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Models of Multifactorial Inheritance: I, Multivariate Formulations and Basic Convergence Results*

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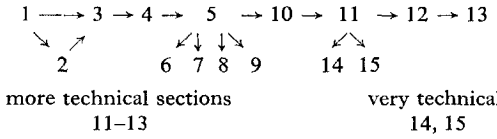
A multivariate dynamic model of a vector phenotype trait under the influence of a selective mating pattern, a hierarchy of parental–offspring transmission rules, and random environmental effects is developed. The formulation can incorporate age class effects, geographical variation, asymmetric maternal and paternal contributions, sex-differentiated offspring expression, selective family adoption procedures, and classes of family and pedigree influences. The mating, transmissible, and environmental aspects of family structure parameters can vary systematically or randomly in time. Cultural and biological variables are handled in one framework. Results on the dynamic and equilibrium behavior of these multivariate processes are set forth and interpreted.

Contents.[1] 1, Introduction and Perspectives; 2, The Principal Components of the General Dynamic Phenotypic Model; 3, The Model Involving Linear Transmission–Segregation and Environmental–Mutational Forces; 4, The Multivariate Gaussian Model of Phenotype Inheritance; 5, A Vector Phenotype Model for a Two-Sex Population Subject to Nonlinear Assortative Mating Pattern; 6, A Phenotypic Nuclear Family Vector Trait Model with Selective Assortative Mating; 7, The Multivariate Gaussian Phenotype Model with Two Age Classes; 8, A Phenotype Selection Model for Adopted Children; 9, Evolution of a Quantitative Trait under Continual Sib-mating; 10, Summary and Discussion; 11, The Principal Convergence Theorems; 12, The Deviation of the Mean Population Phenotype from the "Optimal" Phenotype; 13, The Vector Phenotypic Model with Genetic and Environmental Parameters Varying Systematically or Randomly in Time; 14, Appendix A: The Monotonicity and Concavity of $f(\Sigma)$ Defined in (11.2) and Proof of Result I of Section 11; 15, Appendix B: Discussion of Result II of Section 11.

Dependence relations of the various sections in reading this paper:



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[1] After reading Section 1 and cursorily Section 10 of this work some readers may find it more desirable to go directly to Paper II, which is more specific, before returning to this extensive paper.

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1. Introduction and Perspectives

This is part of a series of papers devoted to the formulation and analysis of phenotypic (quantitative) inheritance.[2] The first concentrates on the classifications and characterizations of the genetic and environmental structures. The multivariate framework provides wide scope and versatility for handling asymmetric (sex-dependent) attributes in parental and sibling relationships, the nonlinear selective processes innate to assortative mating patterns, natural selection forces, the selection embedded in adoption practices, demographic, migration, and population structure facets, and models where the unit of observation is a nuclear family or a multigenerational family set. The basic convergence results for changes in the population mean and covariance over time are set forth in this work.

Papers II and IV present detailed studies of the equilibrium covariance structure, discerning its dependence on the natural selection, assortative mating, transmission, and environmental parameters for a two-sex population model of a scalar trait. In the third paper of this series a hierarchy of kinship covariance calculations is elaborated within the framework of an extended selective mating mechanism. These include covariances involving parent, offspring, siblings, half-sibs, cousins, or more distant family relatives, and of adopted and natural children. Paper V presents contrasts and comparisons of our selective mating constructs with the linear assortative mating models which have been extensively pursued by many authors. The sixth paper elaborates finer properties of the dynamic and equilibrium behavior on vector multifactorial inheritance. Papers VII and VIII investigate some variants of these models in a non-Gaussian setting. We also discuss aspects of cultural selection in several of these works. In the present series (I–VIII), we emphasize qualitative inferences and comparisons, while in subsequent works we plan to provide simulation, numerical, and graphical addenda to the theory developed here.

A "polygenic" character refers to a trait determined by many loci (genes) contributing mostly small effects and commonly manifesting a continuous variation in the trait expression. The concept of a polygenic model is not clear-cut by its very nature. Many genes interact intrinsically and are coupled to environmental stimuli in complex ways. The classical model traces the changes over successive generations of the phenotype frequency distribution as influenced by selection effects, mutation and migration forces, and transmission laws. This model includes mixed mating patterns, varying forms of parent–offspring correlations, and a myriad of environmental factors. The analysis concentrates on changes in phenotypic representations which do not properly account for genotype–phenotype associations and the basic genetic complex of multilocus

[2] For the sake of clarity, throughout this article, subsequent papers in this series are referred to as Karlin (II) through Karlin (VIII), denoting the references, Karlin (1979d) through (1979i), respectively.

interactions. Previous attempts to incorporate the genetic mechanisms have implicitly relied on assumptions of additive gene contributions and global linkage equilibrium which are basically inconsistent with the operation of differential selection effects and/or the existence of various nonrandom mating patterns.

Quantitative inheritance has had its primary stimulus in problems of animal and plant breeding (e.g., Falconer, 1960; Mather and Jinks, 1971). The theory, with some notable exceptions, is largely statistical rather than evolutionary (e.g., Kempthorne, 1957; Wright, 1921). In contradistinction, Robertson (1960), Hill (1970), and Latter (1965, 1972), among others, have studied a few evolutionary problems in the animal breeding framework. Kimura (1965), Slatkin (1970, 1978), Lande (1976a, b), Slatkin and Lande (1976), Cavalli–Sforza and Feldman (1976, 1977), Fleming (1979), Felsenstein (1977), Eshel (1971, 1972, 1973), and Roughgarden (1972), among others, have introduced some aspects of population dynamics into polygenic and/or phenotypic inheritance in other ways.

Intensive activity over the past decade has involved Genetic epidemiologists seeking to discern genetic, environmental, and cultural components which are relevant in assessing risk factors for (common) diseases. The analyses and modeling are based on biochemical, physiological, environmental, and cultural measurements collected on individuals, families, and pedigrees of various structures. For references and representative papers, see the recent conference volumes edited by Morton and Chung (1978) and Sing and Skolnick (1979). The statistical methodology is usually linear covariance analysis founded on linear models of mating and polygenic-Galtonian transmission. Numerous authors of the Birmingham school (Eaves, Jinks, and collaborators), the Virginia group (Nance and colleagues), the Honolulu center (Morton, Rao, *et al.*), the St Lous contingent (Cloninger, Reich, *et al.*), Jencks, Goldberger, Conlisk, Loehlin, and a number of other social scientists, Cavalli–Sforza and Feldman at Stanford, among others (see Feldman and Cavalli–Sforza (1979) for a review and references), motivated primarily by problems of mixed cultural-phenotype–"genetic" transmission have studied a variety of dynamic and/or stationary equation models, primarily in the guise of linear path analysis or by means of linear variance decompositions. Most formulations tend to mimic one-locus theory, where the genetic mechanism is expressed by the within family variance based on additive allelic and independent loci effects. The accommodation of assortative mating is usually done, following Fisher (1918), by postulating a time invariant spouse correlation (or set of correlations) and other stationarity assumptions. These formulations often lead to infinities and indeterminancies of the variance terms. A critique of their procedures, dissecting a number of hidden and manifest assumptions, is contained in Karlin (III) and (V).

Our approach analyzes phenotypic variation in an intrinsic multivariate framework. We consider a large population characterized by a vector $\mathbf{x} =$

$(x_1, x_2, \ldots, x_n)$ of phenotypic traits (generally correlated) whose (multivariate) frequency distribution changes over time as influenced by selection (natural and/or sexual), mutation, and migration forces, mating patterns, and various forms of parent–offspring transmission-segregation structures. The study of the vector version provides much flexibility to the formulation, as will be amply illustrated.

The phenotype component variables may include continuous variables, counts, categorical observables, qualitative measurements, fractions of some fixed quantity, and variables on restricted ranges. Unless stated otherwise, it is understood that proper scalings and standardizations have been invoked, converting all components of $\mathbf{x} = (x_1, x_2, \ldots, x_n)$ to the same units with secular variables regressed out, if necessary. (The standardization problem generally presents a sensitive and formidable task.)

Component variables may include such diverse traits as $\mathbf{x} = (x_1, x_2, x_3) =$ (egg number per unit time, average egg size, texture of albumin); $\mathbf{x} =$ (mathematical aptitude at a specified age, a measure of physical coordination, some metabolic rate/time, wealth, education); $\mathbf{x} =$ (flowering time, average number of fruits/plant, an appropriate fiber strength, degree of resistance to a specified disease); $\mathbf{x} =$ (insulin production/time, glucose absorption rate, nutrition scale, blood pressure scorings), $\mathbf{x} =$ (cholesterol level, triglyceride level, weight, personality type, scale of smoking, a set of blood types).

Geographical distribution of a phenotypic variable can be encompassed in the array $\mathbf{x} = (x_1, x_2, \ldots, x_n)$ where x_i is the trait (or vector trait) observation at the ith locality, such that $\mathbf{x}$ describes a group of indivduals—one from each locality.

The effects of age structure can be incorporated by increasing the number of components for $\mathbf{x} = (x_1, x_2, \ldots, x_L)$ where x_i represents the frequency of the phenotype single or multivariate trait for the ith age grouping so that a trait manifested in different age classes is considered a set of different traits.

The mixed "genetic"-cultural transmission models are based on vector trait variables which partition into three sets.

$$\mathbf{x} = (x_1, x_2, \ldots, x_k; y_1, y_2, \ldots, y_l; z_1, z_2, \ldots, z_m), \tag{1.1}$$

where the $\{x_\nu\}$ variables refer to "genotype" components, the $\{y_\mu\}$ are associated phenotype variables, and $\{z_\lambda\}$ are related environmental and/or cultural variables, e.g., wealth, schooling, customs. The distinction between genotype and phenotype variables in this setting is unclear. An appeal to one-locus theory as a guide for separating these classes of variables is questionable. The attempt is made to reflect the genetics by adding an unobservable random term to the inheritance process, conveying a variance equal to one-half the phenotype variance intended to reflect the within family variance contribution as "Mendelian segregation" (e.g., Cavalli–Sforza and Feldman, 1976, 1977). However, this tactic is limited to the case of additive allelic effects at one locus, without

dominance, without epistasis, disallowing deviation from pure random mating, and other constraints.

In a *model with separate sexes* we double the dimension of the vector trait in the manner

$$\mathbf{z} = (\mathbf{x}, \mathbf{y}) = (x_1, \ldots, x_n; y_1, \ldots, y_n), \tag{1.2}$$

where $\mathbf{x}$ refers to the male and $\mathbf{y}$ corresponds to the female components.

In simple qualitative terms, the dynamics of the population phenotype model involves two major stages, the mating (couple formation) process and the parent–offspring transmission structure. In our approach, a male and female $\mathbf{x}$ and $\mathbf{y}$ pairing are joined by a preference (selection) process which is intrinsically nonlinear. For an established parental couple $(\tilde{\mathbf{x}}, \tilde{\mathbf{y}})$ a male offspring acquires a phenotype value of the form

$$\mathbf{x}' = \mathbf{R}(\tilde{\mathbf{x}}, \tilde{\mathbf{y}}) + \boldsymbol{\epsilon}^{(\mathrm{m})}, \tag{1.3}$$

where $\mathbf{R}$ is a transformation (not necessarily linear) of the parental values and $\boldsymbol{\epsilon}$ conveys a residual (random-environmental) contribution independent of the parental phenotypes. For a female offspring, the analog of (1.3) is

$$\mathbf{y}' = \mathbf{S}(\tilde{\mathbf{x}}, \tilde{\mathbf{y}}) + \boldsymbol{\epsilon}^{(\mathrm{f})}, \tag{1.4}$$

where $\mathbf{S}$ may differ from $\mathbf{R}$, indicating sex-dependent transmission. The residual variable correlations among siblings may depend on their sexes. The standard version takes the vector functions $\mathbf{R}$ and $\mathbf{S}$ as linear transformations operating on the parental phenotypes $\tilde{\mathbf{x}}$ and $\tilde{\mathbf{y}}$.

The vector framework enables us to consider a phenotype trait with reference to a specified family set; for example, a nuclear family consisting of parents and a number of offspring, or even more elaborate pedigree conglomerates. Thus, a nuclear family with two children of both sexes may be represented by a $4n$ component trait:

$$\mathbf{f} = (\tilde{\mathbf{x}}, \tilde{\mathbf{y}}, \mathbf{c}^{(\mathrm{m})}, \mathbf{c}^{(\mathrm{f})}), \tag{1.5}$$

where $\tilde{\mathbf{x}}$ and $\tilde{\mathbf{y}}$ refer to the parental spouse types and $\mathbf{c}^{(\mathrm{m})}$ and $\mathbf{c}^{(\mathrm{f})}$ to the male and female child vector phenotypes. We elaborate this setup in Section 6.

