The Evolution and Genetics of Migration in Insects

DEREK A. ROFF AND DAPHNE J. FAIRBAIRN

Because areas suitable for growth and reproduction are often ephemeral, a primary selective force in the evolution of migratory behavior in insects is the need to colonize new habitats. However, both migration itself and flight capability reduce present reproductive success. Thus the long-term fitness benefit of migration, the colonization of new habitats, is balanced by a short-term reduction in fitness, the result being that variation for migratory ability is preserved in a population. Migration is but one component of a wide suite of functionally connected traits that together form a migratory syndrome. Genetic variation is found in all components of the migratory syndrome, and selection for migration results in a change in the frequency of expression of these components, which can be analyzed and predicted using the mathematics of quantitative genetics. We illustrate this evolutionary interplay with the example of the evolution of wing dimorphism in the sand cricket.

Keywords: insect migration, dimorphism, genetics, evolution

The vast majority of insect species (> 99.9%) belong to the group known as the Pterygota, meaning that they are winged or that they have descended from winged ancestors. Movement in this group may be by flying, walking, swimming, ballooning, or, in a few cases, phoresy (i.e., hitching a ride on another organism, as fleas and lice do). Migratory movements by walking do occur and can be quite spectacular, as shown by the migratory movements of immature locusts (*Locusta migratoria*) and adult Mormon crickets (*Anabrus simplex*) (Cheke and Tratalos 2007). In general, however, large-scale movement occurs by aerial means, and the distances that can be covered by flight are orders of magnitude greater than can typically be covered on foot. In this article, we discuss migration by flight in insects, with particular attention to the genetic basis of the traits that contribute to the migratory tendency and their influence on the evolution and frequency of migration in contemporary populations.

The world is heterogeneous in both time and space, and migration is an evolved response to this heterogeneity. We may reasonably hypothesize that migration among habitat patches is favored whenever environments are likely to vary in time and space, a hypothesis supported by both theoretical and empirical studies (Southwood 1962, Dingle 1989, 1996, Roff 1990a, 1994, Dingle and Drake 2007). These studies demonstrate that genetic lineages in which at least some individuals migrate each generation persist longer than lineages that entirely forgo migration and hence become restricted to single habitat patches. However, migration is a risky strategy that carries distinct individual fitness costs. Migrating individuals may be more susceptible to predation, or may be carried by winds far away from any habitable area (Gatehouse 1997). Thus they may fail to reach a suitable new habitat patch. Even if they are successful, there is no guarantee that the newly colonized habitat patch will be more suitable than the one abandoned. In addition to these overt risks, flight is very energetically expensive (Wegener 1996), and the energy that is used in migratory flight may reduce subsequent fecundity or ability to compete for mates.

The evolution of migration as a strategy in the life cycles of insects reflects a balance between these conflicting costs and benefits, and in particular between the short-term (i.e., within-generation) advantages of not migrating and the longer-term advantages of colonizing new habitats. In many species, this has led to migratory polymorphisms in which only some of the individuals in any given generation undertake a migratory flight (Fairbairn and Desranleau 1987, Gatehouse 1989, Dingle 1996, Kent and Rankin 2001). In extreme cases, the nonmigratory (sedentary) individuals lack fully developed wings or flight muscles, and hence are morphologically and physiologically incapable of flight (Southwood 1961, Roff 1986, 1990a, 1994, Denno et al. 1991, Roff and Fairbairn 1991, Dingle 1996, Zera and Denno 1997). Species in which

---

some individuals have reduced wings are particularly amenable for studies of insect migration because the sedentary morphs can be clearly distinguished from potential migrants, even in field populations. We utilize examples of such "wing-dimorphic" species in our consideration of the evolution and genetic basis of migration by flight.

A study by Stein (1977) of four wing-dimorphic weevil species colonizing newly seeded meadows nicely illustrates the trade-off between the costs and benefits of migration. In one species, *Sitona hispidula,* the wing dimorphism is known to be determined by a single locus with two alleles, with the short-winged morphology being dominant (i.e., heterozygotes have short wings; Jackson 1928), and hence it is reasonable to suppose that the dimorphism is genetically determined in the other three species as well. Because of intervening inhospitable terrain, short-winged (and hence flightless) individuals cannot colonize newly available patches, and hence we would predict that individuals with fully developed wings would comprise the initial colonists. However, in succeeding generations, the frequency of the short-winged morph in each patch would be expected to increase because of differential loss of long-winged individuals through emigration and the expected greater reproductive success (primarily fecundity) of the short-winged individuals, which do not invest in the production and maintenance of the flight apparatus. This is precisely what was observed (figure 1). In such a system, we would expect the equilibrium frequency of the two morphs to depend on the persistence time of patches and the degree to which the migratory polymorphism is genetically determined, an issue taken up in the next section.

The genetic basis of wing dimorphisms

Stein's (1977) weevils illustrate one of the common genetic systems underlying migratory polymorphisms. In almost all wing-dimorphic insects with holometabolous development (i.e., distinct larval, pupal, and adult stages, with complete
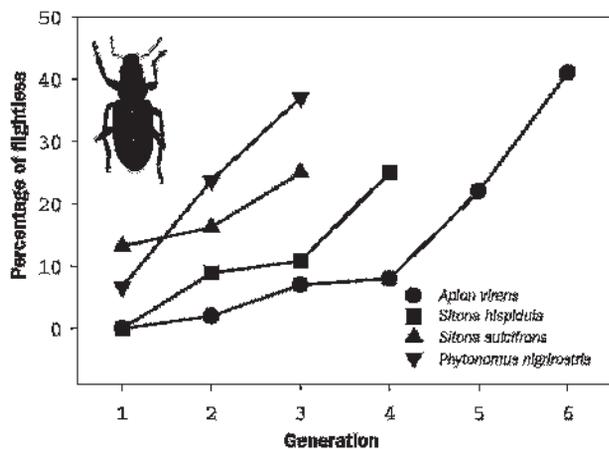
metamorphosis, as in Diptera, Coleoptera, and Lepidoptera), wing dimorphism is under the control of a single locus, with reduced wings being dominant. (As discussed below, wing reduction is accompanied by loss of the flight musculature.) This genetic system has probably been repeatedly favored because dominant alleles are fully expressed in heterozygous individuals, and hence are "available to selection" as soon as they occur. As noted above, all pterygote insects are descended from winged ancestors, and wing loss or reduction is thus the evolutionarily derived state. In the presence of selection against migration in a monomorphically long-winged population, a dominant mutation for wing reduction would spread much more quickly than a recessive mutation because the latter would not be expressed (and hence available to selection) until in sufficiently high frequency to be found in the homozygous state. While rare and primarily masked by the dominant, wild-type allele in heterozygous individuals, recessive mutant alleles also have a high probability of being lost from the population by chance, a process known as genetic drift. A dominant allele is immediately expressed, and hence any advantages accruing to such a mutation will be immediately realized. Simulation modeling (figure 2) demonstrates that in a heterogeneous environment a dominant mutation for wing reduction quickly spreads, but because the short-winged morph cannot colonize new habitats, an equilibrium is reached at which both morphs are maintained in the population, as seen in Stein's (1977) weevils.

