an inverse function of home habitat deterioration (e.g., spruce budworm, Blais 1953; a seedbug, Solbreck 1978).

   A more tenuous method of spreading the risks relative to emigration decisions is for a female to cause a portion of her progeny to stay at home (p) and the remainder $(1 - p)$ to become emigrants. Such maternal influence is more easily accomplished and more likely to be adaptively correct the earlier the dispersal occurs in the ontogeny of the offspring; for example, the gypsy moth female controls the emigration tendencies of hatchlings by varying the amount of food in the eggs (Capinera & Barbosa 1976). Western tent caterpillar females likewise vary the nutrient content of their eggs, but here all hatchlings remain together and maintain an elaborate silk shelter. Nonetheless, the larvae from heavier eggs are more active foragers and eventually produce large adults that emigrate several kilometers. Larvae from lighter eggs produce light adults that remain close to the place of pupation (Wellington 1977).

   The most convincing evidence of maternal influence extending to the

emigration of adult progeny is from aphids. A female vetch or pea aphid (*Megoura viciae* and *Acyrthosiphon pisum*) can parthenogenetically produce varying proportions of winged and wingless daughters (Fig. 17.3). The determination is complete at birth and cannot be reversed by changing the crowding or food of the nymphs (Lees 1967, Sutherland 1969). Consequently, at birth some daughters are denied flight and others are programmed to develop wings and, to an unknown extent, to emigrate. In the bean aphid, *Aphis fabae*, maternal influence is more tentative. Nymphs that would otherwise develop into wingless daughters become winged ones if crowded (Shaw 1970a). Furthermore, at lower densities many of the winged morphs do not emigrate, and of those that do some first deposit progeny on the home plant (Shaw 1970b, 1973). Since the parthenogenetically produced daughter of an aphid is genetically identical to its mother, the reproductive interests of the two are the same, and maternal influence can operate without conflict between parent and offspring. Sexually produced progeny, being genetically different from the mother, have different genetic interests (Trivers 1974), and conflicts relative to maternal influence are expected. Nonetheless, some insects, in game theory terms, "play a mixed strategy" relative to emigration (Dawkins 1980). Stearns (1976) calls this type of risk spreading "bet-hedging."

17.3.5 Stable Habitats

When habitats are transient or erratically fluctuating in quality, the adaptive significance of emigration is clear. What if all sites suitable for colonization are permanent, stable, and already occupied, and migrant mortality is extremely high? Even so, substantial emigration should occur—according to a simple mathematical model developed by Hamilton and May (1977). The qualitative explanation is that genotypes that only stay home have no chance of taking over other sites; no matter how meager the chances of takeover,
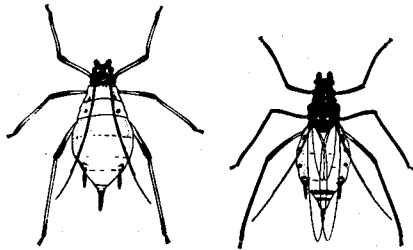

Fig. 17.3   Wingless and winged parthenogenetic females ("apterous and alate virginoparae") of the vetch aphid, *Megoura viciae*. The two morphs are genetically identical; apterous virginoparae produce either morph, depending on crowding; alate virginoparae produce only the apterous morph. (Under some conditions virginoparae produce sexual morphs as well.) (From Lees 1961, *Roy. Entomol. Soc. Lond. Symp.* **1**:69.)

some emigration should win over no emigration, and substantial emigration is apt to win over minimal emigration.

The repertoire of evolutionary gambits operating on insect emigration at the individual or genic level is not exhausted in the paragraphs above but is at least revealed as adequate to accommodate the meager facts we have as to what are the actual patterns of insect emigration.

Models investigated by Roff (1975) and by Hamilton and May (1977) predict that levels of emigration that are evolutionarily stable (without group selection) differ in one direction or the other, from levels making for maximal occupancy of sites and minimal chances of extinction—that is, levels best for the species. In theory the models could be used to test for effects of group selection on emigration; in practice the assumptions of the models are too simple relative to reality to warrant such use.

17.4   SEX RATIO

Sex ratios vary within and between populations, sometimes in such directions and circumstances as to be advantageous to the population (e.g., Landahl & Root 1969, Mitchell 1970, Giesel 1972). Population regulation would result from changes in sex ratio if at low densities the proportion of males was just high enough to insure that all females were fertilized and at higher densities the proportion of males (i.e., nonegglayers) varied directly with density (Fig. 17.4, line a). With this hypothesis in mind Anderson (1961) reviewed the relations of density to sex ratio and found one case of a *decrease* in proportion of males with increased crowded (note line b, Fig. 17.4) and one case of an increase. In none of the remaining cases ($n \simeq 25$) was a significant change in sex ratio proved for the densities studied.

Although Anderson did not note it, most of the data he assembled fit deductions made by R. A. Fisher (1930, 1958) 30 years before. Fisher concluded that genic selection would result in equal parental expenditures on male and female progeny—that is, the most usual sex ratio (regardless of density) should be 0.50 males (Fig. 17.4, line c). He reasoned that in sexually reproducing organisms each individual receives half its genetic material from a male and half from a female; therefore, males and females *as classes* contribute equally to each generation. If one sex should be in the minority, the average individual of that sex will contribute more genetically to the next generation than the average individual of the majority sex. Provided individuals of the minority sex had not been more costly to produce, those parents that had produced an excess of the minority sex would, per parent, have more grandprogeny than those that had produced the two sexes equally or an excess of the majority sex. The consequence would be increased frequencies of the genes that had augmented the production of the minority sex and a shift in sex ratio toward equality. This would
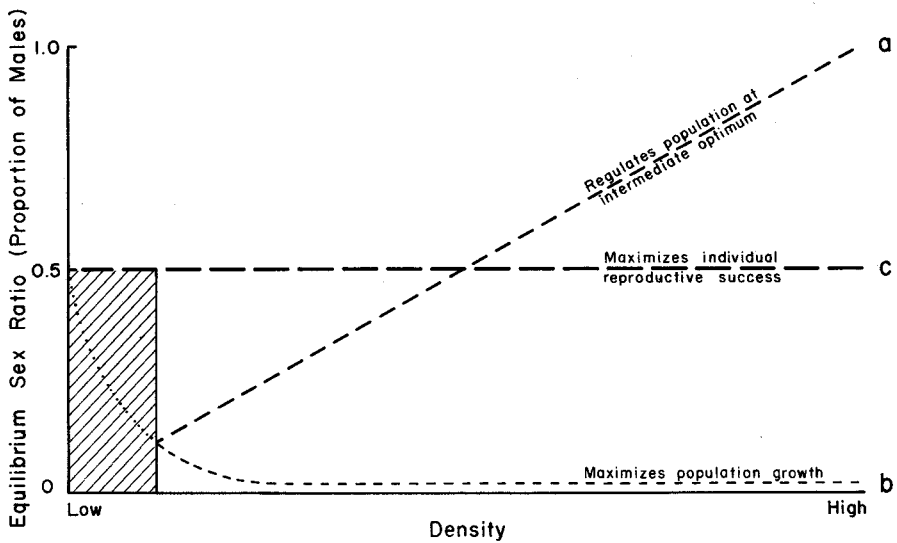
Fig. 17.4   Relation of sex ratio to population density under various assumptions: (a) sex ratio is a population adaptation that stabilizes the population at a favorable density, (b) sex ratio is a population adaptation that produces maximal population growth, (c) sex ratio is a result of selection of genes that maximize individual reproductive success. In shaded area, sex ratios for *a* and *b* may have to be high to insure that all or most females are mated. (It is assumed that males are capable of multiple matings, that costs of sexes are equal, and the mating is random.) (Modified from Williams 1966.)