It is germane to emphasize that the vector phenotype treatment integrates simultaneously anthropometric, physiological, behavioral, biochemical, and/or cultural variables in one framework. The transmission functions $\mathbf{R}$ and $\mathbf{S}$ may operate on all or partial sets of such components. Our models treat phenotypic and cultural variables together as generally correlated traits. The difference in transmission, offspring expressivity, influences of mating pattern, etc., are subsumed in the versatility of the transformation relations that delimit the

model. The general impreciseness of parameter estimates from natural data in conjunction with sampling fluctuations and innate individual and population heterogeneity dictate that we project mostly the qualitative conclusions of the analysis rather than rely on its predictive value (cf. Karlin, 1979b).

In this first paper our primary objective is to establish a broad phenotype model that properly accounts for selection forces, especially assortative mating as a phenomenon of a selective process coupled to a hierarchy of segregation-transmission rules, especially sex-dependent forms, and interactions among collaterals and cohorts. Various results on the dynamic and equilibrium behavior of the covariance structure for the population phenotype trait are presented in Sections 10 through 14.

In the subsequent papers we address inter alia the following issues. How does assortative mating structure affect the parent–offspring covariances compared to sib–sib covariances, e.g., in terms of the strength and concordance parameters between mates? What is the dependence of these covariances on the parental transmission characteristics, especially the influence of asymmetric maternal and paternal contrasts and sex-dependent offspring expression? What is the nature of second- and higher-order kinship covariances in this new framework? Further questions will be amplified in those works.

2. The Principal Components of the General Dynamic Phenotypic Model

The possible types $\mathbf{x}$ of the population are identified with points in E^n (Euclidean n-space). In each generation the population, assumed to be of large size, is described by its frequency measure over E^n. For any set A of E^n, let $\mu_t(A)$ be the proportion in the population at an appropriate census time of generation t consisting of vector types corresponding to A. For ease of exposition, we assume henceforth the existence of the density $p_t(\mathbf{x})$ so that

$$\mu_t(A) = \int_A p_t(\mathbf{x})\, d\mathbf{x}. \tag{2.1}$$

The population composition changes over successive generations under the influence of selection and mutation forces, the segregation mechanisms, and the nature of the population structure and mating pattern. For definiteness, we census the population in each generation at the juvenile stage and stipulate the order of forces to be

Model I

$$\text{natural and/or mating selection} \rightarrow \text{transmission-segregation} \rightarrow \text{environmental-mutation}. \tag{2.2}$$

A second arrangement has the sequence of forces

Model II

$$\text{transmission-segregation} \rightarrow \text{environmental-mutation} \rightarrow \text{selection}, \quad (2.3)$$

and finally, we can consider the sequence of effects to be

Model III

$$\text{environmental-mutation} \rightarrow \text{transmission-segregation} \rightarrow \text{selection}. \quad (2.4)$$

The models for (2.2) and (2.3) are equivalent and merely reflect a different census time in a generation period where the individuals according to (2.3) are sampled at the adult stage, i.e., just prior to mating. The qualitative results in all three models are the same. For definiteness of exposition, we concentrate in this paper on the order of forces of (2.2), Model I.

It is increasingly recognized that assortative mating and mate selection mechanisms and other nonrandom mating operations, e.g., consanguinity, incompatibilities, imprinting, and regular inbreeding schemes, generally act differentially with respect to phenotypic expression; e.g., see O'Donald (1977), Matessi and Scudo (1975), and Karlin (1978b).

A. *Action of Viability Selection*

We consider first forms of viability selection. Later, in the framework of the two-sex model, we emphasize selection effects by way of an assortative mating mechanism based on a mating selection function. In the same vein, we regard the child adoption events as a selection process in Section 8. In this perspective, effects of assortative mating and adoption resemble "fertility" selection differentials relative to mating types which operate nonlinearly over the population system. Actually, the action of viability selection and the operation of couple formations in the two-sex case essentially reduce to the same model. More details on these concepts and their applications are covered in Sections 5 through 8.

The relative fitness of a zygotic type $\mathbf{x}$ compared to that of $\mathbf{y}$ at generation t is $\gamma_t(\mathbf{x})/\gamma_t(\mathbf{y})$. We refer to $\gamma_t(\mathbf{x})$ as the *fitness function*, also variously called the *relative survival function* and *relative viability function*. A realistic version of the influence of selection would have $\gamma_t(\mathbf{x})$ varying stochastically in time and also frequency-dependent, meaning that $\gamma_t(\cdot)$ in generation t could be a functional also of the density $p_t(\cdot)$. The relative number of surviving individuals of type $\mathbf{x}$ due to selection is $\gamma_t(\mathbf{x})\, p_t(\mathbf{x})$. Thus, after selection the density of the population composition in generation t is

$$\tilde{p}_t(\mathbf{x}) = \frac{\gamma_t(\mathbf{x})\, p_t(\mathbf{x})}{\int \gamma_t(\xi)\, p_t(\xi)\, d\xi}. \quad (2.5)$$

To avoid technical complexities and other confounding effects, we mostly assume $\gamma_t(\mathbf{x})$ independent of t.

The nature of $\gamma(\mathbf{x})$ will usually be one of three kinds.

(i) *Directional selection.* ($\gamma(\mathbf{x})$ is monotone increasing in some direction.) Here a type is more advantageous if associated with "larger values." There are three subclasses of special interest:

(a) $\gamma(\mathbf{x}) = e^{\langle \boldsymbol{\lambda}, \mathbf{x} \rangle}$ (λ_i are generally positive constants and $\langle \boldsymbol{\lambda}, \mathbf{x} \rangle = \sum_{i=1}^{n} x_i \lambda_i$ denotes the scalar or inner product of the indicated vectors). In artificial selection schemes $\sum_{i=1}^{n} \lambda_i x_i$ may be a generalized phenotypic index and selection is for high values of this index.

(b) Suppose $E = \mathbb{R}^+$, the positive real line, i.e., $\mathbf{x}$ is scalar, and $\gamma(\mathbf{x}) = x^\alpha$ so that $\gamma(x)$ grows at infinity at an algebraic rate. In this case,

$$\lim_{x \to \infty} \frac{\gamma(x+1)}{\gamma(x)} = 1$$

and the dynamic process is significantly different compared to the exponential fitness function of case (a).

(c) $\gamma(\mathbf{x})$ is increasing from 0 to 1. One interpretation for $\gamma(x)$ is that of a survival probability. Artificial selection programs often specify $\gamma(\mathbf{x})$ of the form $\gamma(x) = 1$, $x \geqslant \gamma_0$; $\gamma(x) = 0$, $x < \gamma_0$, corresponding to truncation selection. In this form, $\gamma(\mathbf{x})$ can also serve as a liability function of a disease trait.

(ii) *Stabilizing or optimizing selection (meaning that $\gamma(\mathbf{x})$ achieves one or more maxima for some intermediate value).* A leading case is

$$\gamma(\mathbf{x}) = \exp[-\tfrac{1}{2}\langle (\mathbf{x} - \boldsymbol{\lambda}), \mathbf{C}(\mathbf{x} - \boldsymbol{\lambda}) \rangle], \tag{2.6}$$

where $\mathbf{C}$ is a positive semidefinite matrix so that $\boldsymbol{\lambda}$ is the optimum fitness type. Another common specification is

$$\gamma(\mathbf{x}) = \begin{cases} 1 & \text{for } \mathbf{x} \text{ in a specified set } S, \\ 0 & \text{otherwise.} \end{cases} \tag{2.7}$$

(iii) *Disruptive or diversifying selection.* Here $\gamma(\mathbf{x})$ achieves a maximum at a number of extremes of the $\mathbf{x}$ range. The case of disruptive selection may be of significance in explicating some cases of speciation.

B. *Parent–Offspring Segregation–Transmission Structure*

Random mating will formally connote that the frequency of the union of an $\mathbf{x}$- and $\mathbf{y}$-type individual is

$$\tilde{p}_t(\mathbf{x})\, \tilde{p}_t(\mathbf{y})\, d\mathbf{x}\, d\mathbf{y}, \tag{2.8}$$

where $\tilde{p}$ is defined in (2.5). A key element in the model is the prescription of the

conditional probability segregation density function $L_t(\mathbf{u}; \mathbf{x}, \mathbf{y})$ which is equal to the probability that the offspring is of type $\mathbf{u}$ given that the parental types are $\mathbf{x}$ and $\mathbf{y}$. Assuming random mating, the density of types in the next generation as a result of segregation-transmission is calculated by the formula

$$p^*_{t+1}(\mathbf{u}) = \iint L_t(\mathbf{u}; \mathbf{x}, \mathbf{y})\, \tilde{p}_t(\mathbf{x})\, \tilde{p}_t(\mathbf{y})\, d\mathbf{x}\, d\mathbf{y}. \tag{2.9}$$

A more general mating scheme than random mating can be incorporated into the model. For example, a case of assortative mating is generated by specifying in generation t, for each $\mathbf{x}$ type, the conditional probability density $\phi_t(\mathbf{y} \mid \mathbf{x})\, d\mathbf{y}$ of the pairing $\mathbf{x}$ and $\mathbf{y}$. The segregation-transmission (2.9) is then adjusted to the form

$$p^*_{t+1}(\mathbf{u}) = \iint L_t(\mathbf{u}; \mathbf{x}, \mathbf{y})\, \phi_t(\mathbf{y} \mid \mathbf{x})\, \tilde{p}_t(\mathbf{x})\, d\mathbf{x}\, d\mathbf{y}. \tag{2.10}$$

The setup of (2.10) is asymmetric in the mating process. If $\phi_t(\mathbf{y} \mid \mathbf{x})\, d\mathbf{y}$ is a degenerate density concentrating at $\mathbf{x}$, then a case of complete assortative mating is operating.

A formulation which distinguishes the sexes refers to a phenotypic vector pair (1.2). Accordingly, consider for generation t the male and female adult phenotype densities at generation t, $\tilde{p}_t(\mathbf{x})$ and $\tilde{q}_t(\mathbf{y})$, respectively. Suppose that all individuals have, a priori, equal probability of meeting mates, so the frequency of an encounter of a male $\mathbf{x}$ phenotype and female $\mathbf{y}$ phenotype is $\tilde{p}_t(\mathbf{x})\, \tilde{q}_t(\mathbf{y})$. Let $\phi(\mathbf{x}, \mathbf{y})$ be the *relative conditional probability* that an encounter between a female type $\mathbf{x}$ and male type $\mathbf{y}$ establishes the couple. Accordingly, the joint density function of successful pairings is

$$\tilde{p}_t(\mathbf{z}) = \tilde{p}_t(\mathbf{x}, \mathbf{y}) = \frac{\tilde{p}_t(\mathbf{x})\, \tilde{q}_t(\mathbf{y})\, \phi(\mathbf{x}, \mathbf{y})}{\iint \tilde{p}_t(\xi)\, \tilde{q}_t(\eta)\, \phi(\xi, \eta)\, d\xi\, d\eta}, \qquad \mathbf{z} = (\mathbf{x}, \mathbf{y}). \tag{2.11}$$

Random mating corresponds to $\phi(\mathbf{x}, \mathbf{y}) \equiv 1$. The transformation (2.11) embraces a range of mating preference schemes. *We refer to* $\phi(\mathbf{x}, \mathbf{y})$ *as the preference mating function or selection mating function.* It acts in the manner of fertility selection on the mating types. We discuss several natural specifications of $\phi(\mathbf{x}, \mathbf{y})$ in Section 5.

The transmission-segregation rule in this framework is characterized by a pair of conditional densities $M_t(\xi; \mathbf{z})$ and $F_t(\eta; \mathbf{z})$, where ξ is the trait value of a male progeny following the conditional distribution law of M_t and η is the trait value of a female progeny following the conditional distribution law of F_t produced by the parental composition $\tilde{\mathbf{z}} = \{\tilde{\mathbf{x}}, \tilde{\mathbf{y}}\}$. The distribution governing the progeny types in this scheme is

$$\begin{aligned} p^{*(\mathrm{m})}_{t+1}(\xi) &= \int M_t(\xi; \mathbf{z})\, \tilde{p}_t(\mathbf{z})\, d\mathbf{z} \qquad \text{(male progeny)}, \\ p^{*(\mathrm{f})}_{t+1}(\eta) &= \int F_t(\eta; \mathbf{z})\, \tilde{p}_t(\mathbf{z})\, d\mathbf{z} \qquad \text{(female progeny)}. \end{aligned} \tag{2.12}$$

We elaborate these transmission-segregation models in the Gaussian multivariate framework in Section 5.

