Annu. Rev. Entomol. 1999. 44:535–60

RISK-SPREADING AND BET-HEDGING IN INSECT POPULATION BIOLOGY[1]

Keith R. Hopper
Beneficial Insect Introduction Research Unit, Agricultural Research Service, United States Department of Agriculture, Newark, Delaware 19713

KEY WORDS: facultative diapause, spatial distribution of oviposition, migration polyphenism, egg size, genetic polymorphism

ABSTRACT

In evolutionary ecology, risk-spreading (i.e. bet-hedging) is the idea that unpredictably variable environments favor genotypes with lower variance in fitness at the cost of lower arithmetic mean fitness. Variance in fitness can be reduced by physiology or behavior that spreads risk of encountering an unfavorable environment over time or space. Such risk-spreading can be achieved by a single phenotype that avoids risks (conservative risk-spreading) or by phenotypic variation expressed by a single genotype (diversified risk-spreading). Across these categories, three types of risk-spreading can be usefully distinguished: temporal, metapopulation, and within-generation. Theory suggests that temporal and metapopulation risk-spreading may work under a broad range of population sizes, but within-generation risk-spreading appears to work only when populations are small. Although genetic polymorphisms have sometimes been treated as risk-spreading, the underlying mechanisms are different, and they often require different conditions for their evolution and thus are better treated separately. I review the types of evidence that could be used to test for risk-spreading and discuss evidence for risk-spreading in facultative diapause, migration polyphenism, spatial distribution of oviposition, egg size, and other miscellaneous traits. Although risk-spreading theory is voluminous and well developed in some ways, rarely has it been used to generate detailed, testable hypotheses about the evolution of risk-spreading. Furthermore, although there is evidence for risk-spreading, particularly in facultative diapause, I have been unable to find any definitive tests

with unequivocal results showing that risk-spreading has been a major factor in the evolution of insect behaviors or life histories. To advance our understanding of risk-spreading in the wild, we need (*a*) explicit empirical models that predict levels of diversifying risk-spreading for several insect populations in several environments that vary in uncertainty, and (*b*) tests of these models using measurements of phenotypes and their fitnesses over several generations in each environment.

OVERVIEW

For all life, the future is uncertain. An insect may not diapause in time to avoid an early winter that freezes its host plant or even kills the insect directly. Another may lay its eggs on a host plant that is then killed by a plant pathogen. On the other hand, an insect that enters diapause earlier than needed may reach a smaller adult size or even miss a generation of reproduction, and an insect that disperses to lay its eggs on another host plant may be devoured by a predator before reaching its destination. In evolutionary ecology, risk-spreading (i.e. bet-hedging) is the idea that unpredictably variable environments favor genotypes with lower variance in fitness at the cost of lower mean fitness (12, 40, 41, 62, 78). Variance in fitness can be reduced by physiology or behavior that spreads risk in time or space. Such risk-spreading can be achieved by a single phenotype that avoids risks (conservative risk-spreading) or by phenotypic variation expressed by a single genotype (diversified risk-spreading) (17, 75, 90).

Much of life-history theory concerns trade-offs between survival and reproduction, which in a stochastic world often involves risk-spreading (93). For example, if iteroparity arose from adults being more resistant than juveniles to environmental fluctuations, this might be a form of temporal risk-spreading (10, 45, 93). Selection for risk-spreading might explain, for example, variation in diapause frequency in species with facultative diapause (5, 30, 38, 46, 53, 108) or migration polyphenism in insects colonizing ephemeral habitats (25, 26, 72, 82, 107, 114). Since the 1960s, a large body of theory has been developed (12–15, 17, 24, 40–43, 45, 55, 56, 58, 61–65, 69, 74, 78, 83, 90, 105, 106) and a limited amount of evidence accumulated (5, 9, 21, 25, 26, 30, 38, 46, 53, 66, 72, 82, 97, 107–110, 114) concerning risk-spreading in insects. Unfortunately, theory has far outrun evidence, and empirical studies have sometimes invoked theory superficially. Nonetheless, risk-spreading appears to have the potential to explain some behavior and life history of insects and other organisms (75, 90).

Risk-spreading has implications for insect pest management and conservation biology. Spatial risk-spreading by parasitoids might explain low levels of parasitism and thus lack of impact of biocontrol agents (66); spatial risk-spreading

by herbivores might explain patterns of attack on crops. Although theory suggests that risk-spreading in spatial distribution of oviposition is rather unlikely in many insect species (19), variation in diapause and migration polyphenism are two areas where risk-spreading appears likely. Both diapause and migration affect management of insect pests (29, 99); thus, understanding how risk-spreading affects them is important for sustainable solutions to pest problems. Risk-spreading in metapopulations has implications for the evolution of migration (69, 74). Given current increases in habitat fragmentation, understanding how risk-spreading is likely to influence evolution of dispersal among populations within metapopulations may have implications for prevention of species extinctions (23, 25, 59).

Risk-spreading is a frequent human response to an uncertain future. A farmer will plant several crops with different needs and markets to spread the risk of crop or market failure. Such diversity of crops helps ensure a more stable income. In part because the idea is easily grasped and intuitively appealing, risk-spreading has been readily accepted by entomologists and ecologists. However, analogies from human behavior to evolution of insect populations are fraught with potential for misinterpretation. Indeed, risk-spreading has sometimes been misunderstood and misapplied, and although many areas of confusion have been discussed (90), misunderstandings and misapplications continue. Furthermore, the evidence that has been used to test for risk-spreading has been varied, but often indirect, leading to problems with interpretation. For these reasons, a critical review of the theory of risk-spreading and its applications is needed to clarify current understanding and perhaps promote better use of the concept in research on insects. Here, I first discuss the theory and then review examples of the evidence for risk-spreading among insects.

THEORY

Geometric Mean Fitness

Evolution occurs over time; what counts is the increase or decrease in gene frequencies over generations of reproduction. Because change in gene frequency over time is multiplicative, long-term fitness in a temporally fluctuating environment is measured by the geometric mean of fitness across generations, rather than the arithmetic mean of the fitness distribution (22, 40–43, 65, 69). Furthermore, if two genotypes have underlying frequency distributions of fitness with the same arithmetic mean but different variances, the one with the lower variance will have the higher geometric mean fitness averaged over generations (40–43, 65, 69). It is this increase in geometric mean with decrease in variance that promotes risk-spreading. For risk-spreading to be favored by natural selection, it must yield a higher geometric mean fitness, and it does

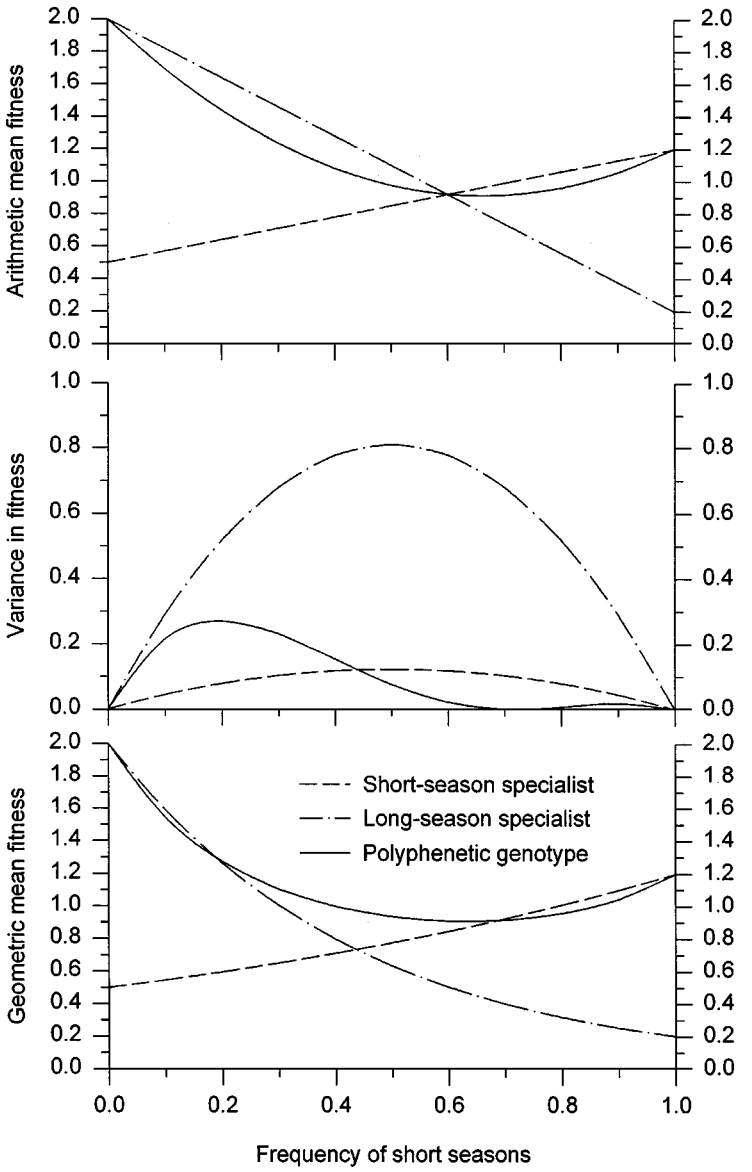
this by lowering temporal (i.e. between-generation) variation in fitness at the expense of arithmetic mean fitness. An example will help clarify this point. Suppose an insect species lives in an environment that fluctuates between either a short, cool growth season or a long, hot growth season. Let us assume there are three genotypes: one that does well in short seasons, another that does well in long seasons, and a third that produces both phenotypes in proportions that match the frequencies of each type of season (Figure 1). Here, the arithmetic mean fitness for the polyphenetic genotype is less than that of the short-season specialist when short seasons are frequent ($f_1 > 0.6$) and less than that of the long-season specialist when long seasons are frequent ($f_1 < 0.6$). However, because the variances in fitness of short- and long-season specialists are often greater than that of the polyphenetic genotype, the latter has higher geometric mean fitness than either specialist when neither short nor long seasons predominate ($0.2 < f_1 < 0.7$), and even at the extremes of season frequencies where the respective specialist has a higher geometric mean fitness, the difference is smaller than that for arithmetic mean fitness.