continue, but as the sex ratio approached equality, the force of selection would weaken.

If sex ratios evolve entirely as Fisher proposed, they should have no role in regulating populations. Except perhaps at the lowest densities there should be more males than required to fertilize all females, and the sex ratio should remain constant rather than increasing with density.

Fisher's insights lay dormant nearly as long as Gregor Mendel's but eventually were seminal to the fuller development of the theoretical aspects of sex ratio evolution—germinating slowly (Shaw & Mohler 1953, Bodmer & Edwards 1960, Kolman 1960), blooming with a paper by W. D. Hamilton (1967), and maturing new seed in the recent book by E. L. Charnov (1982). Insects played prime roles in developing and testing sex ratio theory, and their potential is far from exhausted. Variations in insect sex ratios, within and between populations, will repay close examination. Situations in which sex ratios are expected to differ from equality (i.e., from 0.50 males) are discussed below.

17.4.1   Differential Mortality

Leigh (1970), Trivers (1972), Polis (1980), Marshall (1981), and Duelli (1981) have dealt with this topic. If one sex leads a more hazardous life than the

other, the sex ratio will change with age. Fisher (1930, 1958) noted that if sex-biased mortality occurs during the time of parental investment (as in man), more of the sex at risk should be conceived and fewer should survive the period of dependency on parents. Sex-biased mortality after parental investment has ended should have no effect on earlier sex ratios. Adult males often take more risks than females, with the result that sex ratios that started at equality become heavily female-biased as the reproductive season progresses. For example, most females of the burrowing cricket *Anurogryllus arboreus* are still alive after all males have died (Weaver & Sommers 1969, Walker 1980).

Sex-biased emigration, immigration, or diapause can affect local sex ratios in ways analogous to mortality (Johnson 1969, Denlinger 1981, Shroyer & Craig 1981).

17.4.2 Sex Chromosomes with Meiotic Drive

The topic of sex chromosomes with meiotic drive has been dealt with by several workers (Hamilton 1967, Wood & Newton 1977, Dawkins 1982). If a gene on a sex chromosome subverts meiotic segregation in its favor— that is, causes its sex chromosome to be disproportionately represented among the functioning gametes—it will increase in frequency and distort the sex ratio. A simple case is a gene on a Y chromosome that causes all, or most, functional sperm to carry it. Such a gene will rapidly increase the proportion of males until the population becomes extinct (Hamilton 1967). A "driving-Y" gene (M^D) is known in *Aedes aegypti* (Hickey & Craig 1966a,b; Wood & Newton 1977); it apparently works by causing X chromosome breakage during spermatogenesis. Some X chromosomes are resistant to M^D (progeny of M^D males bearing these are approximately 0.50 male) and some are sensitive (progeny are as much as 0.99 males). Because resistant X chromosomes are widespread in natural populations, the value of M^D for control of this mosquito is limited. Wild populations with M^D have high frequencies of resistant X chromosomes and 0.50–0.60 males; populations lacking M^D have low frequencies of resistant X chromosomes and may be temporarily suppressed by introducing M^D (Wood & Newton 1977).

Driving-X genes are known in *Drosophila* (Novitski 1947, Bryant et al. 1982). They cause female-biased sex ratios and, in the absence of opposing forces, could lead to such a shortage of males that the population would become extinct.

For both driving-Y and driving-X genes, selection at loci on other chromosomes should reduce the distortion and return the sex ratio toward 0.50 males. (In the presence of excess investment in either sex those individuals with genes causing greater investment in the neglected sex have more grandprogeny—Fisher 1930). The facts that more than 0.50 males generally reduces population increase and that less than 0.50 males, in moderation, accelerates population increase are of little consequence in explaining the fates of these variations in sex ratio.

17.4.3   Local Mate Competition (LMC)

This topic has been dealt with by Hamilton (1967, 1979), Maynard Smith (1978), Waage (1982), and Charnov (1982). For the equilibrium sex ratio to be 0.50 males, competition for mates must be populationwide. If competition is local, sons may decrease the mating success of one another; then, even if males are a minority, producing more may not pay off in more grandprogeny. The effect is clearest at the extreme: When only brother–sister matings occur, producing more sons than required to fertilize all daughters decreases the expected number of grandprogeny. Hamilton (1967) listed 25 species of insects and mites, in 16 families, that usually sibmate; the proportions of males in typical sibships were 0.02–0.33, sibships of fewer than 16 generally had a single male (11 of 13 species). So far as known, males of the species listed by Hamilton are haploid and always come from unfertilized eggs (*arrhenotoky*). The females produce the strongly female-biased broods by releasing sperm from the spermatheca on all but one or a few eggs.

Hamilton (1967) and Maynard Smith (1978) calculated what evolutionarily stable sex ratios should occur for different degrees of local mate competition. Hamilton (1979) found that fig wasps of several genera had mean sex ratios roughly correlating with their probabilities of outbreeding (as estimated from other evidence), and Waage (1982) showed similar agreement with LMC theory in an analysis of sex ratios in 31 species of scelionid wasps (Fig. 17.5).