C. *Mutation-Environmental Pertubations*

Let $g_t(\mathbf{v}; \mathbf{u})$ be the conditional probability density that a $\mathbf{u}$ offspring mutates or environmental pertubations change it to that of a $\mathbf{v}$ type. The transformation of phenotypic frequencies due to these factors after segregation is given by

$$p_{t+1}(\mathbf{u}) = \int g_t(\mathbf{u}; \mathbf{v})\, p^*_{t+1}(\mathbf{v})\, d\mathbf{v}. \tag{2.13}$$

Referring to (2.9) or (2.12) it is actually possible to capture the effects of the mutation-environmental perturbations directly as part of the segregation distribution where, indeed, we define the new segregation distribution

$$\tilde{L}_t(\mathbf{u}; \mathbf{x}, \mathbf{y}) = \int g_t(\mathbf{u}; \boldsymbol{\xi})\, L_t(\boldsymbol{\xi}; \mathbf{x}, \mathbf{y})\, d\boldsymbol{\xi}. \tag{2.14}$$

In tracking the phenotypic distributions over time, it is useful to associate formally a vector random variable with the various stages in each generation.

Let $\mathbf{X}_t$ be a vector random variable following the density $p_t(\mathbf{x})$. (2.15)

Let $\tilde{\mathbf{X}}_t$ be a vector random variable in generation t following the density $\tilde{p}_t(\mathbf{x})$. (2.16)

Let $\mathbf{X}^*_{t+1}$ be a vector random variable in generation $t + 1$ following the density $p^*_{t+1}(\mathbf{x})$. (2.17)

The transformations corresponding to the action of selection, transmission-segregation, and mutation can be succinctly described by the diagram

$$\mathbf{X}_t \xrightarrow[\text{and mating}]{\text{selection}} \tilde{\mathbf{X}}_t\,,\ \tilde{\mathbf{X}}_t \xrightarrow{\text{transmission}} \mathbf{X}^*_{t+1}\,,\ \mathbf{X}^*_{t+1} \xrightarrow[\text{environment}]{\text{mutation-}} \mathbf{X}_{t+1}\,. \tag{2.18}$$

The sequence of densities $\{p_t(\mathbf{x})\}$ is referred to as the phenotype dynamic process. The elements of the sequence $\{p_t(\mathbf{x})\}$ are recursively determined from knowledge of $p_0(\mathbf{x})$ (the initial population frequency density of the types), $\gamma_t(\mathbf{x})$ (action of the viability fitness function of (2.5)), $\phi_t(\mathbf{x}, \mathbf{y})$ (the conditional preference mating function of (2.11)), $L_t(\mathbf{u}; \mathbf{x}, \mathbf{y})$ (the transmission and segregation mechanisms), and $g_t(\mathbf{u}; \mathbf{v})$ (the conditional environmental-mutation density).

The structure and properties of the phenotype dynamic process $\{p_t(\mathbf{x})\}$ is the matter under investigation. Specifically, we are interested in discerning the mean, variance, and other properties of the frequencies of the types evolving over time and the limit behavior of $p_t(\cdot)$ evaluated in terms of the fitness, mating pattern, transmission, and environmental parameters.

3. The Model Involving Linear Transmission–Segregation and Environmental–Mutational Forces

We shall assume that the relative fitness function $\gamma_t(\mathbf{x}) = \gamma(\mathbf{x})$ does not vary in time as a first approximation although results can be developed with γ_t varying systematically or randomly in time (cf. Section 13).

We concentrate on the temporally homogeneous case,

$$g_t(\mathbf{x}; \mathbf{y}) = g(\mathbf{x} - \mathbf{y}) \qquad \text{and} \qquad \gamma_t(\mathbf{x}) = \gamma(\mathbf{x}) \tag{3.1}$$

so that the *change* between an offspring type and its altered form follows the density $g(\boldsymbol{\eta})$, called the *distribution of the mutation disturbance*. The stipulation of (3.1) in conjuction with (2.13) is equivalent to the linear relation

$$\mathbf{X}_{t+1} = \mathbf{X}^*_{t+1} + \mathbf{e}_{t+1}\,, \tag{3.2}$$

where $\mathbf{X}_{t+1}$ of generation $t+1$ is determined as a sum of two terms, the offspring type $\mathbf{X}^*_{t+1}$ perturbed by an independent residual $\mathbf{e}_{t+1}$ following the density law $g(\mathbf{e})$ independent of t.

We also assume that the conditional transmission-segregation rule is determined as a linear sum of two independent parts

$$\mathbf{X}^*_{t+1} = R^{(\mathrm{m})}\tilde{\mathbf{X}}_t + S^{(\mathrm{m})}\tilde{\mathbf{Y}}_t + \boldsymbol{\xi}^{(\mathrm{m})}_t, \qquad \mathbf{Y}^*_{t+1} = R^{(\mathrm{f})}\tilde{\mathbf{X}}_t + S^{(\mathrm{f})}\tilde{\mathbf{Y}}_t + \boldsymbol{\xi}^{(\mathrm{f})}_t, \tag{3.3}$$

where R and S are matrices acting on the parental phenotypes $\tilde{\mathbf{X}}$ and $\tilde{\mathbf{Y}}$ which prescribes the segregation contributions to the offspring, while $\boldsymbol{\xi}$ is an independent residual term following the density $h(\cdot)$. The general transmission law $L(\mathbf{u}; \mathbf{x}, \mathbf{y})$ of (2.9) allows for bona fide environment-phenotype interactions in contrast to (3.3).

In a population where each parent transmits in the same way to the offspring type, then $R = S$. In the literature the linear transmission usually has been restricted to the case $R = S$, often with $R = S =$ a diagonal matrix. When the exact replica of the parental types is passed on equally, blending their contributions, then a natural specification has

$$R = S = \tfrac{1}{2}I \qquad (I = \text{identity matrix}) \tag{3.4}$$

such that the offspring inherits the midparental phenotypes modulo some random environmental effects or interactions. When the sexes transmit their types differentially to the offspring type then the condition $R \neq S$ is likely in force.

For the stipulation $R = 0$ two immediate interpretations can be appropriate:

(a) The model is equivalent to a haploid model.

(b) The transmission is uniparental (say manifesting only maternal inheritance), allowing interpretations pertaining to cultural transmission endowments.

(c) If $R^{(m)} = S^{(f)} = I$ and $R^{(f)} = S^{(m)} = 0$, then the genetic transmission reflects pure replication (modulo random environmental terms) of the relevant parental type. The dynamic behavior differs significantly even from the symmetric diploid case with $R = S$; see Karlin (II).

For a scalar trait, (3.3) becomes

$$x_{t+1}^{(m)} = \alpha\tilde{x}_t^{(m)} + \beta\tilde{x}_t^{(f)} + \xi_{t+1}^{(m)}, \qquad x_{t+1}^{(f)} = \gamma\tilde{x}_t^{(m)} + \delta\tilde{x}_t^{(f)} + \xi_{t+1}^{(f)}, \tag{3.5}$$

where $(\tilde{x}_t^{(m)}, \tilde{x}_t^{(f)})$ denote the spouse trait values of generation t and $(x_{t+1}^{(m)}, x_{t+1}^{(f)})$ the progeny male and female trait values. Four special cases of interest are

$$\begin{aligned}
&\alpha = \beta = \gamma = \delta = \tfrac{1}{2} && \text{(midparental transmission)},\\
&\beta = \delta, \quad \{\alpha, \gamma \text{ small}\} && \text{(primary maternal transmission)},\\
&\alpha = \delta, \quad \beta = \gamma && \text{(sex symmetric transmission)},\\
&\alpha = \beta, \quad \gamma = \delta && \text{(equal parental transmission but offspring sex-dependent)}.
\end{aligned} \tag{3.6}$$

The offspring male (female) can inherit more or less than twice the midparental blend according as $\alpha > \beta$ $(\gamma > \delta)$ or $\alpha < \beta$ $(\gamma < \delta)$. The traditional adherance to midparent transmission rests on the proposition that an offspring receives half his genes equally from each parent. There is no reason to assume that a phenotypic (physiological, morphological, behavioral, cultural) trait carries equal parental contributions. With cultural components we could likely expect asymmetrical maternal–paternal transmission. Certainly for cultural variables and even for many physiological phenotypes the parental part of the trait expression may be attenuated or amplified, corresponding to $\alpha + \beta < 1$, $\gamma + \delta < 1$ or $\alpha + \beta > 1$, $\gamma + \delta > 1$, respectively. Some cases of maternal and paternal effects are discussed in numerical terms by Cavalli–Sforza and Feldman (1977) and Rao *et al.* (1979).

The environmental random perturbations $\xi^{(m)}$ and $\xi^{(f)}$ may be correlated but are assumed independent of the parental phenotypes.

The Multideme Polygenic Trait Model

Suppose $\mathbf{x} = (x_1, \ldots, x_N)$ represents a single real phenotype distributed at n localities so that $\mathbf{x}$ describes a group of individuals—one from each locality. Given the state $\mathbf{y}$, let

$$\mathbf{x} = M\mathbf{y} + \boldsymbol{\eta}$$

represent the state resulting from migration among demes, $M = \| m_{ij} \|$ is a

matrix with $\sum_{j=1}^{n} m_{ij} = \mu_i < 1$ for all i, and η is distributed with the density $g(\eta)$. The contribution η can be viewed in the traditional way as the input to the phenotypic value at the respective localities from a large external (or hypothetical) population in equilibrium. In this setup migration can replace mutation forces or be superimposed on the previous forces.

4. The Multivariate Gaussian Model of Phenotype Inheritance

We now specialize the model of Section 2, postulating that the underlying distributions are multivariate normal and that the viability selection and assortative mating functions conform to a normal density. Studies on the non-normal model are presented in Karlin (VII, VIII). We concentrate first on the model under pure random mating and deal with assortative mating structure in Section 5.

The specific assumptions are as follows:

(i) *Fitness (or viability selection) function*

$$\gamma(\mathbf{x}) = \exp[-\tfrac{1}{2}\langle(\mathbf{x} - \boldsymbol{\gamma}), C(\mathbf{x} - \boldsymbol{\gamma})\rangle] = \exp\left\{-\tfrac{1}{2}\sum_{i,j=1}^{n}(x_i - \gamma_i)(x_j - \gamma_j)\, c_{ij}\right\}, \tag{4.1}$$

where $\| c_{ij} \| = C$ conveys optimizing (stabilizing) selection with mode at $\boldsymbol{\gamma}$. We sometimes *write* $C = \Gamma^{-1}$, whenever Γ is defined; the superscript (-1) refers to the inverse matrix. *The expression* (4.1) *is well defined in terms of* c_{ij} *even where* $C = \| c_{ij} \|$ *is not the inverse of a positive definite matrix. In fact, it will be useful at times to take* C *as merely semidefinite and occasionally as just a symmetric matrix without any further restrictions.*

(ii) *Transmission-segregation disturbance distribution*

$h(\xi)$ is a normal density with mean $\mathbf{h}$ and covariance matrix H. (4.2)

(iii) *Mutational disturbance distribution*

$g(\eta)$ is a normal density with mean $\mathbf{m}$ and covariance matrix M. (4.3)

A normal distribution with mean vector $\boldsymbol{\mu}$ *and covariance matrix* Σ *is denoted hereafter by* $N(\boldsymbol{\mu}, \Sigma)$.

It is useful to summarize the forces and parameters of the normal (multivariate) model in tabular form

	Mean	Covariance matrix
Selection $\gamma(\mathbf{x})$ = normal density	$\boldsymbol{\gamma}$	$\Gamma = C^{-1}$ (when C^{-1} exists)
Segregation-transmission conditional distribution $L(\mathbf{x} \mid \tilde{\mathbf{x}}^{(m)}, \tilde{\mathbf{x}}^{(f)})$ multivariate normal	$R\tilde{\mathbf{x}}^{(m)} + S\tilde{\mathbf{x}}^{(f)} + \mathbf{h}$	H
Conditional mutation distribution $g(\mathbf{x}; \boldsymbol{\eta})$ multivariate normal	$\mathbf{m} + \boldsymbol{\eta}$	M

$$\mathbf{x}_{t+1} = R\tilde{\mathbf{x}}_t^{(m)} + S\tilde{\mathbf{x}}_t^{(f)} + \boldsymbol{\xi}$$

where $\boldsymbol{\xi}$, independent of $\tilde{\mathbf{x}}^{(m)}$ and $\tilde{\mathbf{x}}^{(f)}$, is normally distributed with mean $\mathbf{h} + \mathbf{m} = \boldsymbol{\varphi}$ and covariance matrix $F = H + M$.

The dynamics of the phenotypic process $\{X_t\}$ are described in the following familiar result.

