

STOCHASTIC POLYPHENISM: COPING WITH UNCERTAINTY

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"A potentially important source of individual phenotypic variation is examined which has heretofore received little recognition as a general and probably widespread phenomenon." (Cooper and Kaplan 1982, p. 135)

SYNOPSIS

Polyphenism (i.e., polymorphism in which non-genetic differences cause the development of the contrasting forms) can be *conditional* or *stochastic*. In conditional polyphenism, a genotype responds to different current environments that predict different future environments by producing different, appropriate phenotypes. For example, short days may cause the development of the diapause phenotype, appropriate to winter, and long days may cause the development of the nondiapause phenotype, appropriate to summer. In stochastic polyphenism, a genotype responds to differences in its environment that occur with probabilities approximating the probabilities of different future environments. For example, if 30% of winters are severe enough to require diapause and the other 70% yield a corresponding advantage to nondiapausing morphs, a genotype would produce the diapause phenotype 30% of the time and the nondiapause phenotype the other 70%. Modeling and empirical evidence support the concept of stochastic polyphenism.

In a constant environment natural selection should lead to genotypes that develop into individuals adapted to that environment. In a predictably varying environment natural selection should operate to yield genotypes that cause the development of individuals appropriate to whatever environment is coming next. In fact, genotypes that anticipate the deterioration of a local habitat or the progression of the seasons are commonplace in insects. Such genotypes switch development into one path or another in response to present conditions that predict the future.

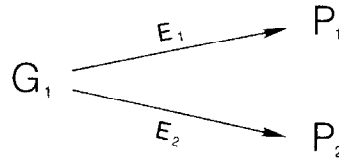
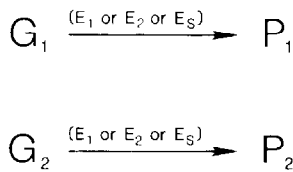
In environments that vary unpredictably—i.e., in truly uncertain environments—the outcome of evolution is hard to predict. The thesis of this paper is that genotypes which have probabilistic or stochastic output should prosper in uncertain environments and that such genotypes exist in insects.

Although I arrived at the notion of stochastic genes independently, the concept is not original. D. A. Roff (1975) investigated models in which the *probability* of dispersal was a function of genotype. W. S. Cooper and R. H. Kaplan (1982, p. 136; also, Kaplan and Cooper 1984) made a strong case for "intra-genotypic strategy-mixing or, less formally, adaptive coin flipping."

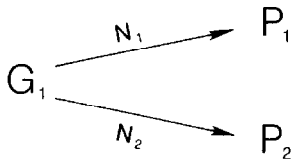
If genotypes have stochastic outputs, outputs of a single genotype could vary continuously (e.g., intensity of diapause, minimum duration of flight in a dispersing individual) or discontinuously (e.g., diapause vs. nondiapause; winged vs. apterous). Stochastic outputs of both types are indicated, but only discontinuous variation will be discussed here because it is more striking and easier to treat.

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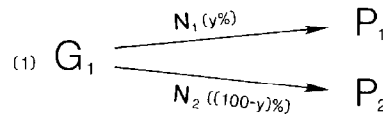
A GENETIC POLYMORPHISM C CONDITIONAL POLYPHENISM



B POLYPHENISM



D STOCHASTIC POLYPHENISM



$$(2) \quad E_s = y \% N_1 + (100 - y) \% N_2$$

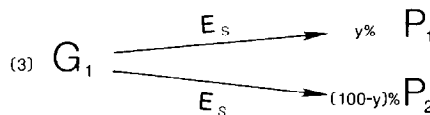


Fig. 1. Diagrams illustrating different types of polymorphism. (A) In *genetic polymorphism*, different phenotypes (P_1 and P_2) develop as a result of differences in genotype (G_1 and G_2). (B) In *polyphenism*, different phenotypes develop as a result of nongenetic differences (N_1 and N_2). (C) In *conditional polyphenism*, the nongenetic differences are environmental circumstances (E_1 and E_2) that predict different future environments to which the different phenotypes are adapted. (D) In *stochastic polyphenism*, (1) the nongenetic differences occur at probabilities that correlate with the probabilities of future environments to which the different phenotypes are adapted; (2) these stochastically occurring alternatives can be considered to comprise an "environment" (E_s), and (3) this environment causes the two phenotypes to be produced in the proportions of N_1 and N_2 .

POLYMORPHISM

Discontinuous variation occurring within a deme between individuals of the same ontogenetic stage is called *polymorphism* (see Kennedy 1961). Polymorphism can be divided into *genetic polymorphism* and *polyphenism* depending on what sort of differences lead to the development of the distinctive phenotypes. In genetic polymorphism the distinctive phenotypes result from differences in genotype, whereas in polyphenism nongenetic differences are responsible (Fig. 1a,b). It should be noted that polyphenism depends on a *genotype* that can be switched by the environment from one path of development to another.

Two modes of selection may lead to polyphenism. First, and most obvious, is that natural selection will increase genotypes that respond to different environments by producing different, appropriate phenotypes.¹ Such genotypes are responsible for *conditional polyphenism* (Fig. 1c). The defining aspect of this type of polyphenism is that