The ability of individual females to vary sex ratios of their broods in response to stimuli that predict the degree of LMC is considered in the next section.

17.4.4   Values of the Sexes Vary with Circumstances

Maynard Smith (1978), Hamilton (1979), Suzuki and Iwasa (1980), Werren (1980a,b) and Charnov et al. (1981) have dealt with this topic. Producing a male or a female may have different payoffs to a parent under different circumstances. A parent that can vary the sex to fit the circumstance has a reproductive advantage over one that cannot.

One well-studied example concerns females of certain parasitoid Hymenoptera that lay male-producing (unfertilized) eggs on smaller host individuals and female-producing eggs on larger ones (see Charnov et al. 1981 for references). Each host produces one parasitoid, the size depending on the size of the host. The smallest functional parasitoid males are generally smaller than the smallest females, and larger females apparently have a greater increase in fertility than do larger males. Consequently, by controlling egg laying in the manner described, a female produces more grandprogeny (and perhaps more progeny, since fertilized eggs may be wasted in minimal-sized hosts—Assem 1971). The overall sex ratio among a fe-
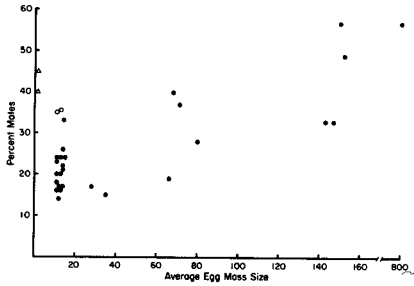
Fig. 17.5 (*Left*) Average sex ratios and average egg mass size of host for 31 species of scelionid wasps. When host egg masses are small, an ovipositing female can parasitize every egg, making it likely that her progeny will mate only with sibs (i.e., that there will be maximum local mate competition). When host egg masses are large (e.g., >50), an ovipositing wasp cannot attack every egg and leaves oviposition opportunities for other females—thereby reducing average LMC and selecting for less female-biased sex ratios. Reduced LMC is also characteristic of species that attack single eggs ($\triangle$) and those that have unusually small daily fecundities ($\bigcirc$). (Modified from Waage 1982.)
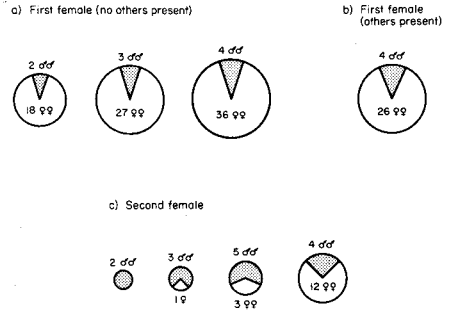
Fig. 17.6 (*Right*) Representative sibships of *Nasonia vitripennis*, a parasitoid wasp that attacks blowfly pupae and adaptively varies sex ratio (data from Werren 1980b). Each circle represents eggs laid by one female on one pupa. (See text for further explanation.)

male's progeny should still approximate 0.50 for an outbreeding population—provided an abundance of minimal-size hosts doesn't make sons cheaper to produce than daughters (see next section and Green 1982).

A second example of individual females adaptively varying their outputs of sons and daughters concerns circumstances that predict different degrees of local mate competition. The best-studied species is a pteromalid wasp that parasitizes blowfly pupae (Wylie 1966, Werren 1980a,b). If a female *Nasonia vitripennis* finds a host puparium that has not been previously parasitized, she lays 20–40 eggs on it. The females in the resulting sibship are generally mated by their flightless brothers. In keeping with LMC theory, males constitute only about 9% of each such sibship when the ovipositing female has no evidence of the presence of peers (Fig. 17.6a—Note that the sex ratio does not change with size of sibship.) However, if the female ovipositing on an unexploited host has come in contact with previously parasitized hosts or with other females (conditions predicting extra-sibship competition among males), she increases the proportion of males among her progeny ($\bar{x} = 14\%$) (Fig. 17.6b). When females oviposit on previously parasitized pupae, they lay fewer eggs ($\bar{x} = 9$) and the proportion of males ranges from 1.00 to approximately 0.25 depending on the number of eggs the second female adds and in keeping with the likely

degree of LMC (Werren 1980a) (Fig. 17.6c). If the second female lays only a few eggs, she maximizes her reproduction (in terms of genes in grand-progeny) by making all eggs males and exploiting the scarcity of males in the first female's clutch; if the second female lays more eggs, she maximizes her reproduction by decreasing the proportion of sons to about 25% because LMC increasingly discounts the value of *her* sons as her progeny form an increasing proportion of the total. (The size of the second female's clutch is partly determined by the time since the first female's attack; if it is 24–48 hr or longer, the second female lays few eggs, in accord with the small share of host resources her brood is likely to garner.)

Although adjusting the proportion of males to the degree of local mate competition may have the effect of population regulation (I. Walker 1967), the details of the phenomenon do not support population regulation as a function: females increase their individual contributions to the overall gene pool by investing in higher proportions of males when populations are more dense.

Colwell (1981) and Wilson and Colwell (1981) emphasize that when two or more females contribute to a small mating group, female-biased sex ratios are selected because of the enhanced reproduction of the group (relative to groups with sex ratios closer to equality) and that within such a group, females having sex ratios closer to equality outreproduce those that make the group successful. In essence a female is confronted with winning within a group at the cost of losing populationwide or losing within a group but winning populationwide. Females that win populationwide are prevalent.

17.4.5 Sexes Unequal in Cost to Parent

Unequal sex-specific costs to the parent have also received attention (Fisher 1930, 1958, Trivers & Hare 1976, Maynard Smith 1978). Sons and daughters cost equally in most insects but not in sexually dimorphic Hymenoptera that progressively provision their larvae or provide sufficient stores at the time the egg is laid. In such cases the larger morph, most often female, receives correspondingly more provisions. As examples, cicada killers (*Sphecius speciosus*) usually give one cicada to their male progeny and two to female progeny (Lin 1979), while a solitary vespid wasp (*Euodynerus foraminatus*) provisions sons with about 130 mg of caterpillars and daughters with 204 mg (Cowan 1979). If such complications as LMC and uncertainties in assessing costs of the sexes are ignored, the predicted sex ratio should be the inverse of the cost ratio—a species in which females cost twice as much as males should manifest twice as many males as females. Trivers and Hare (1976) reported male-biased sex ratios (at emergence) for 11 of 13 species of solitary bees and wasps having females heavier than males. However, the degree of bias showed no sensitivity to the degree of weight discrepancy. Unequal costs are common among eusocial insects, but here

sex ratios are further complicated by rearing being the function of workers with genetic interests different from their parents and one another (Trivers & Hare 1976, Alexander & Sherman 1977, Noonan 1978, Macevicz 1979, Metcalf 1980).