Wing dimorphisms also occur in hemimetabolous insects (i.e., nymphal stages moult directly into the adult form, as in Hemiptera and Orthoptera). However, in these clades, wing dimorphisms are almost universally polygenic (influenced by

Figure 1. Increases in the percentage of flightless individuals (brachypters) in four species of weevils following colonization of newly seeded meadows. Data are from Stein (1977).
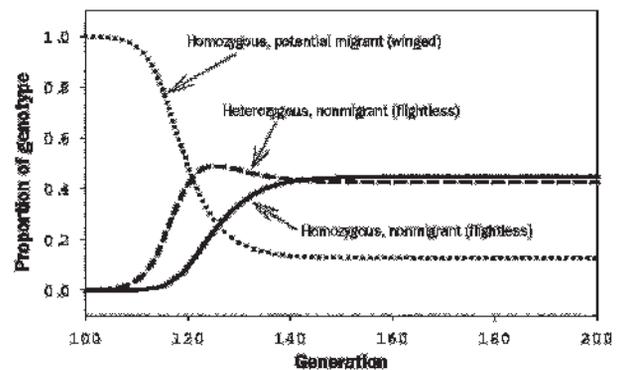
Figure 2. Simulation of the invasion of a dominant mutation that causes the loss or reduction of wings. Each habitat patch persists for 20 generations, with new patches arising at the same frequency. The simulation commences with a homozygous long-winged morph, and a single mutant allele is introduced after 100 generations. Long-winged individuals migrate from a patch with a probability of 0.4 and have a probability of 0.4 of finding another patch. Long-winged individuals have a reproductive success relative to the short-winged morph of 0.6. For further details, see Roff (2002, pp. 341–349).

the interactions of many loci). Why the control of wing production should be distributed in this way among insect lineages is not known. The polygenic system characteristic of hemimetabolous insects can be understood using the threshold model of quantitative genetics (Roff 1986), which we describe below. The methods of quantitative genetics are also appropriate for studying the joint evolution of the suite of behavioral, physiological, and morphological traits that underlie migratory behavior and have come to be known as the "migratory syndrome," wing dimorphism being only one of these.

Traits comprising the migratory syndrome

Before considering the genetic basis of migration further, we need to consider what traits comprise the migratory syndrome in insects. These are traits that are functionally related to the capability or tendency of individuals to undertake migratory movements. A partial list of such traits or trait types would include morphology, hormone titers, development time and growth rate, distribution of energy stores, flight propensity, and age-specific reproduction. We review these in turn below.

Morphology. The most obvious morphological correlate of migratory capability is possession of fully developed wings with associated flight musculature. As noted above, although all insects belonging to the group Pterygota descend from winged ancestors, flight muscles and wings have been secondarily lost or reduced in many species. Insects with reduced, nonfunctional wings are formally designated as brachypterous, micropterous, or apterous, depending on the degree of wing reduction, but we use the collective, vernacular term "short-winged" for any degree of wing reduction or loss resulting in loss of flight ability. Short-winged morphs are favored if the habitat is permanent and continuous, if there is a cost to the possession of the flight machinery, and if flight is not used for foraging or mating. Although some species become fully flightless, wing dimorphisms, as in Stein's (1977) weevils, are common in several insect orders (figure 3; Roff 1990a, Denno et al. 1991, Roff and Fairbairn 1991). In addition to wing reduction, the functional demands of migratory flight may lead to other morphological differences between migratory and nonmigratory morphs. For example, selection may favor large size in migrants to reduce water loss (by reducing the ratio of surface to volume) or because large size increases the energetic efficiency of flight (Roff 1977). Larger size of migratory forms has been found in both wing-dimorphic and monomorphically winged species (Hegmann and Dingle 1982, Fairbairn 1992). Selection for prolonged flight in the monomorphically winged lepidopteran *Spodoptera exempta* led to both increased flight duration and larger size (Gunn and Gatehouse 1993). In wing-dimorphic insects, the long-winged morph may have a larger thorax (to accommodate flight muscles) but a smaller abdomen than the short-winged morph (figure 3b, 3c; Fairbairn 1992), and differences in thorax shape are not uncommon (Fairbairn
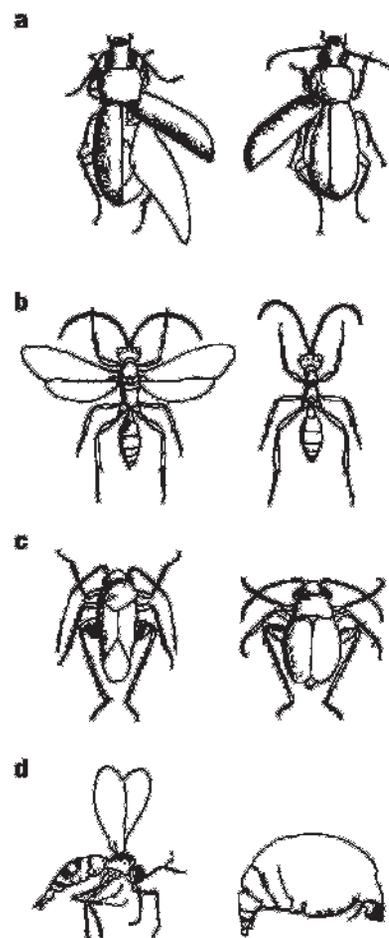
Figure 3. Four examples of dimorphic variation in wing morphology (flightless morph on the right). (a) In the carabid Pterostichus anthracinus*, there are no obvious differences between the morphs except for the size of the wings, which are hidden under the elytra (forewings). (b) The winged morph of the hymenopteran* Gelis corruptor *is distinguished both by the presence of wings and by an enlarged thorax. (c) There are major differences in body morphology in the bug* Halticus chrysolepis*. (d) Differences in body morphology are extreme in the dipteran* Plastosciara perniciosa*, even though the two morphs could be siblings. From Roff (1986).*

1992, Roff and Bradford 1998). In most species, these secondary differences between migratory and nonmigratory morphs tend to be quite subtle and can be revealed only by statistical analysis (figure 3a), but much more profound differences are not uncommon (figure 3d).