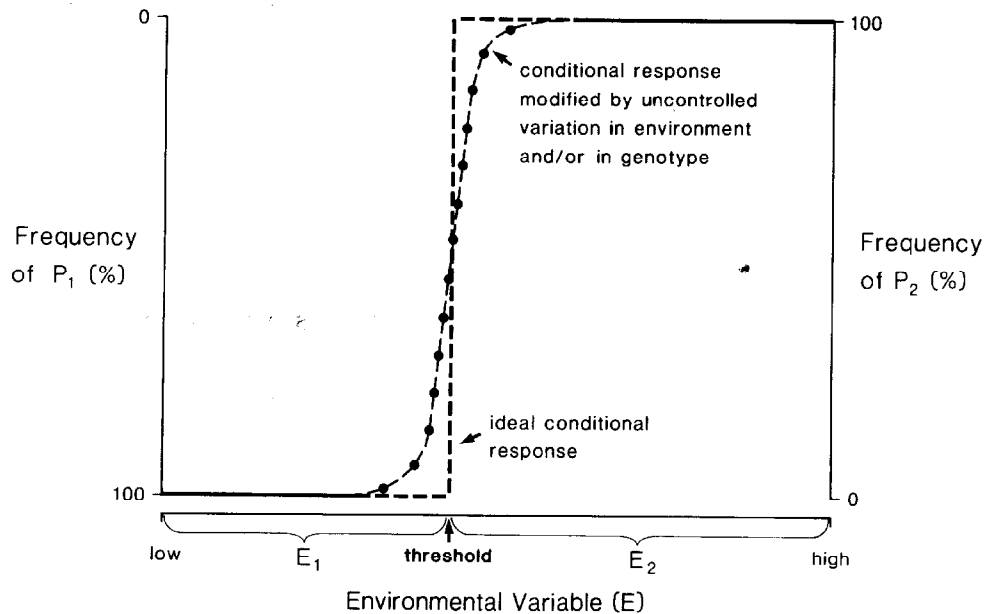


Fig. 2. The concept of *threshold* as applied to conditional polyphenism. An environmental variable ("E", abscissa) that varies continuously is divided into two regimes (E_1 and E_2) by a threshold. The transition from 100% of one phenotype (P_1) to 100% of the other (P_2) is likely to be sigmoid rather than rectilinear, and the best estimate of threshold becomes the value of E at which 50% of the exposed population develop into P_1 and 50% into P_2 .

development of a phenotype adapted to a specific future environment is conditional upon a particular present environment that is correlated with the future one. Examples include development of seasonally appropriate diapausing or nondiapausing individuals (Beck 1980) or flight-worthy or flightless individuals (Harrison 1980) in response to photoperiod. Conditional polyphenism depends on a genotype responding to one environmental condition by developing one phenotype and to an alternative condition by developing another. The transition between these two conditions is a threshold that delimits the production of 100% of one phenotype from the production of 100% of the other (Fig. 2). Ideally the threshold is sharp, but in practice it is likely to be blurred, because individuals in the responding population are not genetically identical (and therefore vary in their thresholds) and because individuals in a population do not experience identical environments even under carefully controlled circumstances (Fig. 2).

The second mode of selection is that a genotype may increase because it produces alternative phenotypes in proportions that approximate the probabilities of future environments that favor the respective phenotypes. Such selection would lead to *stochastic polyphenism* (Fig. 1d).

If stochastic polyphenism occurs, (1) genes must be able to effect a stochastic output and (2) such genes must be able to increase at the expense of genes that have a deterministic output. If these two conditions are met, (3) one would expect to find examples of stochastic polyphenism in insects living in environments exhibiting important, short- to medium-term, unpredictable variation. The next three sections of this paper deal with these subjects.

CAN GENES GAMBLE?

For genes to produce a stochastic output they must make use of environmental differences that occur in the right proportions without pattern. To use the metaphor of Cooper and Kaplan (1982), they must flip coins (properly biased to produce the needed proportions of heads and tails).

Recent advances in genetics suggest a variety of ways that genes might, in effect, gamble with the phenotype of their carrier. Transposable genetic elements ("jumping genes"), identified by Barbara McClintock in maize and now known to be commonplace in other plants and in animals (Fedoroff 1984), offer one such possibility. The developmental effect of a mobile element depends on its position in the genome. For instance, if it attaches to site "A" 40% of the time and to site "B" 60% of the time, the outcome would be 40% of one phenotype and 60% of another. The genomes would be the same except for the positions of their components, but the phenotypes produced would be determined stochastically.² Another possibility was suggested by Spudich and Koshland (1976), who demonstrated individual variation in genetically identical bacterial cells grown in a homogeneous environment. In trying to account for the variations, they noted that certain molecules occur in such small numbers in cells that their numbers are subject to "Poissonian variation" when cells divide and that this could lead to significant random variation in phenotype.

A final example is from immunology. The ability to produce millions of different antibodies is passed from generation to generation in a limited number of genes. These genes specify a "kit of [genetic] components," which are "shuffled" in the developing *B* lymphocytes leading "to a different result in each of millions of lines of cells" (Leder 1982, p. 102). Even millions of different phenotypes can be produced stochastically by a single genotype.

CAN STOCHASTIC GENES COMPETE?

If stochastic genes can arise by mutation from deterministic forebears, how well will they compete. Simulation modeling is a suitable way to investigate this question. Using a microcomputer, I developed two models that test stochastic genes in uncertain environments.³ In each model, the environment was made to vary without pattern between two states.⁴

The first model concerned insects that sometimes benefit from diapause and sometimes benefit by continuing development. One environmental state (e.g., mild winter), occurring $p\%$ of the time, was more favorable to the nondiapause phenotype. The other state (e.g., severe winter), occurring $(100-p)\%$ of the time, was more favorable to the diapause phenotype. Diapause was determined by three alleles at one locus: D_D , producing the diapause phenotype; D_N , nondiapause phenotype; and D_S , $y\%$ diapause phenotype and $(100-y)\%$ nondiapause phenotype.⁵ The following conditions were specified to start the model: dominance relations of the alleles, initial frequencies of alleles, proportion of generations that environment favors diapause phenotype (i.e., p), proportion of diapause phenotypes produced by D_S (i.e., y), relative fitness of diapause phenotype when the environment is less favorable to it, relative fitness of nondiapause phenotype when the environment is less favorable to it, number of generations per run of model, and number of runs.

Table 1 gives representative results for the first model. When allele D_S was recessive to the other two alleles, it quickly replaced them under a variety of scenarios—even if the probability of the diapause phenotype deviated greatly from the probability of the diapause-favoring environment (e.g., Table 1, A and B). When the other alleles were recessive, they persisted more than 400 generation under a wide range of assumptions,

TABLE 1. REPRESENTATIVE RESULTS OF A SIMULATION MODEL IN WHICH AN ALLELE WITH MIXED OUTPUT (D_S) COMPETED WITH ALLELES FOR DIAPAUSE (D_D) AND NONDIAPAUSE (D_N). ENVIRONMENT VARIED WITHOUT PATTERN BETWEEN FAVORING DIAPAUSE AND NONDIAPAUSE PHENOTYPES. ^a RESULTS OF SIMULATIONS MADE WITH SIMILAR ASSUMPTIONS ARE GROUPED (A-D).