(i) *Assume the density of* X_t, $p_t(\mathbf{x})$, *is* $N(\boldsymbol{\mu}_t, \Sigma_t)$, *i.e., follows a Gaussian distribution with mean vector* $\boldsymbol{\mu}_t$ *and covariance matrix* Σ_t. *Then* $p_{t+1}(\mathbf{x})$ *is distributed* $N(\boldsymbol{\mu}_{t+1}, \Sigma_{t+1})$, *where the parameters are determined recursively according to*

$$\Sigma_{t+1} = R(\Sigma_t^{-1} + \Gamma^{-1})^{-1} R' + S(\Sigma_t^{-1} + \Gamma^{-1})^{-1} S' + F, \tag{4.4}$$

with $F = H + M$ (R' *denotes the transpose matrix to* R). The matrix F may be construed as the total contribution to the covariances per generation, accruing from mutational and/or random environmental effects plus perturbation deviations from parental transmission. The mean vector change conforms to the recursion

$$\boldsymbol{\mu}_{t+1} = (R + S)(\Sigma_t^{-1} + \Gamma^{-1})^{-1}(\Sigma_t^{-1}\boldsymbol{\mu}_t + \Gamma^{-1}\boldsymbol{\gamma}) + \boldsymbol{\varphi}, \tag{4.5}$$

where $\boldsymbol{\varphi} = \mathbf{h} + \mathbf{m}$.

Remark 4.1. When the order of forces conforms to (2.3), Model II, such that the population is sampled at the adult stage after selection, (4.4) is replaced by

$$\tilde{V}_{t+1} = ((RV_tR' + SV_tS' + F)^{-1} + \Gamma^{-1})^{-1}, \tag{4.6}$$

and for the order of forces (2.4), Model III, again with census at the adult stage, then (4.4) is modified to the form

$$W_{t+1} = ((R(W_t + F)\,R' + S(W_t + F)S')^{-1} + \Gamma^{-1})^{-1}. \tag{4.7}$$

The substitution $\tilde{F} = RFR' + SFS'$ converts (4.7) into (4.6). The substitution $\tilde{\Sigma} = RVR' + SVS' + F$ converts (4.6) into (4.4). In the case $R = S$ is invertible, the inverse transformations of (4.4) to (4.6) and (4.6) to (4.7) are

$$\tilde{V} = (2^{1/2}R)^{-1}(\Sigma - F)(2^{1/2}R')^{-1}, \qquad \tilde{F} = (2^{1/2}R)^{-1}F(2^{1/2}R')^{-1},$$

respectively.

The relationship (4.4) (as well as (4.6) and (4.7)) can be regarded as a *matrix linear fractional transformation.* Our objective is to ascertain the dynamic and limiting behavior of Σ_t and also that of μ_t. *Without any restrictions we can establish the existence of* $\Sigma_\infty = \lim_{t\to\infty} \Sigma_t$ *independent of the initial* Σ_0 (Result I of Section 11) *where* Σ_∞ *is the unique solution of the matrix equation* (4.4) *obtained by inserting* Σ_∞ *in place of* Σ_t *and* Σ_{t+1}. If F and Γ are positive definite, then Σ_∞ is positive definite and finite. The conditions ensuring $\lim_{t+\infty} \mu_t = \mu_\infty$ are slightly more restrictive (Result II of Section 11).

Explicit representations for Σ_∞ and μ_∞ are available in some important cases, (Karlin, VI). Owing to the uniqueness, Σ_∞ can be approximated by successive iteration of (4.4) (or (4.6), (4.7)) starting with any positive definite Σ_0. The equilibrium parameters Σ_∞ and μ_∞, and thereby $N(\mu_\infty, \Sigma_\infty)$, characterize the evolutionary outcome of the phenotypic vector trait subject to optimizing selection (of the form (4.1)), transmission as described in (3.3) with normal environmental disturbances and mutation residuals.

Regression on the Population Mean

In the spirit of the classic Galtonian formulation the convergence results extend to the model in which the transmission law is

$$\mathbf{x}_{t+1} = \alpha^{1/2}T\,\frac{\tilde{\mathbf{x}}_t^{(1)} + \tilde{\mathbf{x}}_t^{(2)}}{2} + \beta\mu_t + \xi^t, \tag{4.8}$$

where T is a transmission matrix operating on the midparental value, μ_t is the mean population type and ξ is an independent random-environmental contribution (cf. Lande and Slatkin, 1975; see also Karlin *et al.*, 1979). Thus, the transmission law of (4.8) entails a weighted combination of a direct parental input plus a deterministic part directed toward the current population mean phenotype. The magnitudes of the coefficients α and β govern or reflect the relative weights of the two parts.

A possible interpretation has the term $\beta\mu_t$ as a form of social conformance. The other forces (natural and mating selection, transmission, mutation, etc.)

transpire as before. The recursion of the phenotype covariance matrix over time attains a form similar to (4.4). The mean phenotype changes are calculated paraphrasing (4.5).

5. A Vector Phenotype Model for a Two-Sex Population Subject to Nonlinear Assortative Mating Pattern

In a model with separate sexes we consider for generation t the female and male adult phenotype densities at generation t, $p_t(\mathbf{x}) \approx N(\boldsymbol{\lambda}_t, U_t)$ and $q_t(\mathbf{y}) \approx N(\boldsymbol{\upsilon}_t, V_t)$, respectively. Assuming a priori equal probability of meeting mates, the frequency of an encounter of a male $\mathbf{x}$ phenotype and $\mathbf{y}$ female phenotype is $p_t(\mathbf{x})\, q_t(\mathbf{y})$. The assortative mating mechanism is effected via the *selection mating function* $\phi(\mathbf{x}, \mathbf{y})$ that indicates the conditional probability of a successful mating of the $\mathbf{x}$ male and $\mathbf{y}$ female. In line with (2.11) the induced density of a spouse couple is

$$\tilde{p}_t(\mathbf{x}, \mathbf{y}) = \frac{p_t(\mathbf{x})\, q_t(\mathbf{y})\, \phi(\mathbf{x}, \mathbf{y})}{\iint p_t(\boldsymbol{\xi})\, q_t(\boldsymbol{\eta})\, \phi(\boldsymbol{\xi}, \boldsymbol{\eta})\, d\boldsymbol{\xi}\, d\boldsymbol{\eta}}. \tag{5.1}$$

This kind of selection assortative mating operation introduced in a generalized one-gene model with continuous phenotype expression occurs in Wilson (1973) and Wagener (1976).

Conforming to the Gaussian model we take $\phi(\mathbf{x}, \mathbf{y})$ of the form

$$\phi(\mathbf{x}, \mathbf{y}) = \exp\left[-\frac{1}{2} \sum_{i,j=1}^{2n} (z_i - \gamma_i)\, \psi_{ij} (z_j - \gamma_j)\right], \tag{5.2}$$

where

$$\Psi = \| \psi_{ij} \|$$

is a $2n \times 2n$ positive semidefinite matrix and $\mathbf{z} = \{\mathbf{x}, \mathbf{y}\}$ is the $2n$-tuple vector with the female phenotype components $\mathbf{y}$ juxtaposed to the male phenotype $\mathbf{x}$. *We write $\Psi = \Phi^{-1}$ where the positive definite matrix Φ exists.* It may be relevant to have $\boldsymbol{\gamma}$ linearly dependent on the population's mean $\boldsymbol{\mu}_t$ so that $\phi_t(\mathbf{x}, \mathbf{y})$ depends on t (cf. (4.8)). The influence of $\boldsymbol{\gamma}_t = \delta\boldsymbol{\mu}_t + \boldsymbol{\theta}$ does not occur in the analysis of the changes in the population covariance structure Σ_t over time. The special modal phenotype $\boldsymbol{\gamma} = (\gamma_1, \ldots, \gamma_{2n})$ of (5.2) can be construed as a standard that social or cultural conditions impose on population mating tendencies.

It is worth emphasizing that the $\mathbf{x}$ vector-phenotype can include the usual phenotype variables plus cultural variables such that the structure of (5.1) accommodates the possibility that the assortative mating determination is based on a partial set of its phenotype components. In this way, the contingencies of cultural (or "common environmental") versus phenotype assort-

ment is encompassed by the flexibility in the delineation of the vector phenotypes $\mathbf{x}$ and $\mathbf{y}$ and by the determination of the matrix $\Psi = \Phi^{-1}$ that characterizes $\phi(\mathbf{x}, \mathbf{y})$. That is, where $\phi(\mathbf{x}, \mathbf{y})$ concentrates on the cultural components, e.g., education or social class, we can speak of cultural assortment and when $\phi(\mathbf{x}, \mathbf{y})$ depends only on the more usual biological components, e.g., height or blood pressure, assortment can be said to have a primary phenotype ("genetic") basis. The concepts of traditional phenotypic assortment as against cultural assortment and further criteria based on "social homogamy" promulgated in the discussions of Rao *et al.* (1976), Cloninger *et al.* (1978), Cavalli-Sforza and Feldman (1977), among others, are subsumed in the flexibility of the vector trait formulation and the scope of the selection mating function. The essential distinction in our assortment mechanisms rests on the fact that we treat it as a selection process (which makes it automatically nonlinear), while previous studies of phenotypic inheritance incorporating assortative mating follow the linear modeling structure in the style of Fisher (see Section 10 and Karlin (V) for further discussion of this matter).

The mating selection function assignment for a phenotypic scalar trait conforming with (5.2) becomes

$$\phi(x, y) = \exp\left[-\frac{1}{2c(1-r^2)}\left(\frac{x^2}{f^2} - \frac{2rxy}{fg} + \frac{y^2}{g^2}\right)\right],$$

$$c > 0, \quad -1 < r < 1, \quad f, g \text{ positive unrestricted.}$$

Here r serves as a measure of the degree of the *preferential concordance* between the mate phenotype values, possibly also based on appropriate cultural or environmental concomitants (e.g., social homogamy, physical coincidence). The parameter c can be regarded as a measure of the width of the preference function. For $f \to \infty$, the assortment probabilities depend only on the maternal phenotype value y. For $f = g = 1$, a sex semigeometric form,

$$\phi(x, y) = \exp\left[-\frac{1}{2c(1-r^2)}(x^2 - 2rxy + y^2)\right] \tag{5.3}$$

with $-1 < r < 1$, $c > 0$. The probability level curves are decreasing to zero as $|x|$ and/or $|y|$ tend to ∞. Accordingly the chance for a successful mating is diminishingly small when one or both traits are extreme.

Consider next the special mating preference function

$$\phi(x, y) = \exp\left[-\frac{1}{2c^*}(x - y)^2\right]. \tag{5.4}$$

This arises from (5.3) by letting $r \to 1$ and $c \to \infty$ in such a way that $c(1 - r) \to c^*/2$. The probability level curves for (5.4) are straight lines (degenerate ellipsoids). This means that concordance of the male and female phenotype

values (independent of their magnitude) invites for successful matings. The quadratic form of the exponential in (5.4) is *singular of rank* 1 unlike that of (5.3), where the quadratic form is nonsingular positive definite.

An interesting case of $\phi(\mathbf{x}, \mathbf{y})$ of (5.2) generalizing (5.4) prescribes

$$\phi(\mathbf{x}, \mathbf{y}) = \exp\left[-\frac{1}{2c}\sum_{i=1}^{n}(x_i - y_i)^2\right], \qquad c > 0 \tag{5.5}$$

where $1/c$ can be construed as an *index of the strength* (or *width*) of the preference function. Specifically, $c \to +\infty$ renders the mating system as pure random mating. For the specification (5.5) the $2n \times 2n$ associated covariance matrix is

$$\Psi = \begin{pmatrix} \overbrace{\frac{1}{c} & 0 & \cdots & 0}^{n} & -\frac{1}{c} & 0 & \cdots & 0 \\ 0 & \frac{1}{c} & & & 0 & -\frac{1}{c} & & \\ \vdots & & & & & & & 0 \\ 0 & 0 & \cdots & \frac{1}{c} & 0 & 0 & \cdots & -\frac{1}{c} \\ -\frac{1}{c} & & \cdots & 0 & \frac{1}{c} & & & 0 \\ 0 & & & & & & & \\ 0 & & \cdots & -\frac{1}{c} & 0 & 0 & \cdots & \frac{1}{c} \end{pmatrix} \tag{5.6}$$

$$= \frac{1}{c} J \otimes I \quad \text{(the Kronecker product of the matrices } J \text{ and } I\text{)},$$

where I is the $n \times n$ identity matrix and J is the 2×2 matrix $\left(\begin{smallmatrix} 1 & -1 \\ -1 & 1 \end{smallmatrix}\right)$. A natural extension of (5.6) is

$$\phi(\mathbf{x}, \mathbf{y}) = \exp\left\{-\frac{1}{2c}\sum_{i=1}^{n}\theta_i(x_i - y_i)^2\right\}, \tag{5.7}$$

where the θ_i allow different weightings of the components. The mating selection function of (5.6) is singular.

In the case that $\Phi^{-1} = \Psi$ is singular positive semidefinite the convergence behavior of the population covariance matrix Σ_t can be a bit restricted (Karlin, II). The realistic case would likely have $\Phi^{-1} = \Psi$ positive definite.