Hormone titers. Development in both vertebrates and invertebrates is controlled in large measure by age-specific changes in hormone titers (Nijhout 1994, Brakefield et al. 2003). Given the different ontogenetic trajectories of migrants and nonmigrants, it might be expected that hormonal profiles, both in terms of age-specific changes and of actual

titers, would differ between migrants and nonmigrants (Rankin 1989). Such differences have indeed been described, for both wing-dimorphic and long-winged species (Dingle and Winchell 1997, Fairbairn and Yadlowski 1997, Zera and Denno 1997). Some hormonal differences produce long-term irreversible effects, such as the production of a winged or wingless morph, whereas other effects may be inducible and occur only if the individual actually takes a migratory flight. One particularly important hormonal pathway involved in regulation of the suite of traits associated with migration appears to be the juvenile hormone (JH) pathway (Southwood 1961, Zera and Denno 1997). In several wing-dimorphic species, high titers of JH during key developmental periods have been shown to be correlated with the subsequent development of the nonmigratory morph (Zera 2004). The titer of JH during this critical period is in turn regulated at least partially by the activity of the enzyme JH esterase (JHE), such that high levels of JHE are associated with low titers of JH and induction of the migratory, fully winged morph. We will return to this hormonal pathway later in a specific example of migratory traits in the sand cricket (*Gryllus firmus*).

Development time and growth rate. Differences in development time (i.e., total duration of juvenile stages) between migrants and nonmigrants are generally correlated with differences in adult morphology. For example, where the migratory morph is larger, development must be prolonged, or growth rate increased, relative to that of the nonmigratory morph. It is also possible that only larvae that are in conditions in which they suffer no resource restriction achieve the status allowing successful migration, and in this case we might find that the future migrants combine high growth rate with short development time so that they eclose (molt into the adult stage) early and at the largest size (Roff 1995).

Distribution of energy stores. Migrants must synthesize and store flight fuels such as triglycerides. This is energetically demanding and may divert energy from early investment in reproduction (Zera and Denno 1997). Nonmigrants do not have to store flight fuels and hence can channel resources directly into the production of eggs or, in the case of males, into activities that attract mates, allowing them to make a greater reproductive investment early in adult life (Roff and Fairbairn 1991).

Flight propensity. Although individuals with fully developed wings are often characterized as flight-capable or migratory morphs, flight propensity often varies considerably among long-winged individuals within any given population (Fairbairn and Desranleau 1987). Long-winged individuals typically vary in the propensity to initiate a flight, the mean duration of flight, and the propensity to terminate flight in the presence of particular cues such as host plants or habitat types. Some individuals may show little or no propensity to initiate long-distance flights, whereas other individuals may readily take flight and may require long-duration flights be-

fore they are behaviorally and physiologically ready to settle down.

Genetic variation for migratory behavior is often inferred from demonstrations of genetic variation in the propensity for long-distance flight, generally measured as flight duration. The simplest way to do this is to raise a group of long-winged individuals under constant conditions, thereby ensuring that any variation among individuals is not due to different conditions experienced during development. In some species this technique reveals distinct groups of migrants and nonmigrants based on a bimodal pattern of flight durations, as illustrated by the flight times of *Melanoplus sanguinipes* (figure 4). Thus, in these species, a dimorphism for migratory tendency occurs even in the absence of wing dimorphism. However, more typically, flight propensity shows continuous variation and there is no clear delineation between migrants and nonmigrants—for example, *Lygaeus kalmii* (Caldwell and Hegmann 1969), *S. exempta* (Gatehouse 1986), *Epiphyas postvittana* (Gu and Danthanarayana 1992), *Heliothis armigera* (Colvin and Gatehouse 1993), and *Cydia pomonella* (Schumacher et al. 1997). Because of this variation, it is appropriate in wing-dimorphic species to classify short-winged individuals as "nonmigrants," but long-winged individuals as only "potential migrants."

Age-specific reproduction. The onset of reproduction may be delayed until after the migration event. The separation of the adult life of an insect into a migratory phase followed by a reproductive phase is so common that it has received its own designation, the oogenesis-flight syndrome (Johnson 1969). The possible causes of the oogenesis-flight syndrome act at different levels and are not mutually exclusive. For example,
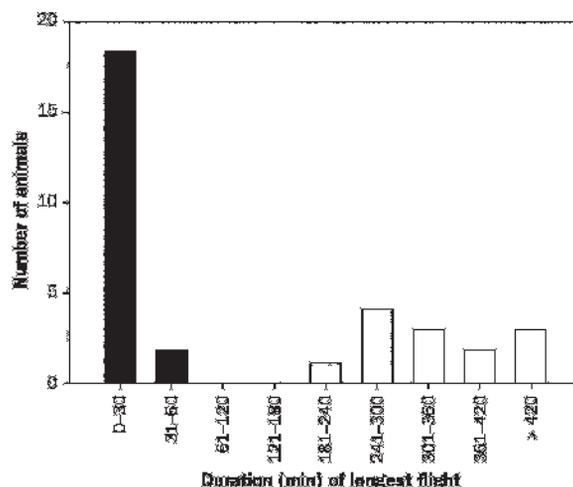
Figure 4. Distributions of durations of tethered flight by male offspring of field-collected Melanoplus sanguinipes. *Each grasshopper was given three opportunities to fly to voluntary cessation, and the longest flight duration was retained. Nonmigrants were defined as those that flew for less than 60 minutes. Redrawn from Kent and Rankin (2001).*

physiological trade-offs may preclude simultaneous migration and reproduction if there is direct competition for resources between reproductive organs or tissues and the flight apparatus (mainly the energy required to maintain flight muscles and to fuel flight). In the African armyworm (*S. exempta*), reproduction was reduced after a prolonged flight unless females had access to sucrose (Gunn et al. 1988), while in the fruitfly (*Drosophila melanogaster*), reproduction was reduced even with the provision of food after a flight (Roff 1977).