Dominance	Prob. diapause envir. ^b (p)	Prob. diapause phenotype ^c (y)	Relative fitness when not favored ^d		Allele frequencies ^e		
			diapause phenotype	nondiap. phenotype	D_D	D_N	D_S
A) $D_D > D_N > D_S$	0.50	0.50	0.01	0.01	0.00	0.00	1.00
$D_D > D_S > D_N$	"	"	"	"	0.00	0.04	0.96
$D_S > D_D > D_N$	"	"	"	"	0.01	0.05	0.94
B) $D_D > D_N > D_S$	0.50	0.01	0.01	0.01	0.00	0.00	1.00
"	"	0.99	"	"	0.00	0.00	1.00
C) $D_S > D_D > D_N$	0.70	0.70	0.01	0.01	0.04	0.04	0.92
"	"	0.50	"	"	0.30	0.19	0.51
"	"	0.90	"	"	0.00	0.33	0.67
D) $D_S > D_D > D_N$	0.50	0.50	0.50	0.01	0.95	0.05	0.00
"	"	0.70	"	"	0.95	0.05	0.00
"	"	0.90	"	"	0.76	0.04	0.20
"	"	0.99	"	"	0.49	0.04	0.47

^aModel assumed discrete generations, random mating, and continuously large population. Initial frequencies of alleles were 0.33 (but results are independent of starting frequencies).

^bProbability that environment favoring diapause over nondiapause will occur. (1.00-p = probability of environment favoring nondiapause.)

^cProportion of D_S - controlled individuals having the diapause phenotype. (1.00-y = proportion having nondiapause phenotype.)

^dFavored phenotype has fitness of 1.00.

^eMean allele frequencies after 400 generations in 100 runs of the model. Standard errors are <0.005 for A and B and <10% of the mean for C and D. No consistent change in frequencies occurred when these allele frequencies were used to initialize the model and the model was run an additional 100 generations. However, when the model was run 1000 generations or more, further changes occurred.

TABLE 2. REPRESENTATIVE RESULTS OF A SIMULATION MODEL IN WHICH AN ALLELE FOR MIXED OUTPUT (P_S) COMPETED WITH ALLELES FOR ONE (P_O) OR TWO (P_T) WINTERS OF DIAPAUSE. ENVIRONMENT VARIED WITHOUT PATTERN BETWEEN GROWING SEASONS THAT FAVORED INDIVIDUALS THAT BROKE DIAPAUSE AND THOSE THAT DISFAVORED THEM.^a A) EFFECT OF DOMINANCE. B) EFFECTS OF PROBABILITY OF UNFAVORABLE GROWING SEASON, w , AND a . C) EFFECT OF MAKING UNFAVORABLE GROWING SEASONS LESS UNFAVORABLE.

Dominance	Prob. unfav. growing season	Maximum winters for P_S^b (w)	Annual attrition of p.d.'s ^c (a)	Fitness in unfav. growing season ^d	Allele frequencies ^e		
					P_O	P_T	P_S
A) $P_O > P_T > P_S$ $P_S > P_T > P_O$	0.50 "	2 "	0.10 "	0.01 "	0.00 0.08	0.00 0.18	1.00 0.74
B) $P_O > P_T > P_S$ " " " " " "	0.50 0.10 0.01 "	6 " " "	0.10 " " 0.01	0.01 " " "	0.00 0.00 0.75 0.10	0.00 0.00 0.08 0.01	1.00 1.00 0.17 0.89
"	"	2	0.10	"	0.55	0.00	0.45
C) $P_O > P_T > P_S$ "	0.50 "	2 "	0.10 "	0.10 0.50	0.00 0.71	0.00 0.09	1.00 0.20

^aModel assumed that individuals entered diapause no more than once, that only diapausing individuals survived winter, that mating was random among those breaking diapause each spring, that the number of their descendants entering diapause that fall depended only on the quality of the growing season, and that breeding populations were always large.

^b P_S allele causes geometrically decreasing proportions of individuals to remain in diapause for 1 to w winters ($0.5/2^{w-1}$ for winters 1 to ($w-1$)).

^cAnnual attrition of prolonged diapausers. Survival each winter beyond first was (1.00- a).

^dProportion of favorable-season number that enter diapause when growing season is unfavorable.

^eMean allele frequencies after 120 generations in 100 runs of the model. Initial allele frequencies were based on pilot runs and did not differ by more than 10% from mean final frequencies. Standard errors <0.01 for A and C; <0.03 for B.

and the average frequency of D_S after 400 generations depended on the assumptions. For example, when y was made to deviate from p and the fitnesses of the disfavored phenotypes were kept equal, the frequency attained by D_S decreased (Table 1, C, lines 2 and 3). On the other hand, if the fitnesses of the disfavored phenotypes were made unequal, the frequency realized by D_S was increased by changing its "setpoint" (i.e., y) toward a higher proportion of the less disfavored phenotype (Table 1, D).

The second model concerned insects in which a portion of diapausing individuals remains dormant for more than a year (e.g., Prebble 1941, Powell 1974, Ushatinskaya 1976). In the wheat-blossom midge (*Sitodiplosis mosellana*), for example, diapause may last as long as 12 years; the emergence from year to year is irregular, but the trend is for a smaller percentage of a cohort to emerge each succeeding year (Barnes 1952). A presumed advantage of prolonged diapause is that the individual may skip a poor or disastrous annual growing season and emerge in a favorable one. If bad seasons cannot be foretold by the time diapause is broken⁶, the *individual* should, on average, benefit by breaking diapause the first year—because the probability of happening on a good year is the same each year but the mortality associated with staying in diapause accumulates year after year. On the other hand, a *gene* that caused most carriers to emerge the first year and lesser numbers to emerge subsequent years should be superior to any single-output gene. The model to test the superiority of genes with mixed outputs made certain assumptions⁷; and, again, initial conditions had to be set.⁸

Table 2 gives representative results. When recessive, the allele with mixed output (P_S) sometimes quickly replaced the other alleles (Table 2, A-C). When unfavorable growing seasons were rare relative to the annual attrition rate, P_S was less successful (Table 2, B). Similarly, when unfavorable growing seasons were less severely unfavorable, P_S attained at lower frequencies (Table 2, C).