17.4.6 Other Eventualities

Other eventualities bear on evolution of sex ratio. For example, extra-chromosomal factors, including microorganisms in the cytoplasm, can cause females to produce all-female or all-male progenies (Poulsen & Sakaguchi 1961, Johnson 1977, Werren et al. 1981, Skinner 1982). Some "species" with female-biased sex ratios are actually two or more genetically isolated lineages: a bisexual line, presumably with a 50:50 sex ratio, and one or more all-female lines in which females mate with males of the bisexual line but produce only daughters genetically like themselves—the male genome is not incorporated in the zygote (e.g., bark beetle, Bakke 1968; geometrid moth, Mitter & Futuyma 1977). More in keeping with the subject of population regulation is *autoparasitism,* a phenomenon known in certain aphel-inid wasps in which males develop as hyperparasites of conspecific females (Fig. 17.7) (Flanders 1967, 1969, Williams 1977). Data on autoparasitism are insufficient to guide speculation—for instance, it is unknown whether females, in nature, ever parasitize their own daughters. Colgan and Taylor (1981) proposed a model that presumes they may. R. I. Sailer (pers. comm.) showed that autoparasitism in *Encarsia lahorensis,* a citrus whitefly parasite recently introduced to Florida, has had the effect of population regulation. When parasitism of the whitefly hosts reached high levels, ovipositing fe-males killed increasing proportions of the females of the next generation as they produced sons—the only, or cheapest, progeny they could produce under the circumstances. Consequently, the next generation of parasites had less potential for further reducing its host population. *Physcus seminotus* on Mauritius (Williams 1977) illustrates the complexities associated with autoparasitism (see Fig. 17.7g): Prior to 1969 (Fig. 17.7g, black arrows and bold numbers), the sugar cane scale insect (*Aulacapsis tegalensis*) was para-sitized by an encyrtid wasp (*Adelencyrtus miyarai*) that was parasitized by a eulophid wasp (*Tetrastichus* sp.). Emergents from parasitized scales in 1968–69 were 33.5% *A. miyarai,* 65% *T.* sp., and 1.5% "other." The introduction of the aphelinid, *Physcus seminotus,* in which the female is a primary parasite of the scale insect and the male develops as a hyperparasite of any parasite in a scale (including a conspecific male!) complicated the trophic relations and changed the proportions of emergents, in 1975, to those in parentheses (Fig. 17.7g). Why are there so few males of *P. seminotus?* What determines population density of *P. seminotus?*

Much is yet to be learned of insect sex ratios and their effects on pop-ulation growth and stability. Improved understanding will have important applications in parasite rearing and biological control.
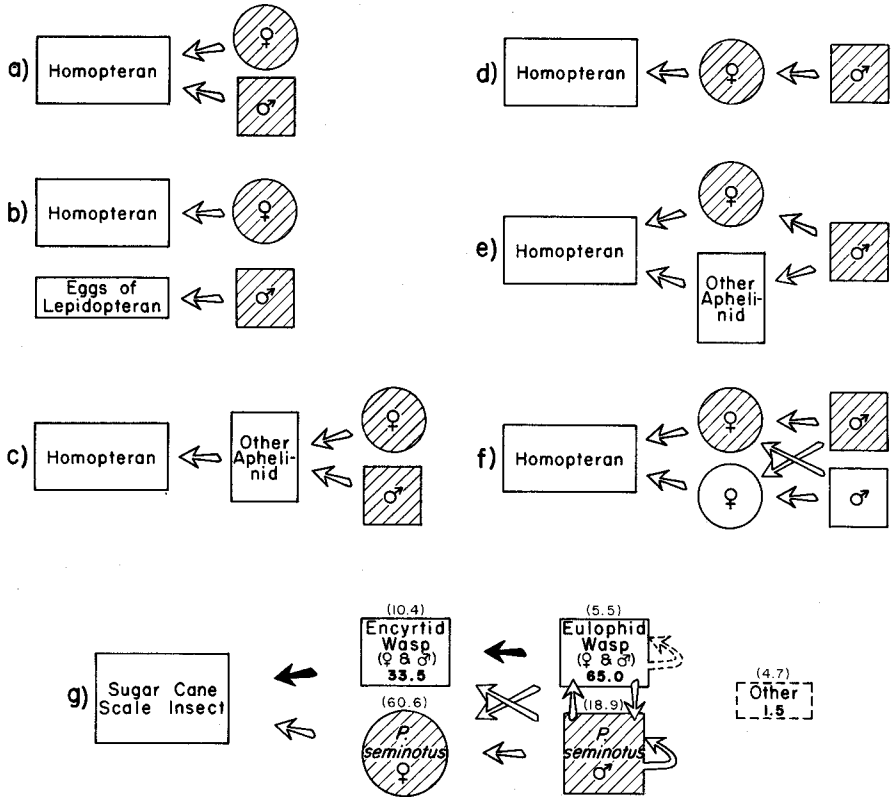
Fig. 17.7 Trophic relations of aphelinid wasps, including examples of autoparasitism (*d-g*). Cross-hatched squares and circles represent the males and females of the subject species. (a) primary parasites; female endoparasitic, male ectoparasitic; (b) primary endoparasites, sexes on different hosts; (c) hyperparasites; (d) obligatory autoparasitism (male develops as parasite of conspecific female); (e) facultative autoparasitism; (f) reciprocal facultative autoparasitism. Further complexities include females that develop parthenogenetically and females that lay male-producing eggs on healthy hosts with the eggs hatching only after another parasite has used the host to develop. (a–f from descriptions in Flanders 1967, 1969, no specific examples or data given for some categories); (g) *Physcus seminotus* on Mauritius (Williams 1977). (See text for explanation.)