A direct analysis on (5.1) reveals that $\tilde{p}_t(\mathbf{x}, \mathbf{y})$ *the density of a viable spouse couple is a normal density with covariance matrix* $\tilde{C}_t$ *of order* $2n \times 2n$ *given by*

$$\tilde{C}_t = (C_t + \Psi^{-1})^{-1}, \tag{5.8}$$

where C_t is the $2n \times 2n$ covariance matrix of the joint density $p_t(\mathbf{x})\, q_t(\mathbf{y})$, which in turn is of the form

$$C_t = \begin{pmatrix} \Sigma_t^{(m)} & \mathbf{0} \\ \mathbf{0} & \Sigma_t^{(f)} \end{pmatrix} \qquad \text{where } \Sigma_t^{(m)}\ (\Sigma_t^{(f)}) \text{ is the covariance matrix of } \mathbf{X}_t^{(m)}\ (\mathbf{X}_t^{(f)}). \tag{5.8a}$$

For those accustomed to thinking in terms of spouse covariance matrices, we have

$$\tilde{C}_t = \begin{pmatrix} \tilde{\Sigma}_t^{(m)} & \tilde{M}_t \\ \tilde{M}_t' & \tilde{\Sigma}_t^{(f)} \end{pmatrix} \qquad \text{where} \quad \tilde{M}_t = \mathrm{Cov}(\tilde{\mathbf{X}}_t, \tilde{\mathbf{Y}}_t) = \Delta_t^{(m)} \Theta_t \Delta_t^{(f)}, \tag{5.8b}$$

where $\Delta_t^{(m)}$ ($\Delta_t^{(f)}$) is the diagonal matrix with male (female) spouse standard deviation values down the diagonal and Θ_t is the spouse correlation matrix of generation t.

The transmission-segregation process associated with an established pairing produces male and female offspring following the transmission law

$$\mathbf{x}_{t+1}^* = \tilde{R}\tilde{\mathbf{z}}_t + \boldsymbol{\xi}_t, \qquad \mathbf{y}_{t+1}^* = \tilde{S}\tilde{\mathbf{z}}_t + \boldsymbol{\eta}_t, \tag{5.9}$$

where $\tilde{\mathbf{z}}$ is a spouse pair distributed according to $\tilde{p}(\mathbf{z}) = \tilde{p}(\mathbf{x}, \mathbf{y})$ of (5.1) and $\tilde{R}$ and $\tilde{S}$ are $n \times 2n$ matrices. Also, the $2n$ environmental-mutation perturbation vector $\boldsymbol{\theta}_t = \{\boldsymbol{\xi}_t, \boldsymbol{\eta}_t\}$ is distributed $N(\boldsymbol{\theta}, F)$, where $\boldsymbol{\theta}$ is a vector of $2n$ coordinates and F is now a covariance matrix of order $2n \times 2n$. $\tilde{R}$ and $\tilde{S}$ may differ under sex-dependent transmission with respect to parental contribution and/or offspring expression.

It follows that a male child of the next generation is distributed according to the density $p_{t+1}^*(\mathbf{x})$, which is the normal density with covariance matrix $\Sigma_{t+1}^{(m)} = U_{t+1}^* = \tilde{R}\tilde{C}_t\tilde{R}' + F_1$, where F_1 consists of the first n rows and columns of F. Similarly, we find that an *independent* female child is governed by the density $q_{t+1}^*(\mathbf{y})$ with covariance matrix $\Sigma_{t+1}^{(f)} = V_{t+1}^* = \tilde{S}_t\tilde{C}_t\tilde{S}' + F_2$, where F_2 consists of the last n rows and columns of F. If viability selection is inconsequential then obviously

$$C_{t+1} = \begin{pmatrix} U_{t+1}^* & 0 \\ 0 & V_{t+1}^* \end{pmatrix}. \tag{5.10}$$

It is useful to express the representation in the form

$$C_{t+1} = \begin{pmatrix} U_{t+1}^* & 0 \\ 0 & V_{t+1}^* \end{pmatrix} = \hat{R}\tilde{C}_t\hat{R}' + \hat{S}\tilde{C}_t\hat{S}' + \hat{F}, \tag{5.11}$$

where $\hat{R}$ is the $2n \times 2n$ extended square matrix $\| \begin{smallmatrix} \tilde{R} \\ 0 \end{smallmatrix} \|$ augmented by a block of n

rows and $2n$ columns of zero entries under the matrix $\tilde{R}$ as displayed. Similarly, we have $\hat{S} = \| \begin{smallmatrix} 0 \\ S \end{smallmatrix} \|$ and define $\hat{F}$ as the block diagonal matrix

$$\begin{pmatrix} F_1 & 0 \\ 0 & F_2 \end{pmatrix}.$$

We should emphasize that the extended covariance matrix C_t refers to a male and female individual in the polulation at large and not to two sibs.

The conjunction of (5.8) and (5.11) provides the recursion relationship (for matrices of order $2n$)

$$C_{t+1} = \hat{R}(C_t^{-1} + \Psi)^{-1}\hat{R}' + \hat{S}(C_t^{-1} + \Psi)^{-1}\hat{S}' + \hat{F}^*, \tag{5.12}$$

which is of the form (4.4). With (5.12) in hand, the convergence theorems (Section 11) apply, establishing (for the two-sex model with the general assortative mating mechanism of (5.1)) that Σ_t converges to a unique equilibrium covariance matrix Σ_∞. Note that if Ψ is only semidefinite (e.g., when the mating selection function depends only on a subset of the variables), then the finite convergence equilibrium covariance matrix often occurs, albeit not universally guaranteed (cf. Karlin, II, Section 2).

We can easily incorporate the consequences of natural selection and then $p^*_{t+1}(\mathbf{x})$ is converted to $p_{t+1}(\mathbf{x})$ having covariance matrix

$$U_{t+1} = ((U^*_{t+1})^{-1} + \Gamma_1^{-1})^{-1} \tag{5.13}$$

and similarly $q^*_{t+1}(\mathbf{y})$ transformed to $q_{t+1}(\mathbf{y})$ is characterized by the covariance matrix

$$V_{t+1} = ((V^*_{t+1})^{-1} + \Gamma_2^{-1})^{-1}. \tag{5.14}$$

The matrices Γ_1^{-1} and Γ_2^{-1} correspond to the Gaussian viability selection functions as in (2.6) for male and female individuals, respectively.

The relations (5.12)–(5.14) imply the connections of the covariance pair

$$C_t = \begin{pmatrix} U_t & 0 \\ 0 & V_t \end{pmatrix} \quad \text{to} \quad C_{t+1} = \begin{pmatrix} U_{t+1} & 0 \\ 0 & V_{t+1} \end{pmatrix} \tag{5.15}$$

over two successive generations. The development of the covariance structure Σ_t and also the mean trends $(\boldsymbol{\lambda}_t, \boldsymbol{\nu}_t)$ over time describes a two-sex phenotypic population evolution subject to natural selection, mating selection entailing both random and assortative mating patterns, a bisexual transmission-segregation mechanism, and mutational and environmental perturbations. Of particular interest would be to contrast the relative influence of mating selection reflected by the existence of mating combinations (in the form of (5.1)) versus the force of natural selection acting as in (2.5) and (2.6) with differential selection expression

between sexes when $\Gamma_1 \neq \Gamma_2$. The requirements for $\Sigma_t \to \Sigma_\infty$ (Results I and II, Section 11) apply to this generalized context. We describe various facets and contrasting properties on bounds and dependence relations of Σ_∞ on the mating, transmission, and environmental parameters in Karlin (II) and (VI).

6. A Phenotypic Nuclear Family Vector Trait Model with Selective Assortative Mating

The following model shows the flexibility and wide scope of the phenotypic vector trait formulation. At first we take the basic unit as a nuclear family composed of two parents and one child. Afterward we briefly indicate the model with a nuclear family having two children, one of each sex; the model carrying a prescribed sibship size can be handled by appropriate modifications. The vector trait is now summarized by three groups of coordinates,

$$\mathbf{x} = (\mathbf{x}^{(m)}, \mathbf{x}^{(f)}, \mathbf{c}), \tag{6.1}$$

comprised of

$$\begin{aligned} \mathbf{x}^{(m)} &= \text{male parent phenotype,} \\ \mathbf{x}^{(f)} &= \text{female parent phenotype,} \\ \mathbf{c} &= \text{child phenotype,} \end{aligned} \tag{6.2}$$

where each is an n-tuple measurement. For convenience of exposition, we deal with a sex symmetric situation where male and female children follow the same distribution law. (It is worth emphasis that a unit of time in the nuclear family model keeps track of related individuals of two generations.) For a second nuclear family represented by $\mathbf{y} = (\mathbf{y}^{(m)}, \mathbf{y}^{(f)}, \mathbf{d})$, we form the $6n$ vector $\mathbf{z} = (\mathbf{x}, \mathbf{y})$ of the two joined nuclear families. We stipulate that a union occurs between the young of the two families with probability

$$\phi(\mathbf{z}) = \exp[-\tfrac{1}{2}\langle \mathbf{z}, \Psi\mathbf{z}\rangle] \tag{6.3}$$

given that they meet. In this nuclear family setup even the parental types may influence the success of the union. If the phenotype trait $\mathbf{x}$ is an n-tuple vector, then $\Psi = \Phi^{-1}$ can in this formulation, a priori, be a $6n \times 6n$ positive definite form. It is suggestive to partition Ψ as indicated

$$\Psi = \begin{vmatrix} \Gamma_{11} & \Gamma_{12} & \Gamma_{13} & \Gamma_{14} & \Gamma_{15} & \Gamma_{16} \\ \Gamma_{21} & \Gamma_{22} & \Gamma_{23} & \Gamma_{24} & \Gamma_{25} & \Gamma_{26} \\ \Gamma_{31} & \Gamma_{32} & \Gamma_{33} & \Gamma_{34} & \Gamma_{35} & \Gamma_{36} \\ \Gamma_{41} & \Gamma_{42} & \Gamma_{43} & \Gamma_{44} & \Gamma_{45} & \Gamma_{46} \\ \Gamma_{51} & \Gamma_{52} & \Gamma_{53} & \Gamma_{54} & \Gamma_{55} & \Gamma_{56} \\ \Gamma_{61} & \Gamma_{62} & \Gamma_{63} & \Gamma_{64} & \Gamma_{65} & \Gamma_{66} \end{vmatrix}, \tag{6.4}$$

where each Γ_{ij} is an nth-order matrix.

When only the children's phenotypes affect the mating probabilities then Ψ effectively reduces to a $2n \times 2n$ matrix. The detailed structure (6.4) can be interpreted to embody factors of social homogamy, customs, wealth status, external environmental, and other familial influences, especially if some of the trait components are cultural attributes. In line with (5.1) the joint distribution of two united families is

$$\begin{aligned}\tilde{p}(\mathbf{z}) &= \tilde{p}(\mathbf{x}, \mathbf{y}) = \tilde{p}(\mathbf{x}^{(m)}, \mathbf{x}^{(f)}, \mathbf{c}; \mathbf{y}^{(m)}, \mathbf{y}^{(f)}, \mathbf{d}) \\ &= Kp(\mathbf{x})\, p(\mathbf{y})\, \phi(\mathbf{z}) \end{aligned} \tag{6.5}$$

where K is a normalizing constant. The $6n$ covariance matrix for $\tilde{p}(\mathbf{z})$is computed by the recipe (5.8), namely,

$$\tilde{C}_t = \left(\begin{pmatrix} \Sigma_t^{-1} & 0 \\ 0 & \Sigma_t^{-1} \end{pmatrix} + \Psi\right)^{-1}, \tag{6.6}$$

where Σ_t is the $3n \times 3n$ covariance matrix of a nuclear family. With the distribution (6.5) in hand we can routinely extract the marginal joint distribution of the newly established couple

$$p(\hat{\mathbf{c}}, \hat{\mathbf{d}}) \tag{6.7}$$

with $\hat{\mathbf{c}} = \mathbf{c}$, $\hat{\mathbf{d}} = \mathbf{d}$; the hat emphasizes that the union is established in accordance with the operation of the mating selection structure induced by (6.4).

The transmission-segregation implementation for the newly formed couple produces an offspring of phenotype value

$$\mathbf{r} = T(\hat{\mathbf{c}}, \hat{\mathbf{d}}) + \boldsymbol{\theta},$$

where T is an $n \times 2n$ matrix operating on the joint vector $(\hat{\mathbf{c}}, \hat{\mathbf{d}}) = (\mathbf{c}, \mathbf{d})$ and $\boldsymbol{\theta}$ is an environmental-mutational perturbation.