The results from these two models suggest that alleles producing a stochastic output can increase in frequency at the expense of competing deterministic alleles and sometimes completely replace them.

Other workers have tested models of stochastic alleles. Roff (1975) created a variety of complex models in which genes controlled the probability of dispersal among subpopulations that varied independently and randomly in their finite rates of increase (generally between 0 and 2.8). In his simplest version, he assumed the population was fixed for an allele that determined a stated dispersal probability, and he showed that populations with probabilities intermediate between 0 and 1.0 would persist. In another version, dispersal was controlled by a single locus with two alleles—a non-dispersal allele and a dispersal allele that produced dispersal and non-dispersal phenotypes in a specified ratio. The probability that dispersers would survive was also specified. Roff found that when the stochastic allele was recessive it either reached 100% and the population survived or, in cases where low survival of dispersers had been specified, the non-dispersal allele reached 100% and extinction ensued. When he made the stochastic allele partially dominant, the model reached an enduring genetic polymorphism under a wide range of assumptions about the probabilities that the two genotypes permitting dispersal would produce dispersal phenotypes.

Cooper and Kaplan (1982) were first to focus directly upon the fitness of genotypes with stochastic output. They used "decision trees" to compare the fitnesses of genotypes with deterministic and stochastic outputs in uncertain environments and concluded that genotypes with mixed output would win over deterministic genotypes under a variety of conditions and even if their output did not match exactly the probabilities of the alternative environments.

All efforts at simulation modeling of genotypes with mixed and pure outputs in uncertain environments have yielded similar results: stochastic alleles can replace or reach a lasting genetic polymorphism with alleles that have a single output.

EXAMPLES

Because stochastic polyphenism is expected to coexist with conditional polyphenism and genetic polymorphism, with all three affecting the same traits, clearcut examples may be difficult to identify even if the phenomenon is frequent. Some examples from insects are instructive.

APHIDS

Aphids are remarkably adapted to the study of polyphenism. All species are polymorphic and produce several to many generations of viviparous parthenogenetic females each year (Dixon 1985a). Parthenogenetic reproduction in aphids apparently includes no recombination, making all parthenogenetically produced females genetically identical to their mothers, except for new mutations (Blackman 1979, Tomiuk and Wöhrmann 1982, Dixon 1985b).⁹ In other words, the asexual descendants of any parthenogenetic female (and there may be millions) constitute a clone. The abundant polymorphism within parthenogenetic aphid lineages is thus entirely polyphenism (Lees 1961, Dixon 1985b).

Much of aphid polyphenism is clearly conditional. For example, the production of sexual females, which lay winter-hardy eggs, is cued variously by short photoperiods, cool temperatures, and season-related changes in host plants (Dixon 1985a). Winter is predictable.

More relevant to possible stochastic determination of morphs is the production of winged and wingless midseason morphs (alatae and apterae). The relative fitnesses of these morphs depend on the future suitability of the home host plant and the quantity and quality of other host plants, reachable only by flight. Host quality and availability are less easily predicted than is winter. In most cases that have been studied, production of alatae is only to a degree conditional to environment correlates of decreasing home host quality (e.g., crowded conditions and decline in nutritional quality of the host) (Dixon 1985a). An important aspect of the response to these correlates is that rarely is it 100%; the response is ordinarily 20 to 95% alatae (and 80 to 5% apterae). The switch is thus generally between development of one phenotype (apterae) and development of two phenotypes (apterae and alatae) rather than between one phenotype and another.

Shaw's (1970a) studies of the bean aphid (*Aphis fabae*) are representative. When he reared offspring of apterae under the crowded conditions of 200/bean stipule, about 40% developed into alatae (65%, if the mothers had been crowded too). This degree of crowding could be construed to be the approximate threshold for the switch from 100% apterae to 100% alatae (see Fig. 2), except that densities nearly half as great and densities twice as great still produced a mixture of phenotypes (Fig. 3).

Most other studies have yielded similar results. Lees (1967) found that crowding caused most apterous female vetch aphids (*Megoura viciae*) to produce mixtures of alatae and apterae, generally 70 to 95% alatae. Sutherland (1969) reported that crowding pea aphids (*Acyrtosiphon pisum*) resulted in a maximum of 80 to 85% alatae (in green and pink strains, respectively). Lamb and MacKay (1979) tested ca. 500 clones of pea aphids taken from alfalfa fields in southern Ontario and found that crowding increased production of alatae to an average of 66% (but never to 100%). On the other hand, Watt and Dixon (1981) reported 100% alatae when one strain of the English grain aphid (*Sitobion avenae*) was crowded—though another strain of the same species gave rise to few alatae when treated the same way.

The general lack of a 100% response in production of alatae suggests that selection has favored genotypes that produce a mixture of alatae and apterae under conditions signaling a decline of the home host. The proportions of the mix can be influenced by genotype, as indicated by consistent interclone variability in proportions of alate and