17.5 DEATH AND SENESCENCE

Emerson (1960, p. 325) wrote that "among the attributes of the individual organism that would seem to be explicable only through the selection of whole population systems is the intrinsic limitation of the life-span and the incorporation of innate death mechanisms," and (p. 343) that "the innate regulation of numbers in unitary populations involves the evolution of death mechanisms in individuals." But Medawar (1952, 1957) and Williams (1957) had already shown that senescence should be expected as a side

effect of individual selection. Williams's theory, later refined by Hamilton (1966) and Emlen (1970), depends on selection diminishing in force beginning with the age of first reproduction. Even without senescence, residual expected reproduction of each successive age is less and less because mortality from external causes continues.* Since the cumulative remaining reproduction diminishes with adult age, an allele that increases survival in young adults but reduces it in old adults will generally cause an overall increase in reproduction and therefore be increased by natural selection. Any modifying gene that postpones detrimental effects of other genes (i.e., to ages with less residual expected reproduction) will likewise increase in frequency over alleles that allow an earlier adult age of onset—but not over those that prevent the bad effect altogether: Selection indirectly causes senescence but acts directly against it, though less and less effectively at ages with lower and lower residual reproductive probability. Experiments using *Drosophila* and *Tribolium* provide support for Williams's theory (e.g., Rose & Charlesworth 1980).

Senescence need not be regarded as an adaptation at any level; it can instead be viewed as "a group of adaptively unfavorable morphogenetic changes that were brought in as side effects of otherwise favorable genes and which have only been partly expurgated by further selection" (Williams 1957, p. 402).

17.6 DENSITY-RELATED GENETIC CHANGES

Different alleles may be favored at high and low population densities, or in a population that is rapidly expanding versus one that is constant or declining. For example, optimal mate finding or predator-deterring behaviors may change with density, and different fertility schedules maximize reproduction in increasing and decreasing populations. To what extent such changes stabilize populations is by no means clear.

The common denominator of density-related genetic changes should be improved individual adaptation to the new density or rate or direction of population change. The specific adaptations need have nothing in common as to how they affect future density or change. For example, assume that a population has reached a higher density than it has had in the past. One adaptation that might result is aggressive defense of resources needed for reproduction (see Table 17.1; also Chitty 1967). This could reduce population density at the same time it increased natality. Another adaptation that might result is reduced minimal size for adults—permitting under-

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*"Residual expected reproduction of each successive age" is from the vantage point of the zygote, *not* from the vantage point of an individual that has attained the specified age. Residual expected reproduction declines from the age of first reproduction and never increases [see Williams (1957) and Hamilton (1966) for fuller explanation]; it is not the same as Fisher's (1930) reproductive value.

nourished individuals some chance of reproduction. This could result in higher numbers of adults but might have no effect on natality or even decrease it. Further complexities could be introduced—such as time lags and frequency-dependent selection—but it should already be evident that density-related genetic changes do not reliably lead to population regulation.

Models relating density change and genetic change include those by Ford (1964), Chitty (1967), Carson (1968), Ayala (1968), and Tamarin (1978).

17.7   COEVOLUTION AND GENETIC FEEDBACK

Except in the most physically stressed, species-poor ecosystems evolution in any one species influences evolution in many others. This occurs because each species is important to the reproductive success of individuals of numerous other species—as food, enemies, allies, competitors, shelter, transportation, Batesian models, or any of a myriad other ways. With any two-species interaction, a change in one species will change the effective environment of the other. Numerous aspects of coevolution are discussed in Chapters 20–22. Here the discussion will center on David Pimentel's proposal (1961, 1968) that populations of herbivores, parasites, and predators are sometimes regulated by a "genetic feedback mechanism." Pimentel reasoned that a host (or prey) population would change genetically in response to the feeding pressure of a herbivore (or parasite, or predator) population and become more resistant to attack—thereby decreasing the reproduction and population level of the herbivore. With lower herbivore densities, the host population might lose some of its resistance and the herbivore population might regain some of its former numbers. "After many such cycles, the numbers of the herbivore populations are ultimately limited, and stability results" (Pimentel 1968:1433).

That there are reciprocal evolutionary changes between herbivore and host or predator and prey conforms to observations and to theory, but numerical stability is not a necessary consequence. Species that have evolved together exist at all population levels, and species that were at one level in the past are at other levels today. Genetic feedback has no "setpoint." It can result in abundance, rarity, or extinction. In fact Van Valen (1973, "Red Queen's Hypothesis"*[3]) proposed that evolutionary interactions generate instabilities that are the principal cause of extinctions. He noted that the effective environment of every species is continually deteriorating because of evolution in other species—the herbivore that becomes better at finding its food reduces the fitness of its host; the plant that becomes more difficult to digest reduces the fitness of its herbivores, and so on. Only those species that change survive; yet changes evoke changes in other spe-

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*"Now, here, you see, it takes all the running you can do, to keep in the same place." (L. Carroll, *Through the Looking Glass.*)

cies, diminishing or reversing the benefits. The ultimate result is extinction at a stochastically constant rate.

Genetic feedback may influence population numbers, but there is no evidence of it stabilizing them other than as an incidental effect of evolution in other contexts. [See Huffaker et al. (1976) for a critique of the role of genetic feedback in population regulation.]

17.8 FINAL REMARKS

As stated at the outset, whether self-regulation occurs in insect populations depends on how *self-regulation* is defined. Phenomena that regulate insect population density include many that are poorly understood, but none so far have been demonstrated to evolve through group selection opposing selection at lower levels. Since *self-regulation* has often been used with precisely this group selection connotation, the expression had best be avoided and unsullied ones substituted: for example, *regulation; population stabilizing effects.*

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REFERENCES

Alcock, J. 1979. Pp. 381–402 in Blum and Blum (Eds.), 1979, referenced here.

Alcock, J. 1980. *Am. Sci.* **68:** 146–153.

Alcock, J., E. M. Barrows, G. Gordh, L. J. Hubbard, L. Kirkendall, D. W. Pyle, T. L. Ponder, and F. G. Zalom. 1978. *Zool. J. Linn. Soc., Lond.* **64:** 293–326.

Alexander, R. D. and G. Borgia. 1978. *Annu. Rev. Ecol. Syst.* **9:** 449–474.