The family of the next generation is the conglomerate triplet $\mathbf{x}' = (\hat{\mathbf{c}}, \hat{\mathbf{d}}, \mathbf{r})$. The transition from

$$\mathbf{x} \text{ of (6.2) to } \mathbf{x}' = (\hat{\mathbf{c}}, \hat{\mathbf{d}}, \mathbf{r}) \tag{6.8}$$

encompasses the changes from a nuclear family of one generation to the next. The transformation of the distribution of $\mathbf{x}$ to $\mathbf{x}'$ or, more accurately, the mapping of $\mathbf{z}$ to $\mathbf{z}'$ ($\mathbf{z}$ determined in (6.5) referring to a pair of nuclear families) is of the form (4.4) and, therefore (by Result I, Section 11), *a globally attracting equilibrium phenotypic covariance array for the transition of nuclear families over successive generations exists.*

Nuclear Family of Four Members

The model of nuclear families with any prescribed family size can be handled by the same means as that above. To be concrete, we concentrate on nuclear

families consisting of two children (one of each sex) and on unions that lead to that same progeny makeup.

A family unit for our present purposes is described by the four n-tuple of phenotype arrays

$$\mathbf{f} = (\mathbf{x}^{(m)}, \mathbf{x}^{(f)}, \mathbf{c}_1^{(m)}, \mathbf{c}_2^{(f)}), \tag{6.9}$$

following a Gaussian distribution $p(\mathbf{f})$. In line with the assortative mating processes of Section 5, when a union is established between two nuclear families of phenotype configurations $\mathbf{f}_1$ and $\mathbf{f}_2$, the realization follows the joint probability density

$$\tilde{p}(\mathbf{f}_1, \mathbf{f}_2) = Kp(\mathbf{f}_1)\, p(\mathbf{f}_2)\, \psi(\mathbf{f}_1, \mathbf{f}_2), \tag{6.10}$$

where $\psi(\mathbf{f}_1, \mathbf{f}_2)$ stands for the conditional probability of a union (i.e., among a pair of children) culminating the contacts of the given nuclear families.

Stipulating $\psi(\mathbf{f}_1, \mathbf{f}_2)$ as a Gaussian selection function akin to (6.3), the normal distribution (i.e., mean and covariance structure) of (6.10) is readily ascertained. With $\psi(\mathbf{f}_1, \mathbf{f}_2)$ possibly depending on all the components of $\mathbf{f}_1$ and $\mathbf{f}_2$, the probability of the union between these families may be influenced by all the members thereof or a part of them; compare to the discussion of mating preference functions like (6.4).

The resulting nuclear family of the union of $\mathbf{f}_1$ and $\mathbf{f}_2$ consists of, for definiteness,

$$\mathbf{g} = (\tilde{\mathbf{c}}_1^{(m)}, \tilde{\mathbf{c}}_2^{(f)}, \mathbf{o}^{(m)}, \mathbf{o}^{(f)}), \tag{6.11}$$

where $\tilde{\mathbf{c}}_1^{(m)} = \mathbf{c}_1^{(m)}$ is the male child of family $\mathbf{f}_1$ marrying the female child $\tilde{\mathbf{c}}_2^{(f)} = \mathbf{c}_2^{(f)}$ of family $\mathbf{f}_2$, where $(\tilde{\mathbf{c}}_1^{(m)}, \tilde{\mathbf{c}}_2^{(f)})$ follow the marginal density induced by that of (6.10).

The children types $\mathbf{o}^{(m)}$ and $\mathbf{o}^{(f)}$ resulting from this union are determined in the usual manner, viz.,

$$\begin{aligned} \mathbf{o}^{(m)} &= R^{(m)}\tilde{\mathbf{c}}_1^{(m)} + S^{(m)}\tilde{\mathbf{c}}_2^{(f)} + \boldsymbol{\epsilon}^{(m)} \\ \mathbf{o}^{(f)} &= R^{(f)}\tilde{\mathbf{c}}_1^{(m)} + S^{(f)}\tilde{\mathbf{c}}_2^{(f)} + \boldsymbol{\epsilon}^{(f)} \end{aligned} \tag{6.12}$$

composed of a parental transmissible part and an independent residual environmental-mutational part. The transmission rule, in general, will be sex-dependent, as the notation of (6.12) indicates. It could even be more elaborate and depend on the grandparent types $(\tilde{\mathbf{x}}^{(m)}, \tilde{\mathbf{x}}^{(f)}, \tilde{\mathbf{y}}^{(m)}, \tilde{\mathbf{y}}^{(f)})$, particularly when cultural components are involved.

Extensions to Multigenerational Pedigrees

It is routine to extend the nuclear family model to allow for multigenerational effects. For example, the family unit would then involve both sets of grandparents,

the parents, and a prescribed sibship composition. The selection process establishing a union (wedding) of the two families paraphrases that of (6.10), where the mating selection function (the analog of ψ) can be influenced by part or all of the family member phenotypes.

It would be of much value to develop a model of nuclear families accommodating variable sibship sizes and their sex ratio, age distribution of children (e.g., two or three age classes), and other family characteristics. In this vein, it would also be of interest to try to assess the influences of sets of demographic, social, and ecological factors (e.g., population density, rural–urban contrasts, customs, life styles, climatic variables). Some of the above factors, e.g., scorings on religious or life-style traits, can be easily integrated as part of the phenotype by appending to the description of the trait vector more components. Geographic effects and migration patterns can also be dealt with by way of the transmission rule. However, the effects of family size engender more formidable problems. Indeed, the multivariate Gaussian distribution endowment is not preserved in the presence of different family sizes. The induced distribution of a family of a given size actually occurs as a mixture of several distributions and this realization corrupts the basic Gaussian distribution description, leaving the dynamic and equilibrium behavior of the process virtually prohibitive to track.

However, the model of this section can be adapted to study transmission on families with a specified set of collateral relatives including the occurrence of monozygotic and/or dizygotic twins.

7. The Multivariate Gaussian Phenotype Model with Two Age Classes

Let $\mathbf{x}_c = (x_1, \ldots, x_n)$ denote a male phenotype array in the child age class and $\mathbf{x}_a$ an adult male phenotype. The notations $\mathbf{y}_c$ and $\mathbf{y}_a$ refer to corresponding female phenotypes. Let these random variables follow the population densities

$$p_c^{(m)}(\mathbf{x}), p_a^{(m)}(\mathbf{x}), p_c^{(f)}(\mathbf{y}), p_a^{(f)}(\mathbf{y}). \tag{7.1}$$

For definiteness, the order of forces is taken as follows:

$$\begin{aligned} &\text{adults census } \underset{\mathbf{x}_a,\mathbf{y}_a}{} \xrightarrow{\text{mating pairings}} \text{couples } \underset{\tilde{\mathbf{x}}_a,\tilde{\mathbf{y}}_a}{} \xrightarrow{\text{phenotypic Galtonian transmission}} \text{child } \underset{\mathbf{x}'_c,\mathbf{y}'_c}{} \\ &\xrightarrow{\text{selection and cultural transmission}} \text{adult in the next generation. } \underset{\mathbf{x}'_a,\mathbf{y}'_a}{} \end{aligned} \tag{7.2}$$

The mating structure can encompass aspects of assortative mating (in terms of

a mating selection function) such that the induced joint distribution of a spouse pair as delineated in Section 5) is determined to be

$$\tilde{p}(\tilde{\mathbf{x}}, \tilde{\mathbf{y}}) = K p_{\mathrm{a}}^{(\mathrm{m})}(\mathbf{x})\, p_{\mathrm{a}}^{(\mathrm{f})}(\mathbf{y})\, \phi(\mathbf{x}, \mathbf{y}) \qquad (\tilde{\mathbf{x}}, \tilde{\mathbf{y}}) = (\mathbf{x}, \mathbf{y}) \tag{7.3}$$

where $\phi(\mathbf{x}, \mathbf{y})$ expresses the probability that the indicated couple mate, conditioned that they meet, and K is a normalizing constant to ensure that $\tilde{p}$ is a density.

As usual, the offspring of the next generation receive a transmissible component depending on the parental types plus an independent random disturbance factor in the form

$$\mathbf{x}_{\mathrm{c}}' = R^{(\mathrm{m})}\tilde{\mathbf{x}}_{\mathrm{a}} + S^{(\mathrm{m})}\tilde{\mathbf{y}}_{\mathrm{a}} + \boldsymbol{\epsilon}^{(\mathrm{m})}, \tag{7.4a}$$

$$\mathbf{y}_{\mathrm{c}}' = R^{(\mathrm{f})}\tilde{\mathbf{x}}_{\mathrm{a}} + S^{(\mathrm{f})}\tilde{\mathbf{y}}_{\mathrm{a}} + \boldsymbol{\epsilon}^{(\mathrm{f})}, \tag{7.4b}$$

where $R^{(\mathrm{m})}$, $S^{(\mathrm{m})}$, $R^{(\mathrm{f})}$, and $S^{(\mathrm{f})}$ are appropriate matrices that characterize the parental segregation-transmission rule where the environmental-mutational-error addends $\boldsymbol{\epsilon}^{(\mathrm{m})}$ and $\boldsymbol{\epsilon}^{(\mathrm{f})}$ are stipulated jointly Gaussian and independent of the parental makeup.

The explicit distributions of the next generation young individuals $p_{\mathrm{c}}^{(\mathrm{m})}(\mathbf{x}')$ and $p_{\mathrm{c}}^{(\mathrm{f})}(\mathbf{y}')$ are directly ascertained on account of the linear relationship of (7.4) and knowledge of (7.3).

The transformation of $\mathbf{x}_{\mathrm{c}}'$ to $\mathbf{x}_{\mathrm{a}}'$ (and $\mathbf{y}_{\mathrm{c}}'$ to $\mathbf{y}_{\mathrm{a}}'$) may involve some natural selection effects coupled to choices and innovations associated with cultural transmission. The action of selection, merging viability selection as (2.5) and cultural offspring–parental interactions, can transform the offspring and parents such that

$$\{\mathbf{x}_{\mathrm{c}}', \tilde{\mathbf{x}}_{\mathrm{a}}, \tilde{\mathbf{y}}_{\mathrm{a}}\} \to \{\mathbf{x}_{\mathrm{a}}^{*}, \tilde{\mathbf{x}}_{\mathrm{a}}, \tilde{\mathbf{y}}_{\mathrm{a}}\}. \tag{7.5}$$

The actual phenotype values of each family triplet remain unchanged but their population distribution is that of the normal density

$$p^{*}(\mathbf{x}^{*}, \tilde{\mathbf{x}}_{\mathrm{a}}, \tilde{\mathbf{y}}_{\mathrm{a}}) = \frac{\hat{p}(\mathbf{x}^{*}; \tilde{\mathbf{x}}_{\mathrm{a}}, \tilde{\mathbf{y}}_{\mathrm{a}})\, \psi(\mathbf{x}^{*}; \tilde{\mathbf{x}}_{\mathrm{a}}, \tilde{\mathbf{y}}_{\mathrm{a}})}{\iiint \hat{p}(\xi^{*}; \xi, \eta)\, \psi(\xi^{*}; \xi, \eta)\, d\xi^{*}\, d\xi\, d\eta}, \tag{7.6}$$

where $\hat{p}(\mathbf{x}_{\mathrm{a}}', \tilde{\mathbf{x}}_{\mathrm{a}}, \tilde{\mathbf{y}}_{\mathrm{a}})$ is the joint density of the parents and child family set after the transmission-segregation stage of (7.4) and $\psi(\xi^{*}; \xi, \eta)$ is a Gaussian selection function which can depend on both the child and parental phenotypes. Even for the specification

$$\psi(\xi^{*}; \xi, \eta) = \mathrm{c}(\xi^{*}), \tag{7.7}$$

where the selection is a function of only the child phenotype, the induced family distribution p^* of $(\mathbf{x}_a^*, \tilde{\mathbf{x}}_a, \tilde{\mathbf{y}}_a) = (\mathbf{x}_c', \tilde{\mathbf{x}}_a, \tilde{\mathbf{y}}_a)$ is ordinarily altered as calculated by (7.6). Even in this circumstance, the marginal distribution $p^*(\tilde{\mathbf{x}}_a, \tilde{\mathbf{y}}_a)$ of the parent couple $(\tilde{\mathbf{x}}_a, \tilde{\mathbf{y}}_a)$ is changed owing to the action of selection on the child in maturing to a young adult, especially if the vector phenotype involves a number of cultural components.

Alternative and/or additional transformations of cultural inheritance can be effected via the linear relationships of (7.4) or by invoking a further linear transformation of the form

$$\mathbf{x}_a' = R\mathbf{x}_a^* + U\tilde{\mathbf{x}}_a + V\tilde{\mathbf{y}}_a + \epsilon' \tag{7.8}$$

where R, U, V are again matrices which bear contrasts in the determination of the adult phenotype of the succeeding generation $\mathbf{x}_a'$.