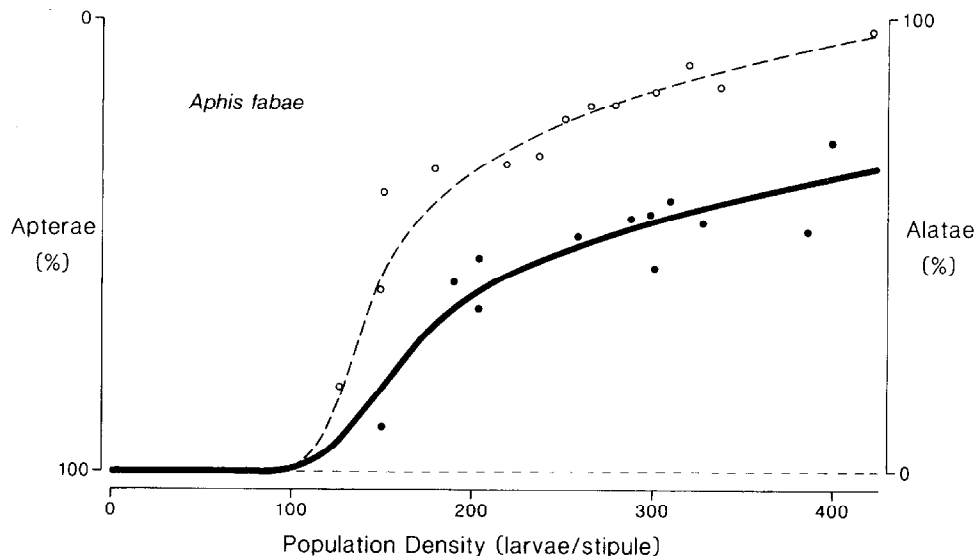


Fig. 3. Response of bean aphids to crowding (data from Shaw 1970a). Filled points and solid line are for the crowded progeny of uncrowded apteræ. Open points and upper dashed line are for crowded progeny of apteræ that were themselves crowded 100-400 per stipule. No alatae are produced at densities below 100 larvae/stipule. The difference between the two data sets shows that the environment of the mother influences the likelihood of her offspring being alatae. (The lower dashed line is for crowded offspring of alatae. Offspring of alatae are never alate, again demonstrating a maternal influence.) (Lines are eye-fitted.)

apterous pea aphids (Lamb and MacKay 1979). Mixed-output genotypes and genetic differences, as well as conditional responses, apparently contribute to wing dimorphism in midseason aphids.

CRICKETS

Dispersal polymorphisms in sexually reproducing insects are common, and they seldom are attributable to simple genetic differences, although the proportions of the morphs can be altered by selection (Harrison 1980). A case in point is wing dimorphism in field crickets (*Gryllus* spp.).

Field crickets are either long-winged or short-winged (Walker and Sivinski 1986). The short-winged morph, like the apterous aphid, never flies. The long-winged morph, characterized by the metathoracic wings extending well beyond the tegmina, has, like an alate aphid, the external equipment for flight. (And again like the alate aphid, it does not necessarily fly—Shaw 1970b; Walker, unpublished data.)

Collections of *Gryllus rubens* the most abundant field cricket in southeastern United States, vary greatly in the proportions of long- and short-winged morphs (Veazey et al. 1976). When progeny of field-collected females are reared under controlled conditions or in outdoor cages, they are generally 5-95% long-winged (Walker, unpublished data). Walker (unpublished data), starting with field-collected stock, selected for long and short wings under controlled temperature and photoperiod ($25 \pm 1^\circ\text{C}$, 16L:8D). The two strains diverged until one was ca. 95% long-winged and the other was ca. 95% short-winged. Even though selection was 100%, in neither strain was the rejected phenotype eliminated in seven generations (Fig. 4). The change in propor-

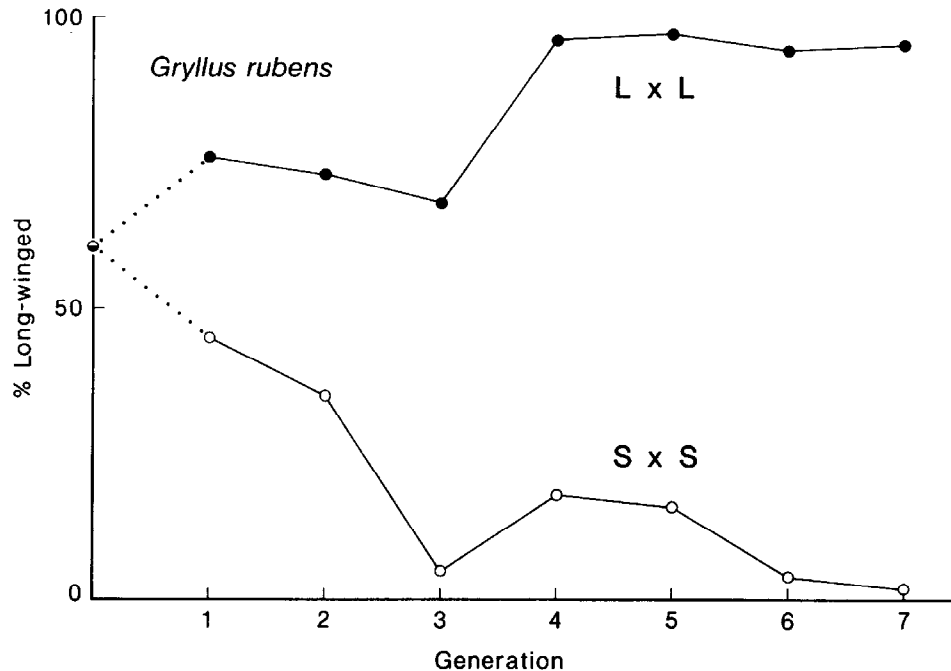


Fig. 4. Results of 100% selection for the long- and short-winged morphs of a field cricket (*Gryllus rubens*) for seven generations. Long- and short-winged parents were collected in Gainesville, Fla., March 1983, and all subsequent rearing and selection was at $25 \pm 1^\circ\text{C}$, 16L:8D. Only long-winged morphs were used as parents in the L x L line and only short-winged morphs were used in the S x S line.

tions of morphs showed that some of the original polymorphism was attributable to genetic differences (as in other *Gryllus*: Harrison 1979, Roff 1984). Conditional polyphenism is also part of the explanation, as shown by a variety of studies of related species by others (see Alexander 1968) and as shown by exposing the F_6 generation of the selected *G. rubens* lines to three rearing conditions (Fig. 5). However, the persistent dimorphism, in the face of selection and under the almost constant environment of a controlled temperature room, suggests that *G. rubens* genotypes have been selected to maintain a mixed output—i.e., that stochastic polyphenism is also involved in the polymorphism.