Aerodynamic constraints may also favor separation of flight and reproduction, particularly for females, if the weight or bulk of eggs increases the cost of flight or makes the female aerodynamically unstable, reducing her flight distance and making her more vulnerable to aerial predators such as bats. Finally, if migration is seasonal or in response to deteriorating habitat conditions, selection should favor postponing oviposition until after the migratory flight. Whatever its cause, one consequence of the oogenesis-flight syndrome is that energy is diverted into flight early in adult life and the reproductive potential of migrants tends to be reduced relative to that of nonmigrants. Thus, for example, the age-specific fecundity function for female migrants may show both a delay in its start and a general lowering at least until migration is completed and resources devoted entirely to reproduction.

The quantitative genetics of migration

Understanding the evolution and adaptive significance of migration requires knowledge of the patterns of variation and covariation among the suite of correlated traits that make up the migration syndrome, as well as the selective forces acting on them. Assessment of the genetic basis of these patterns is essential for predicting evolutionary trajectories or the frequencies of migratory and nonmigratory life histories within populations, which requires the framework of quantitative genetics. The evolutionary trajectory of a single trait can be predicted using the breeder's equation, $R = h^2 S$, where R is the response to selection, h^2 is the heritability of the trait, and S is the selection differential (i.e., the difference between the population mean and the mean of the parents contributing to the next generation).

The genetic component of this equation is the term "heritability," which technically is the ratio of the additive genetic variance to the phenotypic variance. A simple way to view this parameter is to consider a linear regression based on mean phenotypic values for full-sib families from a given population. If the mean offspring phenotype for each family is regressed against the parental mean ("midparent value"), the slope of the least-square regression is the heritability of the trait. Heritability varies between zero (no relationship) and one (all the phenotypic variance is due to additive genetic variance). The actual response to selection will depend on both the heritability of the trait and the strength of selection, but it is useful to estimate heritability, as it gives us an idea of how rapidly a population will respond to even a modest selection pressure.

In general, heritabilities over 0.40 can be regarded as high, whereas those below 0.10 are low. Morphological traits, such as body size, have heritabilities of about 0.50; life history traits, such as fecundity and development time, have heritabilities of about 0.25; and behavioral and physiological traits seem to be intermediate at about 0.30, though there are fewer data on these last two categories (Mousseau and Roff 1987).

The breeder's equation has been used successfully to predict variation in wing dimorphism among species and populations (Roff 1994). However, this approach implicitly assumes that migratory tendency is a purely dichotomous trait, with all long-winged individuals being migrants. This simplification ignores the potentially complex patterns of covariance among the suite of traits associated with migration, the continuity of the spectrum from migratory to nonmigratory, and the possibility of variation within each type even if there is bimodality in some components, such as wing morphology (Fairbairn and Desranleau 1987).

To incorporate these complexities, the simple breeder's equation can be expanded to include multiple correlated traits. The expanded model takes into account not only the heritabilities of each of the traits but the extent to which the genes that control each trait also influence other traits. This overlap of influence is measured by the genetic correlation between traits. This can be illustrated for the simple case of two correlated traits, X and Y. The expanded breeder's equation becomes $R_X = \beta_X h^2_X + \beta_Y h_X h_Y r_g$, where R_X is the response, in phenotypic standard deviation units, in trait X when selection is applied to traits X and Y. The heritabilities of X and Y are h^2_X and h^2_Y, respectively, and r_g is the genetic correlation between the two traits. The multivariate equivalents of the selection differentials are denoted β_X and β_Y and are called the selection coefficients (here measured in phenotypic standard deviations). Together they constitute the selection gradient for this suite of two traits.

This simple two-trait model can be expanded to multiple traits and serves to illustrate the complexity of predicting multivariate evolution. To predict the evolutionary trajectory of the suite of traits comprising the migratory syndrome, we must measure the heritabilities of each of the component traits as well as the genetic correlations between the pairs of traits. Unfortunately, such estimates require formal breeding designs and large sample sizes, and this has limited the number of studies adopting this multivariate approach. Few studies have measured even two or three of the components of the migratory syndrome. In our lab we have examined most of these components in the wing-dimorphic cricket *G. firmus*. We will use this work as an illustrative example, providing additional data on other species where possible.

The migratory syndrome in the sand cricket

The sand cricket is a fairly large cricket (adult weight approximately 0.7 g) found along beaches and other sandy areas from Florida to New Hampshire and on the island of Bermuda (figure 5). Both short-winged and long-winged
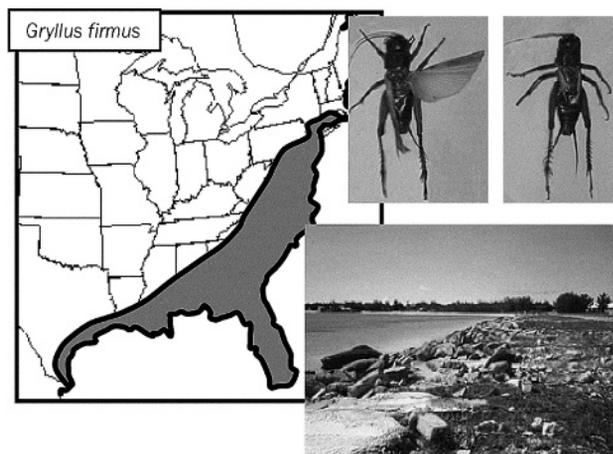
Figure 5. Map of the eastern United States showing the range of the sand cricket (Gryllus firmus). Modified from the Web map at http://buzz.ifas.ufl.edu/481m.htm, produced by Thomas J. Walker, University of Florida. In the upper right photos are two wing morphs; the top wing covers, called the tegminas, have been removed for clarity. The lower right photo shows a typical habitat (Bermuda). Photographs: upper right, Derek A. Roff; lower right, Daphne J. Fairbairn.

Figure 6: Trade-off between fecundity and migratory ability in the sand cricket. Short-winged females (light dots) have larger ovaries and hence produce more eggs than long-winged females (dark dots), and within long-winged crickets, ovary weight varies inversely with the size of the main flight muscles, the dorsolongitudinal muscles (DLM). The change in DLM size reflects the level of wing muscle histolysis, shown in the upper dissections: In the leftmost panel, the flight muscles (a) are fully developed and egg production (b) is low. The middle panel shows an intermediate stage at which flight muscle histolysis has begun (a) and egg production has increased. In the rightmost panel, the flight muscles are fully histolyzed and the egg number is large. Photographs: Derek A. Roff.

morphs are found in this species, with less than 2% of individuals having intermediate wing lengths. In northern areas there is a single generation each year, with adults occurring from August to October, whereas in Florida two peaks (July and October) of adult emergence are typical. Like other *Gryllus* species, the sand cricket is herbivorous but quite catholic in the range of its diet. (In the lab it grows well on rabbit chow.)