The passage of the child $\mathbf{x}_c'$ to the adult stage $\mathbf{x}_a'$ via (7.8) embodies a mixture of effects contributed by the parents *and the child.* This may be particularly apt for cultural transmission in which both the parents and the child are involved without the selection imprint of the type (7.6), but in the presence of (7.8). The joint distribution of $(\mathbf{x}_a', \tilde{\mathbf{x}}_a, \tilde{\mathbf{y}}_a)$, because (7.8) entails a linear transformation, is certainly Gaussian where the marginal parental density $\tilde{p}(\tilde{\mathbf{x}}_a, \tilde{\mathbf{y}}_a)$ without (7.6) would coincide with that of (7.3). On the other hand, in the presence of (7.6) $p^*(\tilde{\mathbf{x}}_a, \tilde{\mathbf{y}}_a)$ generally differs from $\tilde{p}(\tilde{\mathbf{x}}_a, \tilde{\mathbf{y}}_a)$. The essential concept is that the transition from the child phase to adult stage can involve nonlinear interactions as in (7.6) and/or linear relationships as in (7.8). In this perspective the aspects of cultural transmission (as with our assortative mating mechanism) and natural selection forces embrace nonlinear effects depending on the parental types interacting with the child's type.

The details concerning the calculation of covariances and the existence and uniqueness establishing an equilibrium Gaussian distribution for the conglomerate $\{\mathbf{x}_c, \mathbf{x}_a, \mathbf{y}_c, \mathbf{y}_a\}$ emanate from the general analysis of Theorem I, Section 11.

With all the multivariate distributions accessible we can then compute spouse covariances, adult child covariances $\mathbf{x}_a$ with $\mathbf{x}_c$ for the same individual or for different individuals of the same or opposite sexes, say $\mathbf{x}_a$ with $\mathbf{y}_c$ and many other combinations; see Karlin (III).

8. A Phenotype Selection Model for Adopted Children

Our key idea is to regard *the undertaking of an adoption as a selection process* akin to that of mating preference, i.e., mating and adoption formations are analogous in that for mating two individuals are participating whereas with adoptions a spouse couple and a child are united. We formalize this approach; let $p^{(m)}(x)[p^{(f)}(y)]$

be the equilibrium phenotype distribution of the male (female) population. Consider a spouse couple of phenotype $\{\tilde{x}, \tilde{y}\}$ following a distribution law

$$\tilde{p}(x, y). \tag{8.1}$$

For definiteness, the x (or $\mathbf{x}$ when it is vector-valued) stands for the male spouse phenotype value and y for the female phenotype value.

Recall that $\tilde{p}(x, y)$ is the distribution induced by an appropriate mating selection function, as described in Section 5. Consider a male (child) in the population of phenotype z. It seems reasonable to assume that the potential adoptee is a representative child in the population. On the other hand, there is the less well-founded assumption implicit in (8.2) below that the probability density of a couple seeking an adoption is that of (8.1). We would expect that adoptive families are not representative of normal families, and in this sense, the formulation (8.2) should be construed as limited and serves only as an approximation. With this caveat, we assume that the meeting of a couple $\{\tilde{x}, \tilde{y}\}$ with a child of phenotype z, assuming random encounters, occurs with probability

$$\tilde{p}(x, y) p^{(m)}(z). \tag{8.2}$$

Paraphrasing the concept of selective matings, an encounter does not ensure an adoption, but rather $\psi(x, y; z)$ estimates the conditional probability that the adoption is established. (The adoption selection function can emphasize more the family component than the child's.) The joint density of a certified adoption of the couple $\{x, y\}$ of a male child of phenotype z is

$$q^*(x, y; z) = \frac{\tilde{p}(x, y)\, p^{(m)}(z)\, \psi(x, y; z)}{\iiint \tilde{p}(\xi, \eta)\, p^{(m)}(\zeta)\, \psi(\xi, \eta; \zeta)\, d\xi\, d\eta\, d\zeta}. \tag{8.3}$$

As usual, we stipulate that $\psi(x, y; z)$ is a Gaussian density, of the general form

$$\psi(x, y; z) = \exp\left[-\frac{1}{2K}\{f_1 x^2 + f_2 y^2 + f_3 z^2 + 2g_1 xy + 2h_1 xz + 2h_2 yz\}\right] \tag{8.4}$$

with associated matrix

$$C = \frac{1}{K}\begin{pmatrix} f_1 & g_1 & h_1 \\ g_1 & f_2 & h_2 \\ h_1 & h_2 & f_3 \end{pmatrix}. \tag{8.5}$$

When the preference determinations on adoption are symmetric between the spouses we may assume

$$f_1 = f_2, \qquad h_1 = h_2. \tag{8.6}$$

A special symmetric form of $\psi(x, y; z)$ in the spirit of (5.5) uses the adoption selection function

$$\psi(x, y; z) = \exp\left(-\frac{1}{2K}[(x-z)^2 + (y-z)^2]\right), \tag{8.7}$$

where

$$C = \begin{pmatrix} \frac{1}{K} & 0 & -\frac{1}{K} \\ 0 & \frac{1}{K} & -\frac{1}{K} \\ -\frac{1}{K} & -\frac{1}{K} & \frac{2}{K} \end{pmatrix}.$$

A nonsymmetric version assigning different degrees of influence for the two parents in the adoption process may prescribe

$$\psi(x, y; z) = \exp\left[-\frac{1}{2K}\{\rho_1(x-z)^2 + \rho_2(y-z)^2\}\right]. \tag{8.8}$$

In line with the general devlopments leading to (5.8), the density $q^*(x, y, z)$ of (8.3) is Gaussian with covariance matrix

$$C^* = \left[\begin{pmatrix} \tilde{\Sigma}^{-1} & & 0 \\ & & 0 \\ 0 & 0 & 1/v \end{pmatrix} + \begin{pmatrix} f_1 & g_1 & h_1 \\ g_1 & f_2 & h_2 \\ h_1 & h_2 & f_3 \end{pmatrix}\right]^{-1} \tag{8.9}$$

$$= \begin{pmatrix} v^*_{\mathrm{MM}} & c^*_{\mathrm{MF}} & c^*_{\mathrm{MA}} \\ c^*_{\mathrm{MF}} & v^*_{\mathrm{FF}} & c^*_{\mathrm{FA}} \\ c^*_{\mathrm{MA}} & c^*_{\mathrm{FA}} & v^*_{\mathrm{AA}} \end{pmatrix}. \tag{8.10}$$

The explicit evaluations of (8.10) in the circumstance of (8.6) are easily accessible (cf. Karlin, III). With C^* in hand, we can readily calculate the various covariances involving adoptees and other family members. We illustrate some cases under the sex-dependent symmetric transmission rule (3.6) and in the circumstance of (8.7) for a scalar trait.

Consider a biological male offspring for the spouse pair $(\tilde{x}^{(\mathrm{m})}, \tilde{x}^{(\mathrm{f})})$ whose trait value is determined by the linear form

$$x^{(\mathrm{m})} = \alpha\tilde{x}^{(\mathrm{m})} + \beta\tilde{x}^{(\mathrm{f})} + \epsilon. \tag{8.11}$$

The phenotype of the adoptee is, taking account of environmental and other external sources,

$$x_A = z + \epsilon_A \tag{8.12}$$

where the random residual contributions to the phenotype expressions ϵ and ϵ_A may be correlated. The transmission from adoptive parents to adoptive child reflecting cultural and other acquirements from infancy to adulthood can be incorporated in a nonlinear manner similar to our developments of Section 7 distinguishing two or several age classes.

Assuming for ease of exposition the means equal to zero, we have

$$E[x_A x^{(m)}] = \alpha E[\tilde{x}^{(m)} z] + \beta E[\tilde{x}^{(f)} z] + E[\epsilon\epsilon_A] = \alpha c^*_{MA} + \beta c^*_{FA} + \mathrm{Cov}(\epsilon, \epsilon_A)$$
$$\mathrm{Var}[x_A] = v^{(m)}_\infty + \mathrm{var}\ \epsilon_A\,, \tag{8.13}$$

while $v^{(m)}_\infty$ is the equilibrium variance of a regular male individual.

The qualitative and quantitative results for the kinship correlations and for twin correlation models differ from the evaluations following the traditional lines (e.g., of Rao *et al.*, 1976; Nance and Corey, 1977).

9. Evolution of a Quantitative Trait under Continual Sib-mating

In order to convey well the scope of the formulations and because historically (in 1971) I commenced these studies at the Weizmann Institute of Science, with quantitative genetical breeding programs in mind, I devote the present short section to the application of the concepts and methodology to a model of continual sib-mating.

Let $\mathbf{x}$ and $\mathbf{y}$ be two sib vector phenotypes following the distribution law $p_t(\mathbf{x}, \mathbf{y})$ of $2n$th-order covariance matrix S_t. They generate viable offspring with probability $\phi(\mathbf{x}, \mathbf{y}) = \exp[-(\mathbf{z}, \Psi\mathbf{z})]$, $\mathbf{z} = (\mathbf{x}, \mathbf{y})$, Ψ is a positive definite matrix, where $\phi(\mathbf{x}, \mathbf{y}) = \Phi(\mathbf{z})$ exercises a stabilizing selection force. The distribution of a viable sib pair is then

$$\tilde{p}_t(\mathbf{x}, \mathbf{y}) = \frac{p_t(\mathbf{x}, \mathbf{y})\,\phi(\mathbf{x}, \mathbf{y})}{\int p_t(\xi, \eta)\,\phi(\xi, \eta)\,d\xi\,d\eta} \tag{9.1}$$

whose covariance matrix is

$$\tilde{S}_t = (S_t^{-1} + \Psi)^{-1}.$$

Let the matrix T of order $2n \times 2n$ be the transmission-segregation operator

$$\mathbf{z}' = (\mathbf{z}', \mathbf{y}') = T\mathbf{z} + \epsilon \tag{9.2}$$

where ϵ is an independent residual (random environmental-mutational) contribution normally distributed with covariance matrix F. The recursion of the covariance over two successive generations is

$$S_{t+1} = T(S_t^{-1} + \Psi)^{-1}T' + F. \tag{9.3}$$

The recursion (9.3) resembles (4.4) but differs from the model with separate sexes in the following essential way. In the present line of descent for sib mating, S_t is a bona fide $2n \times 2n$ irreducible positive definite matrix whereas in (5.10) the two-sex covariance matrix

$$C_t = \begin{pmatrix} U_t^* & 0 \\ 0 & V_t^* \end{pmatrix}$$

is reducible. The contrasting features of the equilibrium distribution for the sib-mating scheme as against the two-sex model of Section 5 will be underscored in Karlin (VI), especially in the treatment of the parent–offspring and sib–sib covariances.

10. Summary and Discussion

In this work and the companion papers in this series we set forth dynamic models of a vector phenotypic trait subject to assortative mating (selective) forms, viability selection forces, environmental-mutational factors, and various parent–offspring segregation-transmission rules. The formulation is sufficiently flexible to incorporate age class effects, discrete and continuous characters, geographical variation, sex-dependent transmission, and asymmetry with respect to male and female offspring expression, relationships among family and pedigree structures, and aspects of family set sampling units. The components of the vector phenotype can involve physiological, anthropometric, behavioral, and cultural variables. Artificial selection programs for vector quantitative traits, mixed cultural-polygenic inheritance processes, interpretations of population genetic epidemiology surveys, and descriptions of ecological phenotype evolution are unified in one setting.

The three principal forces considered pertain to mating pattern, parental transmission, and nontransmissible environmental perturbations.

Assortative Mating Mechanisms

The essence in our formulation of the assortment process is that it acts as a differential selection force with respect to mating types. The establishment of a spouse pair relates to the frequency of enounters among male and female individuals of varying phenotypes mediated by a relative conditional probability law (induced by a mating-selection function) that the encounter culminates in reproduction. The mating-selection function depends on the phenotype expression of the potential mates. These can emphasize to varying degrees the different vector trait components. In this perspective, the contrasts between phenotype assortment and cultural assortment, or assortment based on social homogamy (as wealth status, religious customs, attitudes) merely concentrate the assortment selection mechanism on one or the other of the phenotype components.

A curious aspect of the assortative mating equilibrium model that occurs in most recent works, e.g., Rao *et al.* (1974, 1976, 1979), Cavalli–Sforza and Feldman (1979), and Cloninger *et al.* (1978), is the tacit assumption that the equilibrium variance of the male (and female) partner from a couple is the same as the variance of a typical male (and corresponding female) in the population at large. This is never the case under our assortative mating process except in the special circumstance of *pure* random mating. In our models the joint distribution of couple phenotypes generally induces a marginal distribution for the male (or the female) spouse with reduced variance compared to the corresponding population members at large.