MOSQUITOES

The pitcher-plant mosquito *Wyeomyia smithii* is dimorphic in its warm season larval development. Some third instars enter diapause and others continue development. Using standardized rearing conditions, including a 15-h photoperiod, C. A. Istock and co-workers studied environmental and genetic determinants of this dimorphism. In one series of experiments, Istock et al. (1975), varied the amount of food per larva. Diapause incidence dropped to as low as 15% with ample food and increased to 100% (among survivors) when food was deficient. In another series of experiments, Istock et al. (1976) maintained conditions that gave 56% diapause in the unselected stock and selected for diapause and nondiapause strains. The course of selection resembled that in Fig. 4 in that progress was irregular and at the end of the study both strains were still producing both phenotypes. After 15 generations of selection for fast, nondiapause development, diapause incidence was 4%. After 7 generations of selection for diapause,

Rearing Conditions	L x L (F6)	S x S (F6)
16L:8D VBC diet 1/cup	G_{LW} <div> <div>98% LW</div> <div>2% SW</div> </div>	G_{SW} <div> <div>6% LW</div> <div>94% SW</div> </div>
16L:8D Cricket Chow 50/jar	G_{LW} <div> <div>89% LW</div> <div>11% SW</div> </div>	G_{SW} <div> <div>3% LW</div> <div>97% SW</div> </div>
11L:13D Cricket Chow 50/jar	G_{LW} <div> <div>47% LW</div> <div>53% SW</div> </div>	G_{SW} <div> <div>1% LW</div> <div>99% SW</div> </div>

Fig. 5. Response of two strains of *Gryllus rubens* to three sets of rearing conditions at $25 \pm 1^\circ\text{C}$. Each strain was in its sixth generation of 100% selection for long or for short wings (see Fig. 4). G_{LW} = genotype(s) of the L x L strain; G_{SW} = genotype(s) of the S x S strain; LW = long-winged phenotype; SW = short-winged phenotype. Rearing conditions differed in photoperiod, diet, type of container, and number of crickets per container. (In the language of Fig. 1, each set of rearing conditions is an "E_s".) (For treatments of L x L, n = 47, 165, and 97; for S x S, n = 63, 170, 86; the proportion of LW from G_{LW} is significantly lower for the short-day treatment than for the two long-day treatments.)

incidence was 88%. In contrast to these results at a 15-h photoperiod, under fall photoperiods (14 h or less) all third instars diapause. Diapause dimorphism in *W. smithii*, like wing dimorphism in *G. rubens*, includes genetic polymorphism and conditional polyphenism as well as what seems to be stochastic polyphenism.

MOTHS

J. G. Sternburg and G. P. Waldbauer (see Waldbauer 1978) found that the silk moth *Hyalophora cecropia* has two modes of emergence in central Illinois. About 10% of the moths emerge in late May (Group I) and the other 90% in late June (Group II). The adult moths are too short-lived to survive the gap between modes, suggesting reproductively isolated populations. However, the majority of the progeny of Group I moths emerge the following year in Group II and a minority of the progeny of Group II emerge in Group I the following year—proving that Group I and Group II emergers belong to the same Mendelian population. Four generations of selection for Group I emergence changed the proportion emerging in Group I to ca. 80% (Waldbauer 1978). The proportion of the wild population that emerged in Group I during the years of selection varied between 4.7% and 10.0%. That genotypic differences are partly responsible for the bimodal emergence is demonstrated by the pronounced increase in proportion of Group I emergers in the selected lineage. That conditional polyphenism is slightly involved is implied by the year to year fluctuation in the proportion of Group I emergers in the wild population (but that fluctuation could also result from changes in genotype frequen-

cies from one year's generation to the next). Finally, the failure of four generations of selection to establish a pure line of Group I emergers shows that mixed emergence is entrenched in *H. cecropia* genotypes, as expected in stochastic polyphenism.

DISCUSSION

If stochastic polyphenism should be a frequent evolutionary response to uncertainty, why might it go generally unrecognized? One reason is that it occurs in combination with other types of polymorphism, and a researcher who proves that one of the other types occurs is likely to emphasize what is explained by accepted principles rather than what is not. A related reason is that chance seems unsatisfactory as a scientific explanation. This misses the point that selection may favor events that are stochastic in effect even though deterministic in means (a flipped coin obeys the laws of physics, but the effect is close enough to 0.50 probability that football games are started with one). A third reason is that in stochastic polyphenism individual fitness is subordinate to fitness at another level. Those who have been trained that selection at the individual level is the only safe way to view evolution may be reluctant to accept as an adaptation a phenomenon that causes some individuals to have reduced fitness for the long term advantage of stochastic genes. However, as Dawkins (1982) has emphasized, selection is as much (or more) the differential survival of genes as the differential reproduction of individuals.

How have those who eschewed stochastic polyphenism explained the type of data used here to support it? Most have ignored the issue. Some have cited maternal influence. A mother aphid that can make her offspring be $y\%$ one phenotype and $(100-y)\%$ another phenotype may, on average, have more grandprogeny than one that produces 100% of what is, on average, the fittest phenotype. Maternal influence seems unlikely when the development of phenotypic differences occurs long after maternal contact has ceased (e.g., wings in field crickets). More to the point, maternal influence is not actually an alternative to stochastic polyphenism. The mother's genotype (rather than the progeny's) simply becomes the one that flips the coin that determines the progeny's phenotype. Furthermore, in aphids, the mother's and progeny's genotypes are generally the same! For sexually reproducing insects, a more credible alternative to stochastic polyphenism is a form of genetic polymorphism—viz., polygenic inheritance with threshold effect (Fig. 6) (see Falconer 1981, Chap. 18).¹⁰ In the case of *Gryllus rubens* or *Wyeomyia smithii*, the production of both phenotypes after 7-15 generations of 100% selection could be attributed to a polygenic complex's normally slow response to selection (Falconer 1981, Chap. 12).¹¹ Finally, frequency dependent selection should be mentioned. Although it is a common cause of genetic polymorphisms (Clarke 1979, Rausher 1986), it cannot account for polymorphism within aphid clones or for the difficulty in artificially selecting for pure breeding strains of long-winged crickets, warm-season diapausing mosquitoes, or early emerging moths.¹²

When consequences of maladaptation are unequal, what should be the effect on the setpoint of stochastic polyphenism? In most of Table 1 the disadvantage of diapause in a nondiapause environment was made equal to the disadvantage of nondiapause in a diapause environment (A, B, C). If the disadvantages are made unequal (as in Table 1, D), stochastic alleles that produce more of the least disadvantaged phenotype achieve higher frequencies.¹³ If one disadvantage is eliminated, then the optimal setpoint becomes 100% of the phenotype that is never disadvantaged—i.e., the polymorphism should cease.