As predicted, on average, the fecundity of long-winged female *G. firmus* is substantially less than that of the short-winged morph (figure 6; note that the fecundity of long-winged females without flight muscles is similar to that of short-winged females; Roff 1984). In males, the duration of calling—an essential element for female attraction—is greatly reduced in the long-winged morph, with a consequent reduction in the number of females attracted (Crnokrak and Roff 1995). Not all long-winged individuals are capable of flight, as many histolyze their flight muscles (i.e., catabolize the muscle tissue) early in adult life. Most significantly, within the long-winged morph there is a trade-off between the reproductive components, fecundity and call duration, and the degree of histolysis of the main flight muscles, the dorsolongitudinal muscles (figure 6).

Given the above information, we initiated a long-term study of the underlying genetic architecture of the suite of traits associated with migratory capability in the sand cricket, which we outline here. A schematic of relationships is shown in figure 7, and we describe the relationships by trait category, as listed in the previous section.

Morphology. Although wing morphology in the sand cricket is bimodal, it is a polygenic trait. Its determination can be understood using the threshold model of quantitative genetics that assumes a continually distributed underlying trait, called the liability, and a threshold that determines the developmental trajectory. If the liability exceeds the threshold, development is shunted in one direction, say to the long-winged morph, whereas if the liability lies below the threshold, development is shunted in the direction producing the alternate morph. This model is useful for understanding the development of different morphologies and can also be applied to other types of dichotomous traits, such as the decision to migrate or not. Using this model, the heritability of the trait (which means the heritability of the liability) in *G. firmus* is 0.65, with values for other insects (mainly crickets) ranging from 0.30 to 0.98 (see table 13.1 in Roff and Fairbairn 2001). We estimated the heritability of the trait in *G. firmus* both by a pedigree analysis (i.e., comparing the frequency of the trait among relatives such as full sibs) and by artificial selection in which only males and females of a given morph were used as the parents for the subsequent generation. Within five generations, the selected lines changed from the starting proportion of 50% of each morph to more than 90% of the selected morph, which gives a good picture of how rapidly a population could change in the wild (Roff 1990b).

long-winged adults (Fairbairn and Yadlowski 1997, Zera and Huang 1999, Zera 2004, 2006). According to the threshold model, selection for an increase in the proportion of a given morph should produce a shift in the mean liability, which means that the average titer of JHE should increase in lines selected to be long-winged and decrease in lines selected to be short-winged. Such a shift was found in the selected lines described above (Fairbairn and Yadlowski 1997). While this supports the classic assumption that the JHE–JH pathway plays a critical role in wing morph determination, a more detailed examination of the genetic architecture indicates that other components also contribute to the liability. If, for example, JHE activity could be equated with the liability, we would expect the two traits to have the same heritability and a genetic correlation of one. In fact, the heritability of JHE activity is lower than that of the liability, and the genetic correlation is significantly less than one (figure 7; Roff et al. 1997), indicating that the liability for wing morph determination is a product of the interaction of several traits, with the JHE–JH pathway playing a major but not sole role in determining wing morphology.

The genetic covariation between JHE and wing morphology (as determined by variation in the liability discussed above; figure 7) that we observed in our laboratory populations has proved useful in understanding the complex patterns of variation among geographically disparate populations of *G. firmus* in the wild. For example, the quantitative genetic equations given above accurately predicted differences in mean JHE activity between populations in Florida (35% long-winged, low JHE) and Bermuda (95% long-winged, high JHE; Roff and Fairbairn 1999). Furthermore, the quantitative genetic threshold model predicted, and hence allowed us to understand, the counterintuitive result that the higher mean JHE activity in the Bermuda population as a whole was actually associated with lower mean JHE activity within each wing morph.

This apparent paradox occurs because the Bermuda population has a higher proportion of long-winged individuals, and hence higher mean JHE overall, than the Florida population. However, the evolutionary shift in the proportion of long-winged individuals has been associated with a shift in the threshold for wing induction such that less JHE is required to induce wing production in the Bermuda crickets. Thus, on average, long-winged crickets from Bermuda have lower JHE activity than long-winged crickets from other populations, and the same is true for comparisons among short-winged morphs. The confirmation of this predicted complex pattern of geographic variation illustrates the importance of the quantitative genetic model in understanding evolutionary trajectories for polygenic threshold traits such as wing morphology.

Development time. In general, there is a positive correlation between development time and body size in insects (Roff 2000), and given that migratory morphs are often larger than nonmigrants, a positive correlation between migratory ca-

Figure 7. Schematic of the genetic relationships between migratory components in the sand cricket. Estimates of heritabilities (h^2) and genetic correlations (r_g) typically vary slightly among experiments, and so here we present estimates to only one decimal place. The arrows indicate the hypothesized direction of influence. For example, we assume that high juvenile hormone esterase (JHE) activity increases the liability for wing production (through its negative effect on juvenile hormone titer) and hence increases the proportion of macropterous crickets, while JHE activity and the liabilities for both wing production and wing muscle histolysis influence fecundity. Double arrows indicate that the direction of influence is unknown. The figure is modified from Roff and Fairbairn (2001).

Other than differences in wing morphology, there are no clear differences in external morphology between the wing morphs of *G. firmus*. In some other cricket species, such as *Allonemobius socius,* there is a very obvious difference in thorax shape and, in males, overall size (Roff and Bradford 1998). Like other morphological traits, size has a modest to high heritability of approximately 0.40 in *G. firmus* (Roff 2000).

Hormone titers. It has long been speculated that the major determinant of wing morphology in insects is the titer of JH at a particular stage of development (Southwood 1961). As noted above, JH titers during the critical period are regulated primarily by the activity of JHE, which therefore indirectly influences the development of the wing morphs. As expected, during the last nymphal instar, nymphs of presumptive short-winged *G. firmus* have lower JHE activity (and hence presumed higher JH titers) than nymphs of presumptive

pability and development time might be expected. Differences in development time between wing morphs are consistent with this prediction in most insect orders, but exceptions do occur, and in general the reverse is true in the Orthoptera (Roff 1995). As is typical for this order, in *G. firmus* the long-winged morph develops faster (i.e., has a shorter development time) than the short-winged morph, and the genetic correlation between liability and development time is negative (figure 7).