Alexander, R. D. and P. W. Sherman. 1977. *Science* **196:** 494–500.

Andersen, F. S. 1961. *Oikos* **12:** 1–16.

Askew, R. R. 1971. *Parasitic Insects.* Elsevier, New York.

Assem, J. van den. 1971. *Neth. J. Zool.* **21:** 373–402.

Ayala, F. J. 1968. *Science* **162:** 1453–1459.

Baker, R. R. 1972. *J. Animi. Ecol.* **41:** 453–469.

Bakke, A. 1968. *Can. Entomol.* **100:** 640–648.

Barber, G. W. 1936. USDA Tech. Bull. 499.

Bell, W. J., S. Robinson, M. K. Tourtellot, and M. D. Breed. 1978. *Z. Tierpsychol.* **48:** 203–218.

Bick, G. H. and J. C. Bick. 1965. *Ecology* **46:** 461–472.

Bitzer, R. J. and K. C. Shaw. 1980. *J. Res. Lepidoptera* **18:** 36–49.

Blais, J. R. 1953. *Can. Entomol.* **93:** 446–448.

Blum, M. S. and N. A. Blum (Eds.). 1979. *Sexual Selection and Reproductive Competition in Insects.* Academic, New York.

Bodmer, W. F. and A. W. F. Edwards. 1960. *Hum. Genet.* **24:** 239–244.

Bradbury, J. W. 1981. Pp. 138–169 in R. D. Alexander and D. W. Tinkle (Eds.), *Natural Selection and Social Behavior.* Chiron, New York.

Brockmann, H. J. and R. Dawkins. 1979. *Behaviour* **71:** 203–245.

Brower, L. P. 1961. *Physiol. Zool.* **34:** 287–296.

Bryant, S. H., A. T. Beckenbach, and G. A. Cobbs. 1982. *Evolution* **36:** 27–34.

Burk, T. 1979. *An Analysis of Social Behavior in Crickets.* Ph.D. Thesis, Oxford University, Oxford. (Univ. Microfilms International, Ann Arbor, Mich.)

Burk, Theodore. 1983. *Fla. Entomol.* 66: 330–344.

Bush, G. L. 1969. *Evolution* **23:** 237–251.

Cade, W. H. 1979. Pp. 343–379 in Blum and Blum (Eds.), 1979, referenced here.

Campanella, P. J. and L. L. Wolf. 1974. *Behaviour* **51:** 49–87.

Capinera, J. L. and P. Barbosa. 1976. *Oecologia* **26:** 53–64.

Carson, H. L. 1968. Pp. 123–137 in R. C. Lewontin (Ed.), *Population Biology and Evolution.* Syracuse University Press, Syracuse, N.Y.

Charnov, E. L. 1982. *The Theory of Sex Allocation.* Princeton University Press, Princeton, N.J.

Charnov, E. L., R. L. Los-DenHartogh, W. T. Jones, and J. van den Assem. 1981. *Nature* **289:** 27–33.

Chitty, Dennis, 1960. *Can. J. Zool.* **38:** 99–113.

Chitty, Dennis. 1967. *Proc. Ecol. Soc. Aust.* **2:** 51–78.

Cirio, U. 1971. *Redia* **52:** 577–600.

Colgan, P. and P. Taylor. 1981. *Am. Natur.* **117:** 564–566.

Colwell, R. K. 1981. *Nature* **290:** 401–404.

Corbet, P. S. and A. Griffiths. 1963. *Proc. Roy. Entomol. Soc. A.* **38:** 125–135.

Cowan, D. P. 1979. *Science* **205:** 1403–1405.

Crombie, A. C. 1944. *J. Exp. Biol.* **20:** 135–151.

Davies, N. B. 1978a. *Anim. Behav.* **26:** 138–147.

Davies, N. B. 1978b. Pp. 317–350 in J. R. Krebs and N. B. Davies (Eds.), *Behavioural Ecology, an Evolutionary Approach.* Sinauer, Sunderland, Mass.

Dawkins, R. 1980. Pp. 331–367 in G. W. Barlow and J. Silverburg (Eds.), *Sociobiology: Beyond Nature/Nurture.* Westview, Boulder, Colo.

Dawkins, R. 1982. *The Extended Phenotype: The Gene as the Unit of Selection.* W. H. Freeman, San Francisco.

DeBach, P. 1944. *Ann. Entomol. Soc. Am.* **37:** 70–74.

Denlinger, D. L. 1981. *Evolution* **35:** 1247–1248.

Duelli, P. 1981. *Res. Popul. Ecol.* **23:** 193–209.

Eberhard, W. G. 1979. Pp. 231–258 in Blum and Blum (Eds.), 1979, referenced here.

Edmunds, M. 1976. *Zool. J. Linn. Soc.* **58:** 1–37.

Eickwort, K. R. 1973. *Am. Natur.* **107:** 452, 453.

Emerson, A. E. 1960. Pp. 307–348 in S. Tax (Ed.), *Evolution after Darwin,* Vol. 1. University of Chicago Press, Chicago.

Emlen, J. M. 1970. *Ecology* **51:** 588–601.

Feaver, M. 1983. Pp. 205–239 in D. T. Gwynne and G. K. Morris (Eds.), *Orthopteran Mating Systems: Sexual Competition in a Diverse Group of Insects.* Westview, Boulder, Colo.

Fisher, R. A. 1930. *The Genetical Theory of Natural Selection.* Oxford University Press, Oxford.

Fisher, R. A. 1958. *The Genetical Theory of Natural Selection* (rev. ed.). Dover, New York.

Fisher, R. A. and D. O. Greenbank. 1979. Pp. 220–229 in R. L. Raab and G. G. Kennedy (Eds.), *Movement of Highly Mobile Insects: Concepts and Methodology in Research.* N. C. State Univ., Raleigh.

Flanders, S. E. 1967. *Entomophaga* **12:** 415–427.

Flanders, S. E. 1969. *Entomophaga* **14:** 335–346.

Ford, E. B. 1964. *Ecological Genetics.* Methuen, London.

Fox, L. R. 1975a. *Annu. Rev. Ecol. & Syst.* **6:** 87–106.

Fox, L. R. 1975b. *Ecology* **56:** 933–941.

Gautier, J. Y. 1978. *Insectes Sociaux* **25:** 289–301.