Although a substantial body of theory and experiment has developed concerning risk-sensitive foraging (76, 77), I do not deal with this subject. Although the foraging strategy of an individual can increase its fitness, it does not necessarily affect variance in fitness within genotype or the trade-off between arithmetic mean and variance in fitness (90).

Environmental Uncertainty and Conservative Versus Diversifying Risk-Spreading

Risk-spreading arises from environmental uncertainty and thus uncertainty as to which phenotype will have highest fitness in the future. Diversified risk-spreading is one response to such uncertainty (17, 90). A single genotype produces multiple phenotypes to hedge against the chance of environmental change. An example is the kind of facultative diapause in which individuals of the same genotype may or may not diapause under identical cues (e.g. photoperiod at a particular stage of development). Such nongenetic phenotypic variation

Figure 1 Arithmetic mean fitness, variance in fitness, and geometric mean fitness versus frequency of short seasons for genotypes that do well in short seasons or long seasons compared with a genotype that produces phenotypes adapted to each season type in proportion to the frequencies of season types. Geometric mean fitness $= (w_1 w_2 \ldots w_j \ldots w_t)^{1/t} = w_1^{f_1} w_2^{f_2} \ldots w_i^{f_i} \ldots w_n^{f_n}$, where $w_j =$ fitness in j^{th} season, $t =$ number of seasons, $w_i =$ fitness in i^{th} type of season, $f_i =$ relative frequency of i^{th} type of seasons, and $n =$ number of season types. Letting 1 represent short season and 2 long season, $w_1 = 1.2$ and $w_2 = 0.5$ for the short-season specialist, and $w_1 = 0.2$ and $w_2 = 2$ for the long-season specialist. The fitness of the polyphenetic genotype was the average of these values weighted by the frequency of each season type.



in response could arise from small differences in conditions under which individuals develop, which might be exaggerated by developmental instability (56, 91).

Although risk-spreading could in theory arise in a predictably fluctuating environment, predictable oscillations are much more likely to favor a single best behavior or physiology that tracks the environment. For some species in some systems, obligate diapause and obligate migration are examples. However, obligate diapause, obligate migration, and many other aspects of insect development and behavior may also be examples of conservative risk-spreading in an uncertain environment, where genotypes that reduce variance in fitness by avoiding risk altogether are favored (90). In a sense, conservative bet-hedging is not bet-hedging at all because the strategy is to always bet that the environment will be adverse; this behavior might be better named risk-aversion. On the other hand, conservative bet-hedging does meet the criterion of reducing variance in fitness by reducing arithmetic mean fitness. An example is the short-season specialist discussed above. This specialist does not do as well in its best season as the long-season specialist does in its, but the short-season specialist also does not do as poorly in its worst season. The arithmetic mean of the short-season specialist is lower than that of the long-season specialist for short-season frequencies less than 0.6 (Figure 1). But the variance in fitness of the short-season specialist is lower than that of the long-season specialist. Thus, the geometric mean fitness of the short-season specialist is greater than that of the long-season specialist even when long seasons are somewhat more frequent. In any case, such a conservative strategy will often have lower fitness than a diversifying bet-hedger that produces appropriate phenotypes for each environment likely to be encountered in rough proportion to the likelihood of each environment (17, 90). An example is the short-season/long-season genotype discussed above; this polyphenetic genotype has higher geometric mean fitness than the short-season specialist over most frequencies of short seasons (Figure 1). Furthermore, as is discussed in more detail below difficulties arise in testing whether a genotype has increased or persisted because of selection for conservative risk-spreading.

Although variation in the abiotic environment (e.g. annual rainfall, time of onset of winter) has received most attention in the literature, variation in the biotic environment (predation, competition) could be equally important. In particular, density-dependent and frequency-dependent intraspecific competition have been shown in theory to amplify variation in fitness among environments and thus the advantage of risk-spreading genotypes.

Genetic Polymorphism versus Risk-Spreading

Although genetic polymorphisms have sometimes been treated as risk-spreading (e.g. 48, 53), the underlying mechanisms are different, and they often require

different conditions for their evolution and thus are better treated separately (75, 90). Both genetic polymorphisms and risk-spreading are favored by spatial and temporal heterogeneity (11, 44, 90), but the conditions for maintenance of genetic polymorphisms are less stringent than those for risk-spreading. However, if the cost of phenotypic variation within a genotype is not too high, a single genotype with variable phenotype can invade and replace a genetic polymorphism that includes the same phenotypes expressed by separate genotypes (17, 90). For example, suppose an insect has a dark morph that does well in cool summers and a pale morph that does well in warm summers. If these morphs arise from different genotypes, each morph would be at a selective disadvantage in the wrong sort of summer. A phenotypically variable genotype that produced both morphs in approximately the frequencies of cool and warm summers would have higher fitness and thus outcompete both specialist genotypes. Thus, one has the superficial paradox that a genotype should increase its phenotypic variation to decrease temporal variation in fitness (17).

Dimensions Across Which Risk Can Be Spread

Three types of risk-spreading can be usefully distinguished: temporal, metapopulation, and within-generation (Table 1). In all types, the key mechanism favoring their evolution is that genotypes with low variation in fitness increase at the expense of genotypes with high variation in fitness. The types differ primarily in what Levins (62) called environmental grain, and this difference affects the quantitative relationship between decrease in variance and increase in geometric mean fitness. Because of differences in this relationship, temporal and metapopulation risk-spreading are much more likely to evolve in insect species than within-generation risk-spreading. Below, I discuss each type and the conditions for its evolution in more detail.

Temporal Risk-Spreading

In temporal risk-spreading, the whole population is exposed to the same fluctuations in the environment. In a temporally fluctuating environment, bad conditions may select for a resistant stage (12, 16). For example, when faced with harsh conditions that can kill directly or destroy their food supply, many insects respond by diapausing in a stage that does not require further nutrition and is more or less resistant to extremes of temperature and humidity (99). Genotypes that diapause too soon forgo favorable conditions for growth and reproduction; those that diapause too late risk dying from adverse conditions. The trade-offs between growing to a larger size versus producing another generation (81) and the timing of diapause and optimal use of photoperiod and other cues have been explored (16, 100–102, 104). In environments where the onset of adverse conditions can be predicted from environmental cues, natural selection

Table 1 Potential responses to environmental variation

Type of environmental variation	Potential responses	Risk-spreading strategy	Examples
Temporal	Temporal risk-spreading	Conservative	Early obligate diapause, Delayed post-diapause emergence
		Diversifying	Variable facultative diapause, Variable post-diapause emergence, Color polyphenism
	Cued developmental, behavioral plasticity	—	Cued facultative diapause, Cued color polyphenism
	Genetic variation	—	Polymorphic diapause, Color polymorphisms
Coarse-grained spatio-temporal	Metapopulation risk-spreading	Conservative	Obligate migration
		Diversifying	Flight polyphenism
	Cued developmental, behavioral plasticity	—	Cued flight polyphenism
	Genetic variation	—	Flight polymorphism
Fine-grained spatio-temporal, genetic, etc	Within-generation risk-spreading	Diversifying	Dispersed oviposition, Multiple mating
	Cued developmental, behavioral plasticity	—	Site-dependent oviposition
	Genetic variation	—	Polymorphic host specificity, Assortative mating

will favor responsiveness to these cues, i.e. an environment-conditional phenotype. When species do not have reliable cues for onset of adverse conditions, natural selection will favor either genotypes with an obligate diapause commencing well before conditions ever become harsh, or genotypes that produce both diapausing and nondiapausing phenotypes with or without modification of diapause frequency by environmental cues (17, 62, 90, 105). The frequency of diapausing forms should increase as the probability of adverse conditions increases during the season (62, 90). If producing diverse phenotypes does not have other fitness costs, diversifying risk-spreading will usually outcompete conservative risk-spreading (17, 90). Thus, there are several possible diapause strategies that may evolve: obligate diapause after one or more generations of reproduction, phenotypically plastic diapause in response to environmental cues, or phenotypically diverse diapause with or without environmental cues (Table 1). Which of these is favored depends on the amount of variation in the timing of adverse conditions, the reliability of environmental cues, and the cost of phenotypic plasticity and phenotypic diversification.