Although the wing morphs of *G. firmus* do not differ in size, faster growth of the potential migrant form has also been documented in some species in which the migrant form is larger (e.g., the milkweed bug [*Oncopeltus fasciatus*]; Hegmann and Dingle 1982). One explanation for the faster growth of potential migrants is that both growth rate and migratory potential are condition dependent, and only the nymphs experiencing favorable conditions, and hence fast growth, can support the energetic demands of migration. Under this scenario, fast growth and migratory potential could be strictly plastic responses to good environmental conditions. However, our research on *G. firmus* indicates that the negative correlation between the liability for wing production and development time is observed under controlled (constant) environmental conditions and is at least partially genetic (figure 7). Thus, individuals that are genetically predisposed to grow fast are also genetically predisposed to become long winged. The physiological mechanisms responsible for this correlation remain unresolved.

Distribution of energy stores. The major flight fuel for *G. firmus* is triglycerides, and although these have been shown to be higher in the long-winged morph (Zera et al. 1994, 1999, Zera and Larsen 2001, Zhao and Zera 2001, Zera and Zhao 2003), no estimates of heritabilities are available. We predicted that selection on the frequency of the wing morph would alter triglyceride levels, those of macropterous individuals being increased, which was confirmed by a comparison of control and lines selected for an increased or decreased proportion of macroptery (Stirling et al. 2001, Zhao and Zera 2002). Evidence from other species also supports this prediction. For example, selection on migration propensity in the wing-monomorphic lepidopteran *S. exempta* and the grasshopper *M. sanguinipes* produced an increase in thoracic lipids (Gunn and Gatehouse 1993, Kent and Rankin 2001). Other studies confirm the potential for evolution of the pathways involved in metabolism of flight fuels. For example, significant heritabilities (0.40) have been estimated for the metabolic enzymes of *D. melanogaster* (Clark 1990), and Haag and colleagues (2005) showed that the flight metabolic rate and the frequency of a specific allele of the metabolic enzyme phosphoglucose isomerase were highest in newly established, isolated populations of the Granville fritillary butterfly.

Flight propensity. Simply having the capability to fly does not necessarily lead to migratory flight. Given that the traits com-

prising the migratory syndrome are genetically correlated (figure 7), we predicted that the flight propensity of long-winged *G. firmus* would increase with an increase in the proportion of long-winged adults. As predicted, long-winged adults from our lines selected for a high proportion of the long-winged morph took flight more readily (i.e., had higher flight propensity) than long-winged adults from lines selected for a high proportion of the short-winged morph (Fairbairn and Roff 1990). Only individuals with fully developed wings and flight muscles were included in these comparisons, and thus the differences in flight propensity reflect differences in the behavioral tendency to fly. No heritability estimate is available from this study, but in the Texan population of *M. sanguinipes,* which is migratory, the heritability of flight propensity lies between 0.50 and 0.60 (Kent and Rankin 2001). Heritabilities of flight duration have been better studied and range from 0.15 to 0.88, with most estimates around 0.40 (six species; Kent and Rankin 2001, Roff and Fairbairn 2001).

Age-specific reproduction. In wing-dimorphic insects, the onset of reproductive activity is delayed, and reproductive success, as measured by fecundity or mate attraction, is reduced in the long-winged morph. In *G. firmus,* egg production in the first week following adult eclosion (our standard measure of fecundity) has a heritability of approximately 0.25, which is typical for fecundity in insects (see tables 4 and 5 in Roff 2000). There is no published estimate of the genetic correlation of fecundity between the wing morphs in *G. firmus,* but for *A. socius* it is –0.53 (Roff and Bradford 1996). Assuming this also holds for *G. firmus,* the quantitative genetic model predicts a decline in fecundity in the lines selected for a high proportion of the long-winged morph, and an increase in fecundity in the lines selected for high proportion of the short-winged morph. Our results confirmed the first prediction, but we found no significant increase in fecundity in the latter type of selection.

This asymmetry in response can be understood by considering the physiological basis of the negative correlation between wing morph and fecundity. The flight muscles are large, energy-demanding structures that compete with the reproductive functions, such as egg production and calling, for resources. The fecundity of long-winged females is reduced because of allocation of resources to these flight muscles (figure 6). However, long-winged females can recoup part of their investment in flight muscles by histolyzing them. This is a common phenomenon in insects and a clear example of the oogenesis-flight syndrome described above (Johnson 1969). The onset of histolysis indicates that the female has switched from a potential migratory phase to a reproductive phase.

Variation in the extent and timing of muscle histolysis is largely responsible for the observed variation in the size of the dorsolongitudinal muscles among long-winged female *G. firmus* (figure 6). The size of the dorsolongitudinal muscles at seven days posteclosion has a heritability of about 0.30, and

the genetic correlation between this estimate of muscle size and fecundity in the long-winged females is about –0.9. Selection on the frequency of the long-winged morph rapidly changes the size of the dorsolongitudinal muscles in the long-winged females, and this in turn produces a correlated change in fecundity during the first few weeks of adulthood. A similar change does not occur in the fecundity of short-winged females, because they never develop flight muscles (figure 6) and hence do not experience a change in the trade-off between flight musculature and ovary development as the proportion of the long-winged morph evolves.

Age-specific fecundity in insects is determined in part by changes in ecdysteroids, JH, and various neuroendocrine hormones (Nijhout 1994). Both ecdysteroids and JH have been shown to modulate ovarian activity in crickets (Strambi et al. 1997), and hence we would predict that the two wing morphs should differ in at least one of these components, if not both. Further, females that histolyze their flight muscles early, resulting in an age-specific fecundity pattern similar to the short-winged morph (figure 6), would be expected to have age-specific hormonal profiles similar to those of the short-winged females. Selection on the proportion of macropterous individuals in a population should change the hormonal profile in the same manner as predicted for the dorsolongitudinal muscles. All of these predictions have been confirmed in *G. firmus* (reviewed in Zera and Bottsford 2001).