Ghiselin, M. T. 1974. *The Economy of Nature and the Evolution of Sex.* University of California Press, Berkeley.

Giesel, J. T. 1972. *Am. Natur.* **106:** 380–387.

Gillett, J. D., E. A. Roman, and V. Phillips. 1977. *Proc. Roy. Soc. Lond. B.* **196:** 223–232.

Gilpin, M. E. 1975. *Group Selection in Predator-Prey Communities.* Princeton University Press, Princeton.

Greany, P. D. and E. R. Oatman. 1972. *Ann. Entomol. Soc. Am.* **65:** 377–383.

Green, R. F. 1982. *J. Theor. Biol.* **95:** 43–48.

Guillot, F. S. and S. B. Vinson. 1972. *Nature* **235:** 169, 170.

Gwynne, D. T. 1980. *Behav. Ecol. Sociobiol.* **7:** 213–225.

Hamilton, W. D. 1966. *J. Theoret. Biol.* **12:** 12–45.

Hamilton, W. D. 1967. *Science* **156:** 477–488.

Hamilton, W. D. 1979. Pp. 167–220 in Blum and Blum (Eds.), 1979, referenced here.

Hamilton, W. D. and R. M. May. 1977. *Nature* **269:** 578–581.

Harrison, R. G. 1980. *Annu. Rev. Ecol. Syst.* **11:** 95–118.

Hickey, W. A. and G. B. Craig, Jr. 1966a. *Genetics* **53:** 1177–1196.

Hickey, W. A. and G. B. Craig, Jr. 1966b. *Can. J. Genet. Cytol.* **8:** 260–278.

Hokyo, N. and E. Kuno. 1977. *Res. Popul. Ecol.* **19:** 107–124.

Huffaker, C. B., F. J. Simmonds, and J. E. Laing. 1976. Pp. 41–78 in C. B. Huffaker and P. S. Messenger (Eds.), *Theory and Practice of Biological Control.* Academic, New York.

Jackson, D. J. 1966. *Trans. Roy. Entomol. Soc. Lond.* **118:** 23–49.

Johnson, C. 1977. *Evolution* **31:** 603–610.

Johnson, C. G. 1969. *Migration and Dispersal of Insects by Flight.* Methuen, London.

King, C. E. and P. S. Dawson. 1972. *Evol. Biol.* **5:** 133–227.

King, P. E., R. R. Askew, and C. Sanger. 1969. *Proc. Roy. Entomol. Soc. Lond.* **44:** 85–90.

Kolman, W. A. 1960. *Am. Natur.* **94:** 373–377.

Landahl, J. T. and R. B. Root. 1969. *Ecology.* **50:** 734–737.

Lees, A. D. 1961. *Roy. Entomol. Soc. Lond. Symp.* **1:** 68–79.

Lees, A. D. 1967. *J. Insect Physiol.* **13:** 289–318.

Leigh. E. G. Jr. 1970. *Am. Natur.* **104:** 205–210.

Lidicker, W. Z. Jr. 1962. *Am. Natur.* **96:** 29–33.

Lin, N. 1963. *Behaviour* **20:** 115–133.

Lin, N. 1979. *Proc. Entomol. Soc. Wash.* **81:** 269–275.

McClure, M. S. 1976. *Biotropica* **8:** 179–183.

Macevicz, S. 1979. *Am. Natur.* **113:** 363–371.

Marshall, A. G. 1981. *Ecol. Entomol.* **6:** 155–174.

Maynard Smith, J. 1964. *Nature* **201:** 1145–1147.

Maynard Smith, J. 1976. *Q. Rev. Biol.* **51:** 277–283.

Maynard Smith, J. 1978. *The Evolution of Sex.* Cambridge University Press, London.

Medawar, P. B. 1952. *An Unsolved Problem of Biology.* H. K. Lewis, London.

Medawar, P. B. 1957. *The Uniqueness of the Individual.* Basic Books, New York.

Metcalf, R. A. 1980. *Am. Natur.* **116:** 642–654.

Milne, L. J. and M. Milne. 1976. *Sci. Am.* **235:** 84–89.

Mitchell, P. L. 1980. *Ann. Entomol. Soc. Am.* **73:** 404–408.

Mitchell, R. 1970. *Am. Natur.* **104:** 425–431.

Mitter, C. and D. Futuyma. 1977. *Entomol. Exp. & Appl.* **21:** 192–198.

Moore, N. W. 1964. *J. Anim. Ecol.* **33:** 49–71.

Naylor, A. J. 1965. *Ecology* **46:** 341–343.

Nicolson, A. J. 1954. *Aust. J. Zool.* **2:** 9–65.

Noonan, K. M. 1978. *Science* **199:** 1345–1356.

Novitski, E. 1947. *Genetics* **32:** 526–534.

Oshima, K, H. Honda, and I. Yamamoto. 1973. *Agric. Biol. Chem.* **37:** 2679, 2680.

Otte, D. and A. Joern. 1975. *J. Anim. Ecol.* **44**(1): 29–54.

Palmer, T. J. 1978. *Nature* **274:** 583, 584.

Pimentel, D. 1961. *Am. Natur.* **95:** 65–79.

Pimentel, D. 1968. *Science* **159:** 1432–1437.

Polis, G. A. 1980. *Behav. Ecol. Sociobiol.* **7:** 25–35.

Polis, G. A. 1981. *Annu. Rev. Ecol. Syst.* **12:** 225–251.

Poulson, D. F. and B. Sakaguchi. 1961. *Science* **133:** 1489, 1490.

Price, P. W. 1970. *Science* **170:** 546, 547.

Price, P. W. 1975. *Insect Ecology.* Wiley, New York.

Prokopy, R. J. 1981. Pp. 181–213 in D. Nordlund, R. L. Jones, and W. J. Lewis (Eds.), *Semiochemicals: Their Role in Pest Control.* Wiley, New York.

Prokopy, R. J., P. D. Greany, and D. L. Chambers. 1977. *Environ. Entomol.* **6:** 463–465.

Prokopy, R. J. and R. P. Webster. 1978. *J. Chem. Ecol.* **4:** 481–494.

Rausher, M. D. and N. L. Fowler. 1979. *Biotropica* **11:** 96–100.

Ringo, J. M. 1976. *Ann. Entomol. Soc. Am.* **69:** 209–214.

