Chapter 2

Elements of a Quantitative Genetic Model of Life History Evolution

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Introduction

Much of the theory of life history evolution is based on optimization models that attempt to predict the equilibrium state(s) of a population by maximizing its growth rate, subject to ad hoc constraints and trade-offs between individual growth, reproduction, and survival (Stearns 1977). However, the types of evolutionary processes that produce life histories that are optimal in some sense can only be determined from genetic models of selection in age-structured populations (Charlesworth 1980). For example, frequency-dependent selection can produce maladaptive evolution, decreasing the mean fitness in a population (Wright 1969, Chapter 5). Despite their greater complexity, genetic models incorporating evolutionary constraints in measurable patterns of genetic variation and natural selection have the advantage of providing a dynamic rather than a static description of evolution and provide a framework for quantitative testing of hypotheses about constraints on life history evolution.

Two fundamental requirements for a robust science of life history evolution are (1) experimental methods of measuring heritable variation in life history characters within populations, and (2) a dynamical theory describing how this variation evolves. Estimates of genetic variance and covariance in quantitative characters can be obtained from phenotypic correlations between relatives or from artificial selection experiments (Falconer 1960, Kempthorne 1957, Mode and Robinson 1959). The same methods can be extended to life history characters measured at specific ages. Using these techniques, Rose and Charlesworth (1980, 1981a, b) estimated strong negative genetic correlations of early fecundity in females with late fecundity and life span but were unable to detect any increase with age in additive or dominance genetic variance for age-specific fecundity. Their findings support the hypothesis that senescence is caused by genes with beneficial effects early in life and deleterious effects in old age (Williams 1957) and do not support an alternative hypothesis that senescence is largely a result

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of mutations that have only detrimental effects, expressed late in life (Hamilton 1966). Data of Sacher and Duffy (1979) indicate that longevity in male mice is determined primarily by metabolic and morphological characters with an intermediate optimum, rather than deleterious genes expressed only in old age, suggesting that most of the genetic variance in longevity is nonadditive (see Wright 1935).

Although experimental techniques for measuring genetic constraints on life history evolution already exist, at present there is no dynamical theory of natural selection on quantitative characters in age-structured populations. Some major questions that such a theory should address are: (1) What is the evolutionary significance of genetic variance and covariance in life history traits? (2) Is there a general principle of adaptation for correlated characters in age-structured populations? (3) Can information on genetic variance and covariance of life history characters be used to draw inferences about the forces of natural selection affecting them?

This chapter outlines some elements of a dynamic theory of life history evolution by combining standard models of quantitative genetics and demography for populations with discrete, overlapping generations. Further discussion of these and other topics, using demographic models with continuous time, can be found in Lande (1982).

Elements of a Dynamic Theory

For simplicity in describing the evolutionary influence of overlapping generations, the following models concern populations that are monoecious, or that have identical selection pressure on both sexes and no sexual dimorphism. Consider a vector of quantitative characters of an individual,

$$ z = \begin{pmatrix} z_1 \\ \vdots \\ z_n \end{pmatrix} $$

(2.1)

some or all of which may be age specific. As frequently can be arranged by a simple scale transformation, most often logarithmic, the joint distribution of the characters in a population of unselected individuals is assumed to be multivariate normal (Gaussian), with mean $\bar{z}$ and phenotypic variance-covariance matrix $\mathbf{P}$. Observations on phenotypic correlations between relatives, or artificial selection experiments, can be employed to partition $\mathbf{P}$ into additive genetic and environmental (plus nonadditive genetic) components. If there are no genotype-environment correlations the additive genetic variance-covariance matrix, $\mathbf{G}$, and the environmental variance-covariance matrix, $\mathbf{E}$, sum to

$$ \mathbf{P} = \mathbf{G} + \mathbf{E} $$

(2.2)

It is also assumed that the distribution of additive genetic effects (breeding values) for the characters is multivariate normal, as will tend to apply for polygenic characters with a Gaussian phenotype distribution.