The relationships between age-specific fecundity and migratory propensity observed for *G. firmus* depend on the specific physiological pathways and genetic correlations connecting wing morphology, fecundity, and flight muscle mass in this species. While we expect the genetic architecture of migratory polymorphisms to be broadly similar among closely related species such as those within a taxonomic family, other patterns of covariation may apply in more distant taxa. For example, in some monomorphically winged species, such as the grasshopper *M. sanguinipes,* migration is an integral and obligatory component of the life cycle, and flight induces and enhances reproduction (Rankin and Burchsted 1992, Min et al. 2004). Similarly, selection for flight duration in long-winged *O. fasciatus,* a true bug in the order Hemiptera, led to earlier onset of egg production and increased fecundity of the long-duration flying line (Dingle 1996). Thus, while the principles of multivariate evolution illustrated by our example are general, specific applications to other insect groups must await further empirical data.

Conclusions

Migration is a common feature of insect life cycles. Given the heterogeneity that is clearly evident in the natural world, the evolution of migration is both explicable and predictable. But evolution can occur only if there is genetic variation for the selected traits. Phenotypic variation in migratory propensity has long been known, but the genetic basis of such variation is still relatively unexplored. We have presented evidence for such variation, but there still exists a dearth of information on genetic architecture. Even more important, although it is recognized that migration is not a single trait but a suite of traits that include both larval and adult components, we need more data on the functional and genetic relationships among traits. Our own study of the sand cricket, *G. firmus,* and that by Dingle and his colleagues (summarized in Dingle 1996) of the milkweed bug, *O. fasciatus,* have shown that surprises await us but also that a coordinated approach to the problem can be successful.

References cited

Brakefield PM, French V, Zwaan BI. 2003. Development and the genetics of evolutionary change within insect species. Annual Review of Ecology, Evolution, and Systematics 34: 633–660.

Caldwell RL, Hegmann JP. 1969. Heritability of flight duration in the milkweed bug *Lygaeus kalmii.* Nature 223: 91–92.

Cheke RA, Tratalos JA. 2007. Migration, patchiness, and population processes illustrated by two migrant pests. BioScience 57: 145–153.

Clark AG. 1990. Genetic components of variation in energy storage in *Drosophila melanogaster.* Evolution 44: 637–650.

Colvin J, Gatehouse AG. 1993. Migration and genetic regulation of the prereproductive period in the cotton-bollworm moth *Helicoverpa armigera.* Heredity 70: 407–412.

Crnokrak P, Roff DA. 1995. Fitness differences associated with calling behaviour in the two wing morphs of male sand crickets, *Gryllus firmus.* Animal Behaviour 50: 1475–1481.

Denno RF, Roderick GK, Olmstead KL, Dobel HG. 1991. Density-related migration in planthoppers (Homoptera: Delphacidae): The role of habitat persistence. American Naturalist 138: 1513–1541.

Dingle H. 1989. The evolution and significance of migratory flight. Pages 99–114 in Goldsworthy GJ, Wheeler CH, eds. Insect Flight. Boca Raton (FL): CRC Press.

———. 1996. Migration: The Biology of Life on the Move. New York: Oxford University Press.

Dingle H, Drake VA. 2007. What is migration? BioScience 57: 113–121. doi:10.1641/B570206

Dingle H, Winchell R. 1997. Juvenile hormone as a mediator of plasticity in insect life histories. Archives of Insect Biochemistry and Physiology 35: 359–373.

Fairbairn DJ. 1992. The origins of allometry: Size and shape polymorphism in the common waterstrider, *Gerris remigis* Say (Heteroptera, Gerridae). Biological Journal of the Linnean Society 45: 167–186.

Fairbairn D, Desranleau L. 1987. Flight threshold, wing muscle histolysis, and alary polymorphism: Correlated traits for dispersal tendency in the Gerridae. Ecological Entomology 12: 13–24.

Fairbairn DJ, Roff DA. 1990. Genetic correlations among traits determining migratory tendency in the sand cricket, *Gryllus firmus.* Evolution 44: 1787–1795.

Fairbairn DJ, Yadlowski DE. 1997. Coevolution of traits determining migratory tendency: Correlated response of a critical enzyme, juvenile hormone esterase, to selection on wing morphology. Journal of Evolutionary Biology 10: 495–513.

Gatehouse AG. 1986. Migration in the African armyworm *Spodoptera exempta:* Genetic determination of migratory capacity and a new synthesis. Pages 128–144 in Danthanarayana W, ed. Insect Flight. Berlin: Springer.

———. 1989. Genes, environment and flight. Pages 115–138 in Goldsworthy GJ, Wheeler CH, eds. Insect Flight. Boca Raton (FL): CRC Press.

———. 1997. Behavior and ecological genetics of wind-borne migration by insects. Annual Review of Entomology 42: 475–502.

Gu H, Danthanarayana W. 1992. Quantitative genetic analysis of dispersal in *Epiphyas postvittana.* I. Genetic variation in flight capacity. Heredity 68: 53–60.

Gunn A, Gatehouse AG. 1993. The migration syndrome in the African armyworm moth, *Spodoptera exempta:* Allocation of resources to flight and reproduction. Physiological Entomology 18: 149–159.

Gunn A, Gatehouse AG, Woodrow KP. 1988. Trade-off between flight and reproduction in the African armyworm moth, *Spodoptera exempta.* Physiological Entomology 14: 419–427.

Haag CR, Saastamoinen M, Marden JH, Hanski I. 2005. A candidate locus for variation in dispersal rate in a butterfly metapopulation. Proceedings of the Royal Society of London B 272: 2449–2456.

Hegmann JP, Dingle H. 1982. Phenotypic and genetic covariance structure in milkweed bug life history traits. Pages 177–186 in Dingle H, Hegmann JP, eds. Evolution and Genetics of Life Histories. New York: Springer.

Jackson DJ. 1928. The inheritance of long and short wings in the weevil, *Sitona hispidula,* with a discussion of wing reduction among beetles. Transactions of the Royal Society of Edinburgh 55: 655–735.

Johnson CG. 1969. Migration and Dispersal of Insects by Flight. London: Methuen.

Kent JW, Rankin MA. 2001. Heritability and physiological correlates of migratory tendency in the grasshopper *Melanoplus sanguinipes.* Physiological Entomology 26: 371–380.

Min KJ, Jones N, Borst DW, Rankin MA. 2004. Increased juvenile hormone levels after long-duration flight in the grasshopper, *Melanoplus sanguinipes.* Journal of Insect Physiology 50: 531–537.

Mousseau TA, Roff DA. 1987. Natural selection and the heritability of fitness components. Heredity 59: 181–198.