The assortative mating formulation following Fisher postulates linear stationary relationships where usually a scalar phenotype is expressed linearly in terms of genotype, family environment (and associated variables that index the family environment), and independent error factors. The assortment correlations are assumed to be invariant over successive generations, which contrasts with our assortment mechanism in that our spouse correlations change in time.

We treat adoption practices (Section 8) similarly to assortative mating. Thus, adoption conforms to a selection (nonlinear) mechanism by way of a differential preference function that binds a couple with a potential adoptee. This approach again diverges from the methodology of linear structural equation models.

Transmission Rules

Our formulation allows sex dependence both with respect to the parental transmission and in the offspring expression, distinguishing it from the usual determinations that treat the sexes symmetrically. In a model with two age classes (Section 7), the cultural transmission scheme passing from the child to adult stage is promulgated in a nonlinear fashion.

Convergence Results (*Sections* 4 *and* 11)

It is perhaps remarkable that under the multivariate Gaussian model with all the influences and effects involved, and more generally with the two-sex structure allowing a quite general transmission-segregation scheme and varied mating patterns (nonlinear selective mating), stabilizing natural selection forces, mutation and migration pressures, and other environmental influences, there is practically always a *unique equilibrium covariance structure* Σ_∞ to which the population covariance matrix Σ_t (of generation t) converges *independent of the initial population composition.* The convergence to the unique limit takes place even with non-Gaussian initial conditions. The global convergence theorem to a *finite* limit is assured when the mating selection function is nonsingular, that is, where Ψ of (5.2) is positive definite.

The calculation of rates of approach to equilibrium in terms of the mating, transmission, and environmental parameters is of interest and this analysis is largely tractable. In fact, the rate of approach of Σ_t is always geometrically fast. These can have bearing on ascertaining rates of selection advance for controlled programs and may be pertinent to understanding many inherited correlated risk factors in certain diseases.

Further developments that allow the mating, transmission and environmental parameters to fluctuate systematically or randomly in time and space are covered in Section 13.

The existence of a *unique* stable equilibrium (as in the multivariate Gaussian models) entails less sensitivity to the model construction retaining "continuity" with respect to small perturbations of the model system. Accordingly, although restrictions to normality may be inappropriate in several cases of stabilizing selection, the qualitative results appear to be robust.

The behavior of the mean population phenotype μ_t is less predictable compared to the population covariance development of Σ_t, depending more finely on the balance of all the mating, transmission, and environmental characteristics. There are cases where μ_t may drift to ∞ yet Σ_∞ is persistently finite. The equilibrium mean population phenotype μ_∞ generally departs from the modal phenotype γ (see (5.2)). An explicit formula of $\mu_\infty - \gamma$ is available; see Eq. (12.1). It is noteworthy that where the environmental and mating forces are concordant, meaning that the components of $\mathbf{x}$ are affected in a "synchronized" manner by the various genetic and environmental forces (formally, R, S, Ψ, F commute), and provided there is no mean environmental trend, then $\mu_\infty = \gamma$. However, even without deterministic trends, but where the interaction among the mating, transmission, and environment covariance terms are not concordant, meaning that some or all of the matrices R, S, Ψ, and F (defined in Section 3) do not commute, then μ_∞ will tend to deviate from γ.

"*Genotypes vs Phenotypes*"

With respect to genotype–phenotype associations, the gene interactions determining the trait are complex and no clear mechanism is likely to be discerned. A distinction is made by several authors between genotype and phenotype components and cultural variables. The genotypic inheritance is often reflected by exact midparent transmission coupled to an added normal variate with variance equal to one-half of the phenotype variance which is supposed to reflect a within–family ("Mendelian segregation") variance term (cf. Cavalli–Sforza and Feldman, 1976, 1977). This tactic seems quite arbitrary, apparently paraphrasing the presumption of additive equal allelic independent loci effects and global linkage equilibrium. These assumptions are generally inconsistent in the presence of assortative mating or almost any kind of selection differentials, dominance deviations, and other demographic and geographical influences in operation.

The nature and/or level of representation of phenotype observables in linear equation models in terms of "genotype," "dominance," "epistatic," and "family environmental" variables is difficult to assess. In our formulation, there are only transmissible phenotypes to varying degrees subject to environmental (non-transmissible) perturbations. The "unobservable" variables such as genotype, dominance, epistatic components are blurred, and probably only meaningful in one-locus contexts.

Alternative Multilocus Approaches for Polygenic Inheritance

An appropriate theoretical model taking account of interactive loci effects, linkage relationships, and other genetic mechanisms has not been duly treated as yet. We propose as a more natural approach in order to unravel the nature of polygenic inheritance and its mechanisms the framework of multilocus interactions. It is meaningful to envision genotypic–phenotypic associations based on 2 to 10 loci contributions coupled to forms of partial expressivity and random-environmental terms as a basis for phenotype expressions. Classifications and characterizations relevant to the dynamics and equilibrium behavior of n-locus theory incorporating components of selection-recombination events, nonrandom mating patterns, and facets of population structure are increasingly amenable to theoretical analyses, appropriate numerical simulation, and suggestive interpretations, see Karlin (1977, 1978a, 1979a), Karlin and Liberman (1979).

Perspectives and Preview

We develop in Karlin (II) and (IV) a detailed analysis of the qualitative properties of the equilibrium covariance structure of a scalar trait for a two-sex population allowing contrasting asymmetric transmission forms and general assortative mating patterns. In Karlin (III), kinship covariance calculations are done on a number of first and second cognate and affine relatives. The effects of selective mating and asymmetric transmission rules are particularly stressed. Contrasts are emphasized between sib–sib and parent–offspring covariances and also for more elaborate pedigree member covariance expressions. The method of computing kinship covariances of half-sibs, cousins, grandparent-child, etc., depends, as usual, on ascertaining the joint distribution of the relatives involved. However, in our approach the calculations depend on a selection mechanism extending the scheme of assortative mating to include more relatives.

The sixth paper in this series develops bounds and establishes various monotonicity properties of the multivariate equilibrium covariance matrix $\Sigma_\infty(T, \Gamma, \Psi, F)$ seeking to elucidate their dependence on the transmission (T), viability selection (Γ), mating selection (Ψ), and environmental (F) variance parameters.

With analytical modeling one should attempt a complete classification of

the formal structures elucidating functional relations between parameter specifications of the pertinent biological and environmental factors and the resulting possible observables. The solution of the model will rarely be quantitatively applicable, but may help provide a deeper qualitative conceptual basis for the interpretation of part of the findings, as well as in stimulating the intuition and imagination for further studies. Mathematical models can at their best reveal the principal potentialities at a given level and uncover their limitations. The analysis of models is in essence an educating process rather than an engineering construction. The qualitative information furnished by relevant analytical models meshed with the intimate experience on the empirical side is more likely to suggest suitable indices or functions for estimation purposes and the use of appropriate descriptive statistics in the interpretation of the data.

Limitations

It is now appropriate to list a number of the limitations, caveats, and cautionary notes concerning the conclusions derived from our models.

(1) Although a general formulation of processes pertaining to multifactorial inheritance is set forth in Section 2 the principal tractable case assumes multinormal distributions. It is recognized that many traits are intrinsically nonnormal. For example, the amount of phenylalanine in the blood plasma is bimodal (Penrose, 1952). Head size (Penrose, 1952) displays unimodal histograms, but diverges from normality. Many epidemiological data sets manifest strong skewness, kurtosis, and nonnormal higher moments, and the normality assumption may be inappropriate, e.g., weight, triglyceride levels, and glucose tolerance rates. Procedures using a power or log transformation on the data (which is intrinsically heterogeneous or likely comes from mixtures of distributions), purporting to remove skewness and thereby achieve approximate normality, are quite moot. The prospect of transforming measurements to a Gaussian state is generally not feasible for collections of random variables. The temptation to transform each component variable separately to normality will generally corrupt the basic correlation structure and obscure the dependence relations among the variables. With categorical data it is even less natural and here probably quantal regression forms are more relevant. Other trait expressions involve intrinsically constrained variables where the Gaussian model in this context is likely unsuitable. The modeling of some cases of nonnormal phenotypic inheritance is dealt with in Karlin (VII, VIII).

(2) A serious limitation in most of the modeling is the linear parent–offspring transmission structure. An important modification seeks to stipulate a nonlinear function of the key variables and/or parameters. We have proposed some nonlinear transmission forms for cultural transmission in our treatment of the model with two age classes (Section 7), which should be regarded as only a first step in this direction.

(3) Our models do incorporate extended family formation and mate selection mechanisms conforming with population genetic concepts of sexual selection and selection differentials over mating types. Unfortunately, in these constructions of phenotypic interactions the genetics is minimal. This is a significant lacuna in all treatments of multifactorial inheritance to date. We have discussed earlier some possibilities with multilocus theory as a more natural structure in order to refine our understanding of polygenic inheritance and more easily and accurately incorporate epistasis, linkage relationships, and variable expressivity for the gene components.

(4) We studied a model of multifactorial inheritance where the sampling unit is a nuclear family of a fixed sibship size. We do not know how to handle the more realistic case allowing variable family sizes and other family structure characteristics (e.g., age distribution of children, the role of sex ratio among siblings, other relatives living in the household). Stratification of variables usually induces mixtures of Gaussian distributions which are no longer Gaussian. New classes of models and techniques are needed here.

(5) In dealing with the comparisons of adopted versus natural children (Section 8) a severe limitation in this discussion is the lack of a reasonable characterization of adoptive families. Similar problems arise in attempting to model and exploit data on sibs reared apart or together.

(6) A number of problems connected with kinship covariance calculations are presented in Karlin (III).

(7) We have been somewhat casual in lumping cultural, physiological, behavioral, and anthropometric variables as part of a general vector trait $\mathbf{x}$. Their transmissibility characteristics are not similar and the environmental influences can be delicate concomitants and confounders. The statistical problems are formidable if not prohibitive.

(8) The whole gamut of our models and its methodology accents departures from linearity in dealing with family formation processes, adoption practices, and cultural transmission forms and may be useful as a further approach in studying some econometric and sociological systems. Maybe some of these ideas can be modified and adapted to lend insights into the nature of distribution of earnings, education processes, etc. The problems are certainly never resolvable in analytical terms. It is our belief that no all-purpose strategy, when it comes to empirical research, exists, but each problem needs several reinforcing or counteracting approaches, including a thorough mix of modeling and empiricism.

The evolutionary polemics at the close of the last century were concerned mostly with continuously distributed traits. With the rediscovery of Mendel the focus turned to discrete single or a few gene-induced traits. Intensive effort has been increasingly devoted to the study of multilocus interactions starting with

the decade of the 1960's. Population genetics theory into the last decade has come full circle, engaging with new vigor the description and analysis of the transmissibility characteristics innate to continuous phenotypic variation over space and time.

11. The Principal Convergence Theorems

To ease the exposition, we take $R = S$ in (4.4) and use the notation $T = 2^{1/2}R$. The recurrence relations (4.4) and (4.5) have the forms

$$\Sigma_{t+1} = f(\Sigma_t), \tag{11.1}$$

where

$$f(\Sigma) = T(\Sigma^{-1} + \Gamma^{-1})^{-1}T' + F, \tag{11.2}$$

defined for Σ positive definite, and

$$\mu_{t+1} = T(\Sigma_t^{-1} + \Gamma^{-1})^{-1}(\Sigma_t^{-1}\mu_t + \Gamma^{-1}\gamma) + \phi.$$

To simplify the analysis we stipulate $\Gamma + F$ positive definite. With mating selection in place of viability selection replace Φ for Γ throughout. The following result of wide scope and utility is proved in Appendix A.

Result I.

(a) *The equation* $f(\Sigma) = \Sigma$ ($f(\Sigma)$ *defined in* (11.2)) *has a unique positive definite solution* Σ_∞ *and from any initial* Σ_0, $\lim_{t\to\infty} \Sigma_t = \Sigma_\infty$.

(b) *Moreover, the matrix-valued function* $f(\Sigma)$ *is strictly increasing and concave, meaning that if*

$$\Sigma \leqslant \Sigma^* \quad \textit{(the ordering defined for symmetric matrices signifies that } \Sigma^* - \Sigma \textit{ is positive semidefinite and positive definite when we write } \Sigma^* > \Sigma\textit{)}, \tag{11.3}$$

then

$$f(\Sigma^*) > f(\Sigma) \quad \textit{and} \quad f\left(\frac{\Sigma^* + \Sigma}{2}\right) > \frac{f(\Sigma^*) + f(\Sigma)}{2} \quad \textit{provided } \Sigma^* \neq \Sigma. \tag{11.4}$$

We next highlight the limit value of μ_t.

Result II. *We denote the spectral radius of a matrix* C *by* $\rho(C)$. *Set* $P_