Temporal risk-spreading need not involve such extreme consequences as mortality from overly delayed diapause. Variation in color morph may hedge against temporal variation in temperature (50). Variation in postdiapause development time may hedge against variation in early season climate or food availability (18). Variation in egg size may affect development time, which in turn affects synchronization with bud-burst (89). Furthermore, the time scale for risk-spreading can range from less than a generation to multiple generations (96). The distinguishing characteristic of temporal risk-spreading is that all individuals in a population are exposed to environmental change, and thus selection acts on all individuals at once. Because of this, temporal risk-spreading can directly reduce between-generation variance in genotypic fitness. Using a model of haploid inheritance, Gillespie (40) showed that, in a temporally fluctuating environment, geometric mean fitness (as measured by offspring number) is approximately $\mu - \sigma^2/2$, where μ is the arithmetic mean of fitness and σ^2 is the temporal variance in fitness. Thus, reduction in variance can have half the effect on geometric mean fitness as increasing arithmetic mean of the distribution of fitnesses.

Metapopulation Risk-Spreading

In temporal risk-spreading, individuals of the risk-spreading genotype reduce temporal variation in fitness by producing one or more phenotypes that will be suited to the future environment. However, if there were other sites simultaneously available with environmental conditions likely to be more favorable than those in the current site, migration to another site would be an alternative to remaining to suffer adverse conditions in the current site (13, 29, 60, 83, 92, 94). I call this metapopulation risk-spreading because it requires a metapopulation structure, that is, a set of sites each capable at least sometimes of supporting a population with dispersal among sites (1, 64). I use the term migration in the genetic sense, that is, without any implication of direction or return, but with the idea of movement from one population to another once in a lifetime. The key feature to evolution of metapopulation risk-spreading is that the sites have different environments at the same time and that all of the individuals in a population are exposed to the environment in a site. Because a migratory genotype occurs in many independently fluctuating populations, the temporal variance in its frequency is reduced and thus its geometric mean fitness increased as compared with a genotype that does not disperse (58, 69, 94).

Although metapopulation risk-spreading resembles temporal risk-spreading in being a response to temporal uncertainty, it differs in that the only trait concerned is migration (although migration itself may be a complex trait). As with temporal risk-spreading, one can identify a conservative strategy where all individuals migrate at some fixed signal (e.g. after one generation of reproduction

or at a certain photoperiod). However, migration between predictably suitable sites (e.g. for summer reproduction and winter diapause) is not risk-spreading because there is no trade-off between mean and variance in fitness: nonmigrants would have lower fitness by any measure. For metapopulations, migration polyphenism is the diversifying risk-spreading strategy. Here, one genotype produces one phenotype for residence (e.g. apterous aphids, brachypterous planthoppers and grasshoppers) and another for migration (e.g. alate aphids, macropterous planthoppers and grasshoppers).

Zera & Denno (114) review the ample evidence for trade-offs between reproduction and dispersal in insects; the gist of this is that nonmigrant forms have higher reproductive rates than migrant forms. Mortality may also be greater during migration, and there is obviously some risk of not finding a suitable habitat, although these risks are hard to estimate and there are few data available. On the other hand, nonmigrants incapable of dispersing long distances run the risk that the environment will become less favorable in the future. Although temporal risk-spreading through diapause appears less risky than metapopulation risk-spreading through migration (92), diapause may not always be possible, and in ephemeral habitats such as temporary pools may actually be more risky. Indeed, metapopulation risk-spreading would be strongly favored where the environment in the current site risked becoming so inhospitable that no phenotype would survive. However, if cues to such deterioration were sufficient that they could be used to decide when to migrate, this could no longer be considered risk-spreading because nonmigrants would have lower fitness by any measure.

An extensive theory concerning metapopulation dynamics has been developed (for review see 47) and would seem relevant to metapopulation risk-spreading (e.g. 11, 44, 60, 73, 80, 83). The theory has rarely been couched in terms of risk-spreading, however, and often addresses other issues (e.g. maintenance of genetic polymorphisms, persistence of predator–prey interactions), so that teasing out predictions relevant to risk-spreading can be difficult. The term risk-spreading as it concerns metapopulations was initially used to refer to behaviors promoting persistence of species in the face of stochastic environmental variation (24, 79). This concept continues to be invoked (25) despite the need for selection at the species level for its evolution and the rareness of the conditions where such selection is likely to overcome individual selection (90). However, if one replaces the terms species and population with genotype in the early literature, these papers then describe metapopulation risk-spreading. Because of the costs of dispersal, migration will be selected against within populations but will often be favored at the level of the metapopulation (25). Although some have argued that migration thus arises from group selection (106), it is really just a question of temporal and spatial scales at which one measures fitness. Selection is against migration at some times and for migration at other times in the

same place. Thus, the frequency of migrants should depend on how frequently sites become unfavorable (82). Indeed, in a model with genetically determined flight propensity, Roff (83) found that the proportion of migrant types declined with persistence time of populations. Although persistence rather than geometric mean rate of growth was used as the measure of fitness in some early work (e.g. 24, 79), the two measures are actually compatible (65, 109), at least for large populations (70).

Almost all of metapopulation theory concerning the evolution of migration treats differences in migration as genetically fixed. If variation in migration is genetic, then it is a polymorphism, not risk-spreading. However, a genetic polymorphism for migration propensity could be invaded by a diversifying risk-spreading genotype that produced migrant and nonmigrant phenotypes in the ratio expected given population persistence times (17, 90). If all individuals migrate, this could be conservative risk-spreading, but theory suggests that conservative risk-spreading often has lower geometric mean fitness than diversified risk-spreading (17, 90). In any case, if cues for environmental deterioration were sufficiently reliable, a genotype that produced migrant and nonmigrant phenotypes in response to the environment should outcompete conservative and diversified risk-spreading genotypes, as well as genetic polymorphisms (Table 1).

Advantages of metapopulation risk-spreading depend greatly on details of the system (e.g. number of populations, temporal variation in habitat quality, spatial correlation in habitat quality, cost of migration) (69, 74, 83). One can readily see that if the number of populations were very large and population extinctions rare, nonmigrants would be depleted very slowly. On the other hand, with only a few populations, migration would become very important (69). In any case, the conditions for metapopulation risk-spreading are much less stringent than within-generation risk-spreading.

Within-Generation Risk-Spreading

An example of within-generation risk-spreading would be female insects that do not lay all eggs on one plant but distribute them among many plants to avoid chance catastrophes that might destroy individual plants independently. Because flight between plants costs time and energy, genotypes that spread eggs among plants would have lower arithmetic mean fitness than ones that did not, but the risk-spreaders would also have lower variance in fitness. Indeed, such spatial risk-spreading is the most commonly invoked case of within-generation risk-spreading. Although the rationale for such behavior is readily grasped, Gillespie (41–43) was perhaps the first to formally propose that within-generation variation in fitness could affect temporal geometric mean fitness. Using a model of haploid inheritance with constant population size,

he showed that when offspring number varies within a generation, geometric mean fitness between generations is approximately $\mu - \sigma^2/N$, where μ is the arithmetic mean of fitness, σ^2 is within-generation variance in fitness and N is population size.

In temporal risk-spreading, all individuals of a genotype in a population experience the same environment. Thus, temporal risk-spreading directly reduces between-generation variation in fitness and increases geometric mean fitness. In within-generation risk-spreading, however, different individuals of a genotype in a population experience different environments. Thus, this form of risk-spreading only indirectly reduces between-generation variance in genotypic fitness through extinction of lineages within a population. For this reason, the impact of within-generation risk-spreading varies inversely with population size, and given magnitudes of fitness differences, within-generation risk-spreading is likely only for small populations (e.g. $N < 100$) (19).

Females may distribute progeny among patches to avoid density-dependent effects from competitors, predators, or other factors (2, 66). However, this is not risk-spreading in the sense discussed here because there is no trade-off between mean and variance in fitness for this sort of behavior.

In metapopulation risk-spreading, as in within-generation risk-spreading, different individuals of a genotype in the metapopulation also experience different environments. But there is a crucial difference: In metapopulation risk-spreading, all individuals of a genotype in a population experience the same environment, so what counts is the number of populations, not population size. In other words, environmental grain is much coarser for metapopulation risk-spreading than for within-generation risk-spreading. Although these two forms of risk-spreading may intergrade depending on spatial and temporal scale of dispersal and environmental fluctuations, separating them avoids confusion about the conditions favoring risk-spreading.

EVIDENCE

I found 62 empirical studies on risk-spreading in plants, invertebrates, and vertebrates with searches of computer databases (with the keywords risk and spreading and bet-hedging), literature cited in reviews and other papers, and a scan of the 1996–1997 volumes of *American Naturalist*, *Ecological Entomology*, *Entomologia Experimentalis et Applicata*, *Oecologia*, *Oikos*, and *Journal of Animal Ecology*. Here, I examine examples of the research on insects and spiders, first by discussing the types of evidence that could be used to test for risk-spreading (Table 2), and then discussing evidence for risk-spreading in facultative diapause, migration polyphenism, spatial distribution of oviposition, egg size, and other traits.

Table 2 Types of evidence for risk-spreading

Category	Example (strategy)	Types of evidence[a]
Temporal risk-spreading	Obligate diapause (conservative)	Geometric mean fitness of genotype with early diapause greater than that with late diapause, Elimination of alternative hypotheses for apparently maladaptive early diapause
	Facultative diapause (diversifying)	Geometric mean fitness of genotype with high variability in diapause timing greater than that with low variability, Gradient in within-genotype variation in diapause timing across gradient in uncertainty of season length, Gradient in variation in diapause timing across gradient in uncertainty of season length, Elimination of alternative hypotheses for higher than expected variation in diapause timing, Greater than expected variation in diapause timing, Variation in diapause timing
Metapopulation risk-spreading	Obligate migration (conservative)	Geometric mean fitness of genotype that migrates greater than that which does not, Elimination of alternative hypotheses for apparently maladaptive migration
	Flight polyphenism (diversifying)	Geometric mean fitness of genotype with high variability in migration greater than that with low variability, Gradient in within-genotype variation in migration across gradient in uncertainty of season length, Gradient in variation in migration across gradient in uncertainty of season length, Elimination of alternative hypotheses for higher than expected variation in migration, Greater than expected variation in migration, Variation in migration
Within-generation risk-spreading	Oviposition dispersion (diversifying)	Geometric mean fitness of genotype with dispersed oviposition greater than that with concentrated oviposition, Gradient in within-genotype dispersion of oviposition across gradient in risk of complete loss of clutches, Gradient in dispersion of oviposition across gradient in risk of complete loss of clutches, Elimination of alternative hypotheses for higher than expected dispersion of oviposition, Greater than expected dispersion of oviposition, Dispersed oviposition

[a]Ranked in order of strength of support (1 strongest) for risk-spreading versus other hypotheses.

Types of Evidence

The theoretical work on risk-spreading has dealt with general principles and thus has often not provided detailed, testable predictions. However, risk-spreading theory does make the broad prediction that phenotypic variation from diversifying risk-spreading should increase with environmental uncertainty (i.e. both variability and unpredictability). One could test this prediction by comparing phenotypic variation among populations or species across a gradient in environmental uncertainty. The gradient could be temporal or spatial, but if it were temporal, one would have to assume sufficient genetic variance in phenotypic variability that evolution could occur; thus spatial gradients are more tractable. Because population comparisons control for phylogenetic constraints, they should usually yield stronger tests than species comparisons, although problems with phylogenetic constraints can be minimized with the proper analysis (26). Comparison of trends in related species across a gradient in environmental uncertainty could provide evidence for the generality of the risk-spreading strategy. Wise choice of both trait and environmental parameters is needed because failure to find the pattern expected from risk-spreading may raise objections to one or both. A variety of methods have been used to test natural selection in the field (37, 67), several of which are applicable to testing for risk-spreading. However, diversifying risk-spreading has the additional complication that variation in phenotype results from a single genotype.

Specifically for diapause, theory suggests that in environments with unpredictable lengths of favorable seasons, the proportion of individuals diapausing should increase during the favorable season as the likelihood of completing another generation declines (62, 90). Thus, a gradual increase in proportion diapausing as the season advances suggests risk-spreading, as long as the variation in diapause frequency is not genetic (90).

For metapopulation risk-spreading, there must be a trade-off between the costs and benefits of migration, and for diversifying risk-spreading, a single genotype must express migration and nonmigration phenotypes under the same environmental conditions (17, 90).

Risk-spreading has been invoked as an explanation when phenotypic variation was found to be greater than expected. However, to actually determine expected phenotypic variation with and without risk-spreading requires measurement of the frequencies of various environments and measurement of phenotypic fitnesses in these environments. This is difficult to do and in fact has not been done. Instead, the criterion used to indicate risk-spreading has been that the observed phenotypic variation appears maladaptative unless one assumes risk-spreading. Because a fitness cost is a condition for risk-spreading, one could test for this condition by measuring fitnesses of risk-spreading and non–risk-spreading genotypes.

Some researchers have tested for risk-spreading by eliminating alternative hypotheses for apparently maladaptive behavior, but as is apparent below, this approach suffers from the weakness that one may fail to consider all alternative hypotheses.

Regardless of how the pattern in phenotypic variation is analyzed, one must also show that phenotypic variation in putative diversifying risk-spreading is not genetically based, for otherwise, it is a genetic polymorphism and not risk-spreading (17, 90).

Testing for conservative risk-spreading suffers from the difficulty of choosing a non–risk-spreading strategy for comparison. For example, an insect species may have evolved an obligatory diapause in response to the fluctuating length of the favorable season. But if so, how would one measure the reduction in mean fitness that is sacrificed to lower variance in fitness? One could imagine a hypothetical species with another generation before diapause and compare its arithmetic and geometric mean fitnesses, but why not make the comparison with a hypothetical species with a fast enough development time to fit another generation in the season, or with a longer development time and larger body size to take more advantage of the favorable season? Perhaps because of difficulties with testability, no one appears to have used conservative risk-spreading as an explanation in real systems. Thus, I will not consider conservative risk-spreading in examining the evidence for risk-spreading below.

Facultative Diapause

Because the fitness costs of diapausing or not diapausing can be large and the trait itself is readily measured, much research has been done on the mechanisms and evolution of diapause (99). Indeed, most of the research on risk-spreading in insects deals with timing of diapause, and I had expected to find the strongest case for risk-spreading in phenotypic variation in diapause timing.

Individual females of *Gryllus firmus* (Orthoptera: Gryllidae) lay a mixture of diapausing (slow development) and nondiapausing (rapid development) eggs in northern Florida, which suggests that these females are risk-spreading (108). However, several complications make the presence of risk-spreading hard to affirm. First, the proportion of diapausing eggs does not increase during the season, as expected under risk-spreading. Second, although rapid-development eggs laid in the fall do not diapause when reared under laboratory conditions, many of them do diapause in the field, indicating that eggs respond to environmental cues independently from those experienced by their mothers. Third, eggs laid in the fall that do not diapause are not doomed to die because they can overwinter as nymphs; all stages appear to survive well in winter temperatures in northern Florida. Indeed, voltinism is rather complicated in northern Florida: univoltine, bivoltine, and even trivoltine development is possible.

Fourth, spring droughts may favor overwintering as nymphs, and fall droughts overwintering as eggs, suggesting a complex trade-off between winter temperatures and drought. Fifth, the potential for migration of univoltine genotypes from northern populations further complicates the issue. Sixth, the fitness costs of diapausing versus producing another generation were not measured. Lastly, some of the variation in diapause among eggs may be genetic because other *Gryllus* spp. show heritable variation in diapause among eggs (3).

When reared in the laboratory in uniform conditions, larvae of *Wyeomyia smithii* (Diptera: Culicidae) from single females collected in the wild vary in development time and tendency to diapause (53). Because adults cannot survive the winter, and diapause occurs in the third instar and thus before length of season can be known with certainty, Istock (53) concluded that this was a risk-spreading strategy in response to variable length of the favorable season. However, fitness trade-offs for diapausing versus producing another generation were not measured. Furthermore, Istock (53) and others (8) found that variation in *W. smithii* diapause is genetic, which makes it difficult to interpret as risk-spreading. Indeed, Istock (54) describes diapause in *W. smithii* as a genetic polymorphism maintained by balancing selection. Also, in the field, diapause at a particular photoperiod is strongly modified by food supply, suggesting a strategy based in part on environmental cues. Lastly, interannual variability in season length may be too low to affect *W. smithii* phenology strongly (57).

In Iowa, adult females of *Oncopeltus fasciatus* (Hemiptera: Lygaeidae) undergo a reproductive diapause and migrate under short day lengths, and in Puerto Rico, no females diapause and few migrate at any time (30, 31). On the other hand, in Florida, time to first reproduction (a measure of reproductive diapause) varies greatly at the same photoperiod and temperature (30, 31). Dingle (28) argues that the gradient in diapause results from the differences in environmental favorability and predictability: *O. fasciatus* cannot survive the winter in Iowa but can survive and reproduce year round in Puerto Rico. In Florida, conditions of temperature and host plant availability vary greatly and unpredictably from year to year, so that different diapause durations are favored at different times. Thus, variation in reproductive diapause of *O. fasciatus* in Florida might be risk-spreading. Although Dingle (28) measured the fitness cost of diapausing under summer conditions, he did not measure the long-term costs of not diapausing. Furthermore, Dingle et al (32) found a heritability of 0.71 for time to first reproduction in Iowa *O. fasciatus*. If heritability were similar for Florida *O. fasciatus*, this would leave little room for risk-spreading. Indeed, Dingle (28) suggests that the variation in Florida diapause is genetic and is maintained by migration and fluctuating selection.

In southeastern Australia, seasons are not long enough for two complete generations a year of *Caledia captiva* (Orthoptera: Acrididae); yet females lay egg pods containing mixtures of diapausing and nondiapausing eggs (46). Nymphs

from nondiapausing eggs and adults can overwinter successfully and begin reproduction again in the spring. Adults from diapausing eggs reproduce later in the summer and fall. Groeters (46) suggested that this variation in diapause might be risk-spreading by females. However, the fitness costs of diapausing or developing directly were not measured. Furthermore, the potential for a genetic basis to within-pod variation in diapause was not examined.

In central Brazil, females of *Deois flavopicta* (Homoptera: Cercopidae) lay diapausing and nondiapausing eggs; the proportion diapausing increases as the season progresses, and postdiapause development time of eggs is highly variable (38). Fontes et al (38) argue that variation in diapause and in postdiapause development are risk-spreading responses to uncertainty in the onset of the rainy season. However, they did not measure the fitness costs of diapausing versus not diapausing or short versus long postdiapause development. Furthermore, they did not test whether the observed variation was genetic.

In Virginia, females of *Allonemobius socius* (= *fasciatus*) (Orthoptera: Gryllidae) lay mixtures of diapausing and nondiapausing eggs, and the proportion of diapausing eggs increases gradually during the season (5). Such a graded response suggests risk-spreading; but a series of experiments, observations, and models that provide the most thorough empirical evidence concerning risk-spreading of any sort for insects, showed there was little risk-spreading in this system (71, 4–7). The gradual increase in diapause frequency results from variation among females; within females, the switch from laying nondiapausing eggs to laying diapausing eggs is very rapid (5). Variation among females in median date for egg diapause has a strong genetic component: in the early-season environment, heritability was 0.69, and in the late-season environment, it was 0.39 (4). Using actual weather data from the collection area, Bradford & Roff (7) modeled the effect of interannual variation in season length on fitness. They found that the optimal diapause strategy included mixtures of nondiapausing and diapausing eggs only over a two-week period, and the central 60% of the switch included only five days. These results matched well the pattern observed in the field (5, 7). Apparently, interannual variation in season length was too low to favor much risk-spreading; the high level of genetic variation in timing of diapause may result from migration (7). Genetic differences and environmental cues gave the appropriate phenotype for the season and geographical zone (6).

Given the above results, it appears that genetic variation (8, 27, 33, 51, 52, 71) and conditional responses to environmental cues (98, 99, 103) explain most phenotypic variation in diapause, without invoking risk-spreading. It may be that year-to-year variation in favorable season length is often too small to select for risk-spreading (57, 105).

Periodical insects such as *Magicicada septemdecim* (Homoptera: Cicadidae) show the reverse of temporal risk-spreading, i.e. synchronization rather than

spreading out of adult emergence (48, 68). Paradoxically, periodical life cycles may have arisen from greater than annual variation in larval development time in univoltine insects, and this variation may have been favored because it spread risk of reproductive failure in temporally uncertain environments (48, 68). Although such variability in development time shows promise as a candidate for risk-spreading, it has been neglected (75), and rigorous studies have not been done on fitness costs and incidence of multigeneration variation in development time across gradients of environmental uncertainty.

Migration Polyphenisms

Although there have been few field studies on metapopulation dynamics compared with the amount of theoretical work (47), metapopulation structure appears to be common for insect populations (e.g. 36, 49, 95). Furthermore, there are well-documented trade-offs between migration and reproduction. Migrant phenotypes often have lower fecundity than nonmigrants from producing wings and flight muscles and from the cost of actually flying (26, 35, 83–85, 113, 114), and migrants may also suffer higher mortality while in transit, although this is not well documented. Thus, two of the conditions (metapopulation structure and a cost for migration) mentioned above as necessary for metapopulation risk-spreading are likely to be met for many insect species.

For several groups of insects, migratory forms are more abundant in ephemeral habitats (25, 26, 29, 72, 82, 92, 107, 114). In a thorough study of the influence of habitat persistence and insect density on wing form (brachyptery versus macroptery) in planthoppers (Homoptera: Delphacidae), Denno et al (26) found that percent macroptery decreased significantly and became more sensitive to density with increased habitat persistence. This was so for a combined analysis of 35 species (41 populations), for pairwise comparison of congeners, and for interpopulation comparisons of *Prokelisia marginata*. In an analysis of wing dimorphism and diapause within and among 9 waterstriders species in the genus *Gerris* (Hemiptera: Gerridae), Vepsalainen (107) found that long wings or short-wing/long-wing dimorphism cued to environmental conditions was associated with ephemeral habitats, such as small ponds likely to dry up. Because these studies reveal a trend in phenotypic variability across a gradient in environmental uncertainty, they suggest that risk-spreading may have favored wing dimorphism. However, it is logistically difficult to test the role of risk-spreading in migration. Furthermore, migration is often under genetic control (85, 107, 112, 114), and in these cases is not risk-spreading. When differences in migration are not genetic, migration is usually cued by an environmental influence either directly on an individual or on its mother or grandmother (26, 27, 34, 39, 107, 114). Thus, although risk-spreading might indeed influence the evolution of migration, its role is obscured by genetic variation

and environmental cues. I have been unable to find any studies that show strong evidence for phenotypic variation that was not either genetic in origin or cued to changes in the environment.

Spatial Distribution of Oviposition

Risk-spreading has been invoked as an explanation for the spatial distribution of progeny among host plants by herbivores or among host patches by parasitoids (21, 66, 87, 97). In some cases, the behavior described is avoidance of density-dependent mortality (e.g. 66) and thus is not risk-spreading in the sense used here. In other cases, risk-spreading in space and time have not been distinguished (e.g. 97), so analysis of mechanisms is difficult. However, there have been a few determined efforts to study spatial risk-spreading in insect oviposition.

In both laboratory and field experiments, females of *Anagrus delicatus* (Hymenoptera: Mymaridae) with many unlaid eggs left patches containing abundant unparasitized planthopper hosts (*Prokelisia marginata*) (21). In an elegant series of experiments and observations, Cronin & Strong (21) showed that departing parasitoids carried viable eggs; parasitoids were not limited by handling time per host; unparasitized hosts left behind were accessible and suitable for development; parasitoids did not leave because of low host density or high frequency of parasitized hosts; and parasitoids departed before oviposition rate declined. Because 20–30% of host plant leaves senesced in the field before eclosion of *A. delicatus* progeny, Cronin & Strong (21) concluded that risk-spreading to avoid loss of too many eggs at once on senescing leaves was the most likely explanation for the low parasitism of *P. marginata* in patches visited by *A. delicatus*. However, using mathematical models of parasitoid foraging, Rosenheim & Mangel (88) showed that avoidance of self-superparasitism could also explain the behavior of *A. delicatus*, if it were egg limited and discriminated poorly among parasitized and unparasitized hosts, which is the case (20). This does not mean that *A. delicatus* does not bet-hedge in its distribution of eggs among host patches, but it points out a weakness in testing a hypothesis by eliminating other hypotheses: One cannot always be certain that the list of hypotheses eliminated is complete. An analysis like that of Bradford & Roff (7), who explicitly calculated long-term fitness and expected behavior under risk-spreading would be useful. A comparison of oviposition behavior at several levels of leaf senescence would be even more useful.

Females of *Pieris rapae* (Lepidoptera: Pieridae) fly more linearly when searching for host plants on which to oviposit than when foraging for nectar, pass up apparently suitable host plants, and usually lay eggs singly on host plants (87). Root & Kareiva (87) eliminated the following hypotheses as explanations for these behaviors: differences in host plant suitability between plants

passed over and those chosen for oviposition; a refractory period between ovipositions; and density-dependent mortality of eggs and larvae at several spatial scales. Root & Kareiva (87) concluded that *P. rapae* females were spreading their eggs spatially to avoid unpredictably variable mortality among host plants. Simulations with a mathematical model of *P. rapae* oviposition and mortality, using actual data on mortality among plants and gardens, indicated that female replacement rate increased with spread of eggs among plants and gardens (87). Because spreading of eggs among hosts plants appears costly, Root & Karieva (87) argued that *P. rapae* was trading lower mean fitness for lower variance in fitness, i.e. risk-spreading. In a critique of this research, Courtney (19) pointed out that theory suggests that within-generation risk-spreading is likely only at small population sizes (41–43) and that there was evidence for density-dependent mortality of *P. rapae* larvae. Root & Kareiva (86) rebutted that other models, including their own, came to different conclusions and disagreed with Courtney's analysis of the data on density-dependent versus density-independent mortality.

Rehashing the evidence on density-independent versus density-dependent mortality at this point would not be worthwhile. Nevertheless, I make two points about theory. First, Root & Kareiva (86) lump models of within-generation risk-spreading and metapopulation risk-spreading. However, as discussed above, within-generation risk-spreading depends on population size, but metapopulation risk-spreading does not. Although in the analysis of mortality, Root & Kareiva (87) consider both metapopulation and within-generation risk-spreading, their observations on oviposition concern within-generation risk-spreading. Second, although the text is ambiguous, it appears that the empirical mathematical model Root & Kareiva (87) developed was actually for temporal risk-spreading and not within-generation or metapopulation risk-spreading. Thus, it was inappropriate for their analysis of *P. rapae* behavior. A comparative analysis of behavior across a gradient of environmental uncertainty would be more powerful than elimination of other hypotheses. If geometric mean fitnesses could be measured, all the better, although perhaps one should not ask for too much.

Janzen (55) argues that aphid individuals are not genetic individuals. Thus, the unit of selection is actually a clone because individuals of a clone share the same genotype and because members of a clone descend from a more or less recent common ancestor. If one accepts this argument, it means that the spread of individuals of a clone in space is may be risk-spreading. Because it is within-generation risk-spreading, it is likely to be selected for only in the face of countervailing selection when population size is small; but here the population size is the number of clones, which might actually be rather small.

Other Traits

Risk-spreading has been invoked to explain phenotypic variation in various traits besides diapause and migration—for example, egg size (9, 89), ovariole number (109), mate choice (110). If, as in *Lymantria dispar* (Lepidoptera: Lymantriidae) (89), egg size affects development time, phenotypic variation in egg size could be diversifying temporal risk-spreading. However, small eggs in *L. dispar* incur large fitness costs later in life (89), and a few days' difference in development time seems hardly adequate to compensate for these costs. Variation in ovariole number in aphids (109) may be a response to temporal uncertainty in the environment. Yet even aphid clones may not remain genetically homogeneous (111), so the possibility of genetic variation rather than risk-spreading needs to be explored. Females of *Linyphia litigiosa* (Araneae: Linyphiidae) mate with multiple males (110). After eliminating search for the best male and maximization of genetic diversity as hypotheses to explain this behavior, Watson (110) concluded that female *L. litigiosa* were risk-spreading to avoid having all their progeny result from union with an inferior male. However, because many individuals of the same genotype in a population have the opportunity to mate with many males, this would be a form of within-generation risk-spreading, and thus unlikely unless population sizes were very small. All of these studies would be strengthened by tests for genetic differences among phenotypes, estimates of arithmetic versus geometric mean fitnesses, and comparisons across gradients in environmental uncertainty.

CONCLUSIONS

The theory of risk-spreading has held out the promise of explaining patterns in behavior and life history of insects. Although the theory is voluminous and well developed in some ways, it has rarely been used to generate detailed, testable hypotheses about the evolution of risk-spreading. Furthermore, although there is evidence for risk-spreading, particularly in facultative diapause, I have been unable to find any definitive tests with unequivocal results showing that risk-spreading has been a major factor in the evolution of insect behaviors or life histories. In many studies, evidence and theory have not been tightly linked, which has weakened tests of the role of risk-spreading. Environments are rarely completely unpredictable, and natural selection strongly favors use of signals about the future environment. This does not mean that risk-spreading does not contribute to evolution of behaviors or life histories, but it does make testing for risk-spreading difficult. Despite clarifications concerning what should and should not be considered risk-spreading (90), few studies that address diversifying risk-spreading have eliminated genetic variation as a cause of phenotypic

variation. Phenotypic variation in many traits has some genetic component, and although the nongenetic component of phenotypic variation may indeed be risk-spreading, distinguishing the effects of genetic versus nongenetic variation is challenging.

To advance our understanding of risk-spreading in the wild, we need (*a*) explicit empirical models that predict levels of diversifying risk-spreading for several insect populations in several environments that vary in uncertainty and (*b*) tests of these models using measurements of phenotypes and their fitnesses over several generations in each environment. One might argue that this is asking too much for many traits and insect populations. However, facultative diapause is amenable to such an analysis, and indeed much of the above has been done for one system (5–7, 71). In this system, the amount of risk-spreading was relatively minor. What about other insect systems?

ACKNOWLEDGMENTS

Jay Rosenheim, Fred Gould, and Owain Edwards provided stimulating discussion of the ideas. Richard Turcotte helped greatly in obtaining the literature for review.

Literature Cited

1. Andrewartha HG, Birch LC. 1954. *The Distribution and Abundance of Animals*. Chicago: Univ. Chicago Press
2. Ayal Y, Green RF. 1993. Optimal egg distribution among host patches for parasitoids subject to attack by hyperparasitoids. *Am. Nat.* 141:120–38
3. Bigelow RS. 1960. Interspecific hybrids and speciation in the genus *Acheta* (Orthoptera, Gryllidae). *Can. J. Zool.* 38: 509–24
4. Bradford MJ. 1991. *The role of environmental heterogeneity in the evolution of life history strategies of the striped ground cricket*. PhD thesis. McGill University, Montreal. 134 pp.
5. Bradford MJ, Roff DA. 1993. Bet hedging and the diapause strategies of the cricket *Allonemobius fasciatus*. *Ecology* 74:1129–35
6. Bradford MJ, Roff DA. 1995. Genetic and phenotypic sources of life history variation along a cline in voltinism in the cricket *Allonemobius socius*. *Oecologia* 103:319–26
7. Bradford MJ, Roff DA. 1997. An empirical model of diapause strategies of the cricket *Allonemobius socius*. *Ecology* 78:442–51
8. Bradshaw WE, Holzapfel CM. 1990. Evolution of phenology and demography in the pitcher plant mosquito, *Wyeomyia smithii*. In *Insect Life Cycles: Genetics, Evolution and Coordination*, ed. F Gilbert, pp. 47–67. Berlin: Springer
9. Capinera JL. 1979. Qualitative variation in plants and insects: effect of propagule size on ecological plasticity. *Am. Nat.* 114:350–61
10. Charnov EL, Schaffer WM. 1973. Life history consequences of natural selection: Cole's result revisited. *Am. Nat.* 107:791–3
11. Chesson PL. 1985. Coexistence of competitors in spatially and temporally varying environments: a look at the combined effects of different sorts of variability. *Theor. Popul. Biol.* 28:263–87
12. Cohen D. 1966. Optimizing reproduction in a randomly varying environment. *J. Theor. Biol.* 12:119–29
13. Cohen D. 1967. Optimization of seasonal

migratory behavior. *Am. Nat.* 101:5–17
14. Cohen D. 1967. Optimizing reproduction in a randomly varying environment when a correlation may exist between the conditions at the time a choice has to be made and the subsequent outcome. *J. Theor. Biol.* 16:1–14
15. Cohen D. 1968. A general model of optimal reproduction in a randomly varying environment. *J. Ecol.* 56:219–28
16. Cohen D. 1970. A theoretical model for the optimal timing of diapause. *Am. Nat.* 104:389–400
17. Cooper WS, Kaplan RH. 1982. Adaptive "coin-flipping": a decision-theoretic examination of natural selection for random individual variation. *J. Theor. Biol.* 94:135–51
18. Corkum LD, Ciborowski JJH, Poulin RG. 1997. Effects of emergence date and maternal size on egg development and sizes of eggs and first-instar nymphs of a semelparous aquatic insect. *Oecologia* 111:69–75
19. Courtney SP. 1986. Why insects move between host patches: some comments on 'risk-spreading.' *Oikos* 47:112–14
20. Cronin JT, Strong DR. 1993. Superparasitism and mutual interference in the egg parasitoid *Anagrus delicatus* (Hymenoptera: Mymaridae). *Ecol. Entomol.* 18:293–302
21. Cronin JT, Strong DR. 1993. Substantially submaximal oviposition rates by a mymarid egg parasitoid in the laboratory and field. *Ecology* 74:1813–25
22. Crow JF, Kimura M. 1970. *An Introduction to Population Genetics Theory*. New York: Harper & Row
23. Dempster JP. 1991. Fragmentation, isolation, and mobility of insect populations. In *Conservation of Insects and Their Habitats*, ed. NM Collins, JA Thomas, pp. 143–53. New York: Academic
24. Den Boer PJ. 1968. Spreading of risk and stabilization of animal numbers. *Acta Biotheor.* 18:165–94
25. Den Boer PJ. 1990. The survival value of dispersal in terrestrial arthropods. *Biol. Conserv.* 54:175–92
26. Denno RF, Roderick GK, Olmstead KL, Dobel HG. 1991. Density-related migration in planthoppers (Homoptera: Delphacidae): the role of habitat persistence. *Am. Nat.* 138:1513–41
27. Dingle H. 1979. Adaptative variation in the evolution of insect migration. In *Movement of Highly Mobile Insects: Concepts and Methodology*, ed. RL Rabb, GG Kennedy, pp. 64–87. Raleigh: North Carolina State Univ. Press
28. Dingle H. 1981. Geographic variation and behavioral flexibility in milkweed bug life histories. In *Insect Life History Patterns: Habitat and Geographic Variation*, ed. RS Denno, H Dingle, pp. 57–73. New York: Springer
29. Dingle H. 1996. *Migration: the Biology of Life on the Move*. Oxford: Oxford Univ. Press
30. Dingle H, Alden BM, Blakley NR, Kopec D, Miller ER. 1980. Variation in photoperiodic response within and among species of milkweed bugs (*Oncopeltus*). *Evolution* 34:356–70
31. Dingle H, Blakley NR, Miller ER. 1980. Variation in body size and flight performance in milkweed bugs (*Oncopeltus*). *Evolution* 34:371–85
32. Dingle H, Brown CK, Hegmann JP. 1977. The nature of genetic variance influencing photoperiodic diapause in a migrant insect, *Oncopeltus fasciatus*. *Am. Nat.* 111:1047–59
33. Dingle H, Mousseau TA, Scott SM. 1990. Altitudinal variation in life cycle syndromes of California populations of the grasshopper, *Melanoplus sanguinipes* (F.). *Oecologia* 84:199–206
34. Dixon AFG, Wellings PW. 1982. Seasonality and reproduction in aphids. *Intl. J. Invert. Reprod.* 5:83–89
35. Dixon AFG, Wratten SD. 1971. Laboratory studies on aggregation, size and fecundity in the black bean aphid, *Aphis fabae* Scop. *Bull. Entomol. Res.* 61:97–111
36. Eber S, Brandl R. 1996. Metapopulation dynamics of the tephritid fly *Urophora cardui*: an evaluation of incidence-function model assumptions with field data. *J. Anim. Ecol.* 65:621–30
37. Endler JA. 1986. *Natural Selection in the Wild*. Princeton: Princeton Univ. Press
38. Fontes EG, Pires CSS, Sujii ER. 1995. Mixed risk-spreading strategies and the population dynamics of a Brazilian pasture pest, *Deois flavopicta* (Homoptera: Cercopidae). *J. Econ. Entomol.* 88:1256–62
39. Gatehouse AG. 1997. Behavior and ecological genetics of wind-borne migration by insects. *Annu. Rev. Entomol.* 42:475–502
40. Gillespie JH. 1973. Natural selection with varying selection coefficients—a haploid model. *Genet. Res.* 21:115–20
41. Gillespie JH. 1974. Natural selection for within-generation variance in offspring number. *Genetics* 76:601–6

42. Gillespie JH. 1975. Natural selection for within-generation variance in offspring number. II. Discrete haploid models. *Genetics* 81:403–13
43. Gillespie JH. 1977. Natural selection for variances in offspring numbers: a new evolutionary principle. *Am. Nat.* 111: 1010–14
44. Gillespie JH. 1981. The role of migration in the genetic structure of populations in temporally and spatially varying environments: III. Migration modification. *Am. Nat.* 117:223–33
45. Goodman D. 1984. Risk spreading as an adaptive strategy in iteroparous life histories. *Theor. Popul. Biol.* 25:1–20
46. Groeters FR. 1994. The adaptive role of facultative embryonic diapause in the grasshopper *Caledia captiva* (Orthoptera: Acrididae) in southeastern Australia. *Ecography* 17:221–28
47. Hastings A, Harrison S. 1994. Metapopulation dynamics and genetics. *Annu. Rev. Ecol. Syst.* 25:167–88
48. Heliovaara K, Valsanen R, Simon C. 1994. Evolutionary ecology of periodical insects. *Trends Ecol. Evol.* 9:475–80
49. Hill JK, Thomas CD, Lewis OT. 1996. Effects of habitat patch size and isolation on dispersal by *Hesperia comma* butterflies: implications for metapopulation structure. *J. Anim. Ecol.* 65:725–35
50. Hoffman RJ. 1978. Environmental uncertainty and evolution of physiological adaptation in *Colias* butterflies. *Am. Nat.* 112:999–1015
51. Holtzer TO, Bradley JR, Rabb RL. 1976. Geographic and genetic variation in time required for emergence of diapausing *Heliothis zea*. *Ann. Entomol. Soc. Am.* 69: 261–65
52. Hoy MA. 1978. Variability in diapause attributes of insects and mites: some evolutionary and practical implications. In *Evolution of Insect Migration and Diapause*, ed. H Dingle, pp. 101–26. New York: Springer
53. Istock C. 1981. Natural selection and life history variation: theory plus lessons from a mosquito. In *Insect Life History Patterns: Habitat and Geographic Variation*, ed. RF Denno, H Dingle, pp. 113–27. New York: Springer
54. Istock CA. 1978. Fitness variation in a natural population. In *Evolution of Insect Migration and Diapause*, ed. H Dingle, pp. 171–90. New York: Springer
55. Janzen DH. 1975. What are dandelions and aphids? *Am. Nat.* 109:586–89
56. Kaplan RH, Cooper WS. 1984. The evolution of developmental plasticity in reproductive characteristics: an application of the "adaptive coin-flipping" principle. *Am. Nat.* 123:393–410
57. Kingsolver JG. 1979. Thermal and hydric aspects of environmental heterogeneity in the pitcher plant mosquito. *Ecol. Monogr.* 49:357–76
58. Kuno E. 1981. Dispersal and the persistence of populations in unstable habitats: a theoretical note. *Oecologia* 49:123–26
59. Leimar O, Norberg U. 1997. Metapopulation extinction and genetic variation in dispersal-related traits. *Oikos* 80:448–58
60. Levin SA, Cohen D, Hastings A. 1984. Dispersal strategies in patchy environments. *Theor. Popul. Biol.* 26:156–91
61. Levins R. 1962. Theory of fitness in a heterogeneous environment. I. The fitness set and adaptive function. *Am. Nat.* 96:361–73
62. Levins R. 1968. *Evolution in Changing Environments: Some Theoretical Explorations*. Princeton: Princeton Univ. Press
63. Levins R. 1969. Some demographic and genetic consequences of environmental heterogeneity for biological control. *Bull. Entomol. Soc. Am.* 15:237–40
64. Levins R. 1970. Extinction. *Lect. Math. Life Sci.* 2:75–107
65. Lewontin RC, Cohen D. 1969. On population growth in a randomly varying environment. *Proc. Nat. Acad. Sci. USA* 62: 1056–60
66. Mackauer M, Voelkl W. 1993. Regulation of aphid populations by aphidiid wasps. Does parasitoid foraging behaviour or hyperparasitism limit impact? *Oecologia* 94:339–50
67. Manly BFJ. 1985. *The Statistics of Natural Selection*. New York: Chapman & Hall
68. Martin A, Simon C. 1990. Temporal variation in insect life cycles: lessons from periodical cicadas. *Bioscience* 40:359–67
69. Metz JAJ, de Jong TJ, Klinkhamer PGL. 1983. What are the advantages of dispersing: a paper by Kuno explained and extended. *Oecologia* 57:166–69
70. Mountford MD. 1971. Population survival in a variable environment. *J. Theor. Biol.* 32:75–79
71. Mousseau TA, Roff DA. 1989. Adaptation to seasonality in a cricket: patterns of phenotypic and genotypic variation in body size and diapause expression along a cline in season length. *Evolution* 43:1483–96
72. Novotny V. 1994. Relation between temporal persistence of host plants and wing length in leafhoppers (Hemiptera: Auchenorrhyncha). *Ecol. Entomol.* 19: 168–76
73. Olivieri I, Michalakis Y, Gouyon P-H.

1995. Metapopulation genetics and the evolution of dispersal. *Am. Nat.* 146:202–28
74. Palmer AR, Strathmann RR. 1981. Scale of dispersal in varying environments and its implications for life histories of marine invertebrates. *Oecologia* 48:308–18
75. Philippi T, Seger J. 1989. Hedging one's evolutionary bets, revisited. *Trends Ecol. Evol.* 4:41–44
76. Real L, Caraco T. 1986. Risk and foraging in stochastic environments. *Annu. Rev. Ecol. Syst.* 17:371–90
77. Real L, Ott J, Silverfine E. 1982. On the tradeoff between the mean and the variance in foraging: effect of spatial distribution and color preference. *Ecology* 63:1617–23
78. Real LA. 1980. Fitness, uncertainty, and the role of diversification in evolution and behavior. *Am. Nat.* 115:623–38
79. Reddingius J, Den Boer PJ. 1970. Simulation experiments illustrating stabilization of animal numbers by spreading risk. *Oecologia* 5:240–84
80. Roff DA. 1974. The analysis of a population model demonstrating the importance of dispersal in a heterogeneous environment. *Oecologia* 15:259–75
81. Roff DA. 1983. Phenological adaptation in a seasonal environment: a theoretical perspective. In *Diapause and Life Cycle Strategies of Insects*, ed. VK Brown, I Hodek, pp. 253–73. The Hague: Junk
82. Roff DA. 1990. The evolution of flightlessness in insects. *Ecol. Monogr.* 60:389–421
83. Roff DA. 1994. Habitat persistence and the evolution of wing dimorphism in insects. *Am. Nat.* 144:772–98
84. Roff DA, Bradford MJ. 1996. Quantitative genetics of the trade-off between fecundity and wing dimorphism in the cricket *Allonemobius socius*. *Heredity* 76: 178–85
85. Roff DA, Fairbairn DJ. 1991. Wing dimorphisms and the evolution of migratory polymorphisms in the Insecta. *Am. Zool.* 31:243–51
86. Root RB, Kareiva P. 1986. Is risk-spreading so unrealistic? *Oikos* 47:114–16
87. Root RB, Kareiva PM. 1984. The search for resources by cabbage butterflies (*Pieris rapae*): ecological consequences and adaptive significance of Markovian movements in a patchy environments. *Ecology* 65:147–65
88. Rosenheim JA, Mangel M. 1994. Patch-leaving rules for parasitoid with imperfect host discrimination. *Ecol. Entomol.* 19:374–80
89. Rossiter MC. 1991. Maternal effects generate variation in life history: consequences of egg weight plasticity in the gypsy moth. *Funct. Ecol.* 5:386–93
90. Seger J, Brockman HJ. 1987. What is bet-hedging? *Oxf. Surv. Evol. Biol.* 4:182–211
91. Simons AM, Johnston MO. 1997. Developmental instability as a bet-hedging strategy. *Oikos* 80:401–6
92. Southwood TRE. 1962. Migration of terrestrial arthropods in relation to habitat. *Biol. Rev.* 37:171–214
93. Stearns SC. 1976. Life-history tactics: a review of the ideas. *Q. Rev. Biol.* 51:3–47
94. Strathmann R. 1974. The spread of sibling larvae of sedentary marine invertebrates. *Am. Nat.* 108:29–44
95. Sutcliffe OL, Thomas CD, Moss D. 1996. Spatial synchrony and asynchrony in butterfly population dynamics. *J. Anim. Ecol.* 65:85–95
96. Takahashi F. 1977. Generation carryover of a fraction of population members as an animal adaptation to unstable environmental conditions. *Res. Popul. Ecol.* 18: 235–42
97. Tallamy DW, Denno RS. 1981. Alternative life history patterns in risky environments: an example from lacebugs. In *Insect Life-History Patterns: Habitat and Geographic Variation*, ed. RS Denno, H Dingle, pp. 129–47. New York: Springer
98. Tanaka S. 1992. The significance of embryonic diapause in a Japanese strain of the migratory locust, *Locusta migratoria* (Orthoptera: Acrididae). *Jpn J. Entomol.* 60:503–20
99. Tauber MJ, Tauber CA, Masaki S. 1986. *Seasonal Adaptations of Insects*. New York: Oxford Univ. Press
100. Taylor F. 1986. The fitness functions associated with diapause induction in arthropods I. The effects of age structure. *Theor. Popul. Biol.* 30:76–92
101. Taylor F. 1986. The fitness functions associated with diapause induction in arthropods II. The effects of fecundity and survivorship on the optimum. *Theor. Popul. Biol.* 30:93–110
102. Taylor F. 1989. Diapause induction in changing photoperiods. *J. Theor. Biol.* 139:103–16
103. Taylor F, Spalding JB. 1986. Geographical patterns in the photoperiodic induction of hibernal diapause. In *The Evolution of Insect Life Cycles*, ed. F Taylor, R Karban, pp. 66–85. Berlin: Springer-Verlag
104. Taylor F, Spalding JB. 1988. Fitness functions for alternative developmental path-

ways in the timing of diapause induction. *Am. Nat.* 131:678–99
105. Taylor F, Spalding JB. 1989. Timing of diapause in relation to temporally variable catastrophes. *J. Evol. Biol.* 2:285–97
106. Van Valen L. 1971. Group selection and the evolution of dispersal. *Evolution* 25: 591–98
107. Vepsalainen K. 1978. Wing dimorphism and diapause in Gerris: determination and adaptative significance. In *Evolution of Insect Migration and Diapause*, ed. H Dingle, pp. 218–53. New York: Springer
108. Walker TJ. 1980. Mixed oviposition in individual females of *Gryllus firmus*: graded proportions of fast-developing and diapause eggs. *Oecologia* 47:291–98
109. Ward SA, Dixon AFG. 1984. Spreading the risk, and the evolution of mixed strategies: seasonal variation in aphid reproductive biology. In *Advances in Invertebrate Reproduction*, ed. W Engels, 3: 367–86. New York: Elsevier
110. Watson PJ. 1991. Multiple paternity as genetic bet-hedging in female sierra dome spiders, *Linyphia litigiosa* (Linyphiidae). *Anim. Behav.* 41:343–60
111. Wilhoit LR, Mittler TE. 1991. Biotypes and clonal variation in greenbug (Homoptera: Aphididae) populations from a locality in California. *Environ. Entomol.* 20:757–67
112. Wilson K. 1995. Insect migration in heterogeneous environments. In *Insect Migration: Tracking Resources Through Space and Time*, ed. VA Drake, AG Gatehouse, pp. 243–63. Cambridge, UK: Cambridge Univ. Press
113. Wratten SD. 1977. Reproductive strategy of winged and wingless morphs of the aphids *Sitobion avenae* and *Metopolophium dirhodum*. *Ann. Appl. Biol.* 85: 319–31
114. Zera AJ, Denno RF. 1997. Physiology and ecology of dispersal polymorphism in insects. *Annu. Rev. Entomol.* 42:207–31

Annual Review of Entomology
Volume 44, 1999

CONTENTS