Nijhout HF. 1994. Insect Hormones. Princeton (NJ): Princeton University Press.

Rankin MA. 1989. Hormonal control of flight. Pages 140–163 in Goldsworthy GJ, Wheeler CH, eds. Insect Flight. Boca Raton (FL): CRC Press.

Rankin MA, Burchsted JCA. 1992. The cost of migration in insects. Annual Review of Entomology 37: 533–559.

Roff DA. 1977. Disperal in dipterans: Its costs and consequences. Journal of Animal Ecology 46: 443–456.

———. 1984. The cost of being able to fly: A study of wing polymorphism in two species of crickets. Oecologia 63: 30–37.

———. 1986. The evolution of wing dimorphism in insects. Evolution 40: 1009–1020.

———. 1990a. The evolution of flightlessness in insects. Ecological Monographs 60: 389–421.

———. 1990b. Selection for changes in the incidence of wing dimorphism in *Gryllus firmus.* Heredity 65: 163–168.

———. 1994. Habitat persistence and the evolution of wing dimorphism in insects. American Naturalist 144: 772–798.

———. 1995. Antagonistic and reinforcing pleiotropy: A study of differences in development time in wing dimorphic insects. Journal of Evolutionary Biology 8: 405–419.

———. 2000. Trade-offs between growth and reproduction: An analysis of the quantitative genetic evidence. Journal of Evolutionary Biology 13: 434–445.

———. 2002. Life History Evolution. Sunderland (MA): Sinauer.

Roff DA, Bradford MJ. 1996. Quantitative genetics of the trade-off between fecundity and wing dimorphism in the cricket *Allonemobius socius.* Heredity 76: 178–185.

———. 1998. The evolution of shape in the wing dimorphic cricket, *Allonemobius socius.* Heredity 80: 446–455.

Roff DA, Fairbairn DJ. 1991. Wing dimorphisms and the evolution of migratory polymorphisms among the Insecta. American Zoologist 31: 243–251.

———. 1999. Predicting correlated responses in natural populations: Changes in JHE activity in the Bermuda population of the sand cricket, *Gryllus firmus.* Heredity 83: 440–450.

———. 2001. The genetic basis of dispersal and migration and its consequences for the evolution of correlated traits. Pages 191–202 in Clobert C, Nichols J, Danchin JD, Dhondt A, eds. Causes, Consequences and Mechanisms of Dispersal at the Individual, Population and Community Level. Oxford (United Kingdom): Oxford University Press.

Roff DA, Stirling G, Fairbairn DJ. 1997. The evolution of threshold traits: A quantitative genetic analysis of the physiological and life history correlates of wing dimorphism in the sand cricket. Evolution 51: 1910–1919.

Schumacher P, Weber DC, Hagger C, Dorn S. 1997. Heritability of flight distance for *Cydia pomonella.* Entomologia Experimentalis et Applicata 85: 169–175.

Southwood TRE. 1961. A hormonal theory of the mechanism of wing polymorphism in Heteroptera. Proceedings of the Royal Entomological Society of London 36: 63–66.

———. 1962. Migration of terrestrial arthropods in relation to habitat. Biological Reviews 37: 171–214.

Stein VW. 1977. Die Beziehung zwischen Biotop-Alter und Auftreten der Kurz-Flügeligkeit bei Populationen dimorpher Rüsselkäfer-Arten (Col., Curculionidae). Zeitschrift für Angewandte Entomologie 83: 37–39.

Stirling G, Fairbairn DJF, Jensen S, Roff DA. 2001. Does a negative genetic correlation between wing morph and early fecundity imply a functional constraint in *Gryllus firmus?* Evolutionary Ecology Research 3: 157–177.

Strambi A, Strambi C, Cayre M. 1997. Hormonal control of reproduction and reproductive behavior in crickets. Archives of Insect Biochemistry and Physiology 35: 393–404.

Wegener G. 1996. Flying insects: Model systems in exercise physiology. Experientia (Basel) 52: 404–412.

Zera AJ. 2004. The endocrine regulation of wing polymorphism in insects: State of the art, recent surprises, and future directions. Integrative and Comparative Biology 43: 607–616.

———. 2006. Evolutionary genetics of juvenile hormone and ecdysteroid regulation in *Gryllus:* A case study in the microevolution of endocrine regulation. Comparative Biochemistry and Physiology, pt. A 144: 365–379.

Zera AJ, Bottsford J. 2001. The endocrine-genetic basis of life-history variation: The relationship between the ecdysteroid titer and morph-specific reproduction in the wing-polymorphic cricket *Gryllus firmus.* Evolution 55: 538–549.

Zera AJ, Denno RF. 1997. Physiology and ecology of dispersal polymorphism in insects. Annual Review of Entomology 42: 207–230.

Zera AJ, Huang Y. 1999. Evolutionary endocrinology of juvenile hormone esterase: Functional relationship with wing polymorphism in the cricket, *Gryllus firmus.* Evolution 53: 837–847.

Zera AJ, Larsen A. 2001. The metabolic basis of life history variation: Genetic and phenotypic differences in lipid reserves among life history morphs of the wing-polymorphic cricket, *Gryllus firmus.* Journal of Insect Physiology 47: 1147–1160.

Zera AJ, Zhao Z. 2003. Morph-dependent fatty acid oxidation in a wing-polymorphic cricket: Implications for the trade-off between dispersal and reproduction. Journal of Insect Physiology 49: 933–943.

Zera AJ, Mole S, Rokke K. 1994. Lipid, carbohydrate and nitrogen content of long- and short-winged *Gryllus firmus:* Implications for the physiological cost of flight capability. Journal of Insect Physiology 40: 1037–1044.

Zera AJ, Sall J, Otto K. 1999. Biochemical aspects of flight and flightlessness in *Gryllus:* Flight fuels, enzyme activities and electrophoretic profiles of flight muscles from flight-capable and flightless morphs. Journal of Insect Physiology 45: 275–285.

Zhao Z, Zera AJ. 2001. Enzymological and radiotracer studies of lipid metabolism in the flight-capable and flightless morphs of the wing-polymorphic cricket, *Gryllus firmus.* Journal of Insect Physiology 47: 1337–1347.

———. 2002. Differential lipid biosynthesis underlies a tradeoff between reproduction and flight capability in a wing-polymorphic cricket. Proceedings of the National Academy of Sciences 99: 16829–16834.