Roff, D. A. 1975. *Oecologia* **19:** 217–237.

Rose, M. and B. Charlesworth. 1980. *Nature* **287:** 141, 142.

Rothschild, M. and L. M. Schoonhoven. 1977. *Nature* **266:** 352–355.

Rudinsky, J. A., P. T. Oester, and L. C. Ryker. 1978. *Ann. Entomol. Soc. Am.* **71:** 317–321.

Schowalter, T. D. and W. G. Whitford. 1979. *Am. Midland Natur.* **102:** 182–184.

Schuster, J. C. 1975. *Comparative Behavior, Acoustical Signals and Ecology of New World Passalidae (Coleoptera).* Ph.D. thesis, Univ. Fla. Gainseville. (Univ. Microfilms International, Ann Arbor, Michigan.)

Shaw, M. J. P. 1970a. *Ann. Appl. Biol.* **65:** 191–196.

Shaw, M. J. P. 1970b. *Ann. Appl. Biol.* **65:** 197–203.

Shaw, M. J. P. 1973. *Ann. Appl. Biol.* **74:** 1–7.

Shaw, R. F. and J. D. Mohler. 1953. *Am. Natur.* **87:** 337–342.

Shroyer, D. A. and G. B. Craig, Jr. 1981. *Environ. Entomol.* **10:** 147–152.

Skinner, S. W. 1982. *Science* **215:** 1133, 1134.

Solbreck, C. 1978. Pp. 195–217 in H. Dingle (Ed.), *Evolution of Insect Migration and Diapause.* Springer-Verlag, New York.

Southwood, T. R. E. 1962. *Biol. Rev.* **37:** 171–214.

Spieth, H. T. 1978. *Evolution* **32:** 435–451.

Springett, B. P. 1968. *J. Anim. Ecol.* **37:** 417–424.

Stearns, S. C. 1976. *Q. Rev. Biol.* **51:** 3–47.

Steiner, A. L. 1975. *Quaest. Entomol.* **11:** 113–127.

Stinner, R. E., R. L. Rabb, and J. R. Bradley, Jr. 1977. *Proc. 15th Int. Congr. Entomol., 1976,* pp. 622–642.

Sutherland, O. R. W. 1969. *J. Insect Physiol.* **15:** 2179–2201.

Suzuki, Y. and Y. Iwasa. 1980. *Res. Popul. Ecol.* **22:** 366–382.

Tamarin, R. H. 1978. *Am. Natur.* **112:** 545, 555.

Taylor, R. A. J. and L. R. Taylor. 1979. Pp. 1–27 in R. M. Anderson, B. D. Turner, and L. R. Taylor (Eds.), *Population Dynamics.* Blackwell, London.

Trivers, R. L. 1972. Pp. 136–179 in B. Campbell (Ed.), *Sexual Selection and the Descent of Man, 1871–1971.* Aldine, Chicago.

Trivers, R. L. 1974. *Am. Zool.* **14:** 249–264.

Trivers, R. L. and H. Hare. 1976. *Science* **191:** 249–263.

Tschinkel, W. R. 1978. *Physiol. Zool.* **51:** 300–313.

Tschinkel, W. R. 1981. *Anim. Behav.* **29:** 990–996.

Tychsen, P. H. 1977. *J. Aust. Entomol. Soc.* **16:** 459–465.

Uéda, T. 1979. *Res. Popul. Ecol.* **21:** 135–152.

Urquhart, F. A. and N. R. Urquhart. 1978. *Can. J. Zool.* **56:** 1756–1764.

van Lenteren, J. C. 1976. *Neth. J. Zool.* **26:** 1–83.

Van Valen, L. 1973. *Evol. Theory* **1:** 1–30.

Vinson, S. B. 1976. *Annu. Rev. Entomol.* **21:** 109–133.

Waage, J. K. 1982. *Ecol. Entomol.* **7:** 103–112.

Wade. M. J. 1978. *Q. Rev. Biol.* **53:** 101–114.

Walker, I. 1967. *Ecology* **48:** 294–301.

Walker, T. J. 1980. *Evol. Biol.* **13:** 219–260.

Weaver, J. E. and R. A. Sommers. 1969. *Annu. Entomol. Soc. Am.* **62:** 337–342.

Wellington, W. G. 1977. *Environ. Entomol.* **6:** 1–8.

Werren, J. H. 1980a. *Science* **208:** 1157–1159.

Werren, J. H. 1980b. *Studies in the Evolution of Sex Ratios.* Ph.D. thesis, University of Utah, Salt Lake City. (Univ. Microfilms International, Ann Arbor, Mich.)

Werren, J. H., S. W. Skinner, and E. L. Charnov. 1981. *Nature* **293:** 467, 468.

Wheeler, W. M. 1930. *Demons of the Dust, a Study in Insect Behavior.* W. W. Norton, New York.

Whitham, T. G. 1979. *Nature* **279:** 324, 325.

Williams, G. C. 1957. *Evolution* **11:** 398–411.

Williams, G. C. 1966. *Adaptation and Natural Selection. A Critique of Some Current Evolutionary Thought.* Princeton University Press, Princeton, N.J.

Williams, G. C. (Ed.). 1971. *Group Selection.* Aldine-Atherton, Chicago.

Williams, G. C. 1975. *Sex and Evolution.* Princeton University Press, Princeton, N.J.

Williams, J. R. 1977. *Entomophaga* **22:** 345–350.

Wilson, D. S. 1975. *Proc. Nat. Acad. Sci.* **72:** 143–146.

Wilson, D. S. 1980. *The Natural Selection of Populations and Communities.* Benjamin Cummings, Menlo Park, Calif.

Wilson, D. S. and R. K. Colwell. 1981. *Evolution* **35:** 882–897.

Wilson, E. O. 1975. *Sociobiology, the New Synthesis.* Harvard University Press, Cambridge, Mass.

Wilson, F. 1961. *Aust. J. Zool.* **9:** 739–751.

Wood, R. J. and M. E. Newton. 1977. *Proc. 15th Int. Congr. Entomol., 1976,* pp. 97–105.

Wylie, H. G. 1966. *Can. Entomol.* **98:** 645–653.

Wynne-Edwards, V. C. 1962. *Animal Dispersion in Relation to Social Behaviour.* Oliver and Boyd, Edinburgh.