What more general evolutionary principles is stochastic polyphenism a subset of? One is that natural selection does not always favor genotypes that produce higher average numbers of offspring. Gillespie (1977) showed that genotypes with lesser vari-

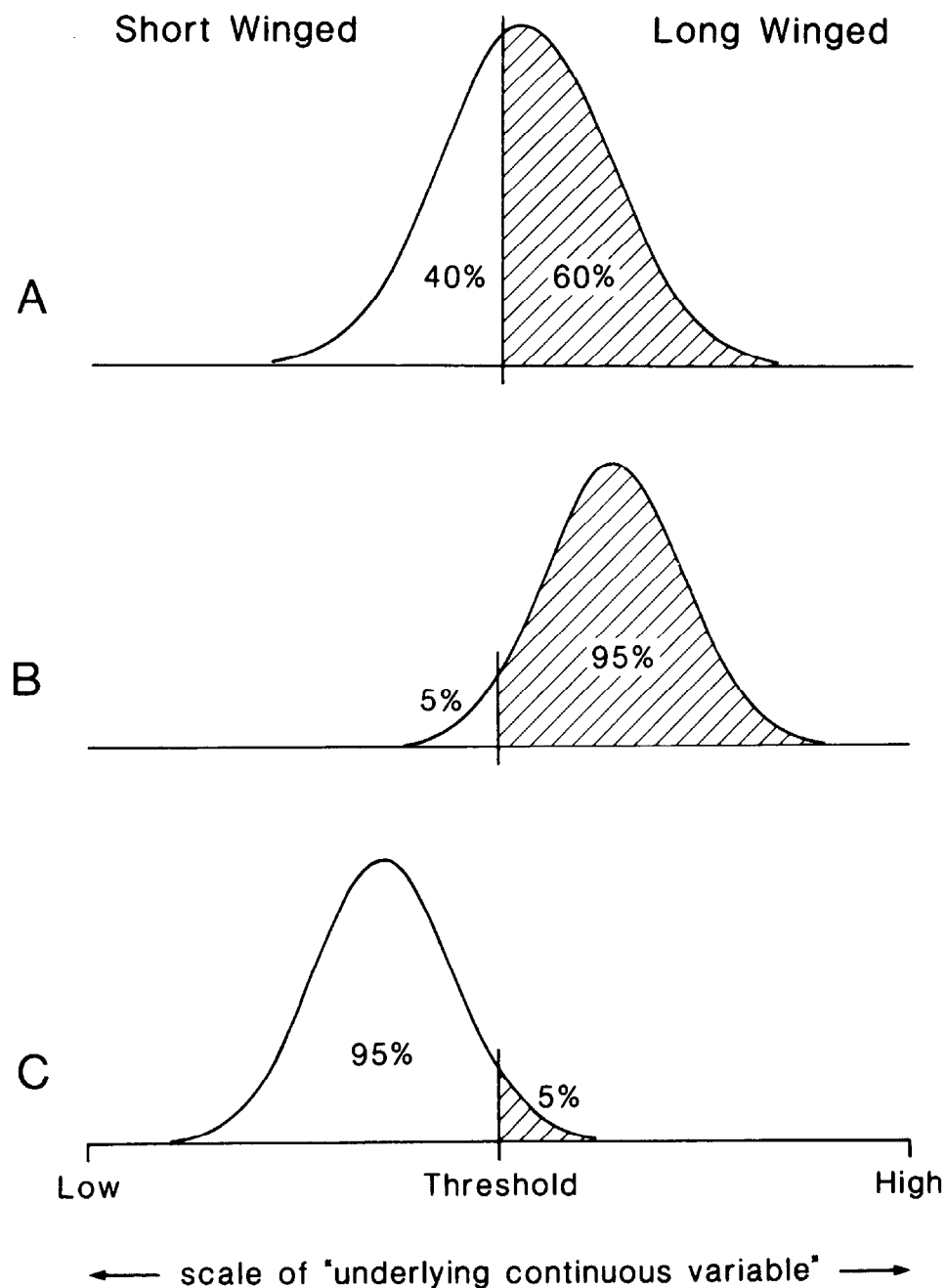


Fig. 6. Diagram illustrating polygenic inheritance with threshold effect as an alternative explanation of wing dimorphism in *G. rubens* (Fig. 4). An underlying continuous variable controls the dimorphism (e.g., the concentration of a wing-promoting substance). This variable is controlled by genes at many loci with additive effects, producing a normal distribution of levels. Some of the loci are linked in opposite phase, prolonging the time required to develop a pure breeding short- or long-winged strain. A. Unselected. B. After 6-8 generations of 100% selection for long-winged morph. C. After 6-8 generations of 100% selection for short-winged morph. (Diagrams modified from Falconer 1981).

ances in offspring numbers can increase at the expense of those with higher average numbers of offspring. In the case of stochastic temporal fluctuations of the environment, he found the best measure of fitness to be the geometric mean of offspring number, averaged over time (the procedure used by Cooper and Kaplan, 1982, in their decision tree analyses). Another principle is that evolution is more easily understood when analyzed at the level of the replicator (Dawkins 1982). As Cooper and Kaplan (1982, p. 145) explained, a strategy-mixing genotype causes some individuals to be stuck with an inferior phenotype—"for the sake of the long term advantage of the genotype."

What terms should be used for the types of polyphenism? Dawkins (1980), writing of evolutionarily stable strategies, distinguished *conditional* from *mixed* or *stochastic* strategies in individual behavior. This distinction aided my theorizing about polyphenism. Cooper and Kaplan (1982; also, Kaplan and Cooper 1984) used *predictive* and *coin-flipping* to name the same distinction and were first to apply it to "adaptive phenotypic plasticity," which includes polyphenism. Both sets of terms are appealing and nearly self-defining. I chose Dawkins' because they are more likely to be familiar to behavioral ecologists. Less this choice be viewed as deemphasizing Cooper and Kaplan's priority in applying the distinction to polyphenism, I end as I began by quoting from their synopsis (1982, p. 135): "The variation is a product of the action of genetically controlled stochastic processes; metaphorically, it is produced because individuals are genetically programmed to 'flip coins' to decide what characteristics to adopt. Thus it is not the variable phenotypic traits themselves that are genetically specified, but only the nature of the coin-flipping process that will ultimately determine them. . . . coin-flipping strategies of this kind are robust and can evolve under a variety of conditions."

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APPENDIX

¹In sexually reproducing animals, natural selection increases genotypes indirectly—by altering frequencies of alleles that in turn alter the probability of particular genotypes forming in the next generation and by altering linkage relationships.

²The effect of a transposable element is that of a mutation and its back mutation being exceedingly frequent, making the occurrence of the phenotypes they produce unpredictable except on a probabilistic basis. That a mobile element can actually control a polymorphism has been demonstrated in the bacterium *Salmonella* (Simon et al. 1980, p. 1370), in which the inversion of a 970-base-pair DNA sequence "behaves like a flip-flop switch activating and inactivating *H2* gene transcription. Depending on the frequency of switching, a fraction of the population expresses one flagellar antigen while the rest of the cells express the other flagellar antigen."

³These models were written in BASICA for an IBM PC. They are menu-driven and suitable for classroom use. I will make copies for anyone sending me two blank diskettes (one of which I will return with the programs).

⁴This was accomplished through the random-number generating function of BASICA.

⁵Phenotypes were assigned individuals in the proportions $y:(100-y)$. For large populations this procedure should not differ in effect from true stochastic assignment with probabilities y and $(100-y)$.

⁶In some instances, circumstances in one growing season foretell circumstances the following year, making possible control of prolonged diapause by conditional polyphenism. For example, a dense population (as in an outbreak) can predict scarce resources or high parasitoid populations for the next year.

⁷The assumptions were: (1) Bad years occur at random. (2) Three alleles control duration of diapause— P_0 , which causes emergence after one winter; P_T emergence after two winters; and P_S , 50% emergence after one winter, 25% emergence after two winters, 12.5% emergence after three winters, etc. up to w winters, with all remaining individuals emerging after the w^{th} winter (e.g., for $w=4$, 12.5% emerged the last winter). (3) The annual mortality caused by remaining in diapause is a constant proportion of survivors. (4) Each year's cohort (i.e., those entering their initial winter of diapause) is numerically independent of the number that had broken diapause earlier that year to produce it but dependent on whether the growing season had been good or bad.

⁸In running the prolonged-diapause model these values were set: dominance relations of alleles, initial frequencies of alleles, maximum number of winters in diapause for individuals controlled by P_S , probability of a bad growing season, number going into diapause after a bad growing season (as a proportion of the number for a good growing season), number of generations per run, number of runs.

⁹Mutation-caused morphs should be easily separable from polyphenism because such morphs would occur rarely and, once present, the responsible allele should be passed to all parthenogenetically produced descendants of the original mutant individual.

¹⁰A way to refute the hypothesis of polygenic inheritance with threshold effect as an alternative explanation (for data that suggest stochastic polyphenism in sexually reproducing insects) is the establishment of homozygous strains through inbreeding. As strains become more and more homozygous through generations of inbreeding, the polymorphism may be maintained if it is stochastic polyphenism and should be reduced and then eliminated if it is dependent on genetic difference. As an example of this approach, Wright and Chase (1936) (see also, Festing 1976) studied white spotting in inbred and outbred lines of guinea pigs and found that in inbred lines the degree of spotting was almost entirely under nongenetic control. Individuals ranging from nearly white to nearly "self colored" (i.e., 5-95% white spotted) occurred in the same litter. In one inbred line the environment controlled 97% of the variance, and less than 10% of this environmental component was attributable to age of mother and other factors common to litter mates—89% of the variance was random. Even in an outbred line, 52% of the variance in degree of spotting was random.

¹¹One of the causes of slow response is linkage of polygenes of opposite sign. If such linkage is tight, the group of linked loci can act as a "supergene" for stochastic polyphenism—by keeping the level of the "underlying continuous variable" close enough to the threshold of response for individuals of both phenotypes to occur.

¹²Frequency dependent selection occurs, by definition, whenever fitness of a genotype is an inverse function of its frequency. Wing dimorphism in crickets is partly a result of genetic differences among individuals, and the frequency of alleles for long- and short-winged morphs may depend in part on long-winged and short-winged morphs becoming more fit on average as they (respectively) become rarer. On the other hand the relative success of long- and short-winged morphs must depend in part on uncertain meteorological events. Frequency dependent selection may occur as a result of unpredictable environmental changes but it is not an adaptation to them.

¹³Cooper and Kaplan (1982) derived this formula for calculating the optimal setpoint for a stochastic genotype:

$$q_0 = \frac{\lambda_{1,0}}{\lambda_{1,0} - \lambda_{0,0}} P_1 + \frac{\lambda_{1,1}}{\lambda_{1,1} - \lambda_{0,1}} P_0$$

where q_0 = proportion of phenotype 0

$\lambda_{1,0}$ = finite rate of increase, in environment favoring phenotype 1, of phenotype 0
 $\lambda_{0,0}$ = finite rate of increase, in environment favoring phenotype 0, of phenotype 0
 P_1 = probability of environment favoring phenotype 1
 etc.

If $0 < q_0 < 1$, then a mixed strategy is superior to either pure strategy and the optimal proportions of phenotypes 0 and 1 are q_0 and $1 - q_0$.

Applying this formula to the circumstances in Table 1,C, yields an optimal y of 0.704; to the circumstances in Table 1,D, an optimal y of 0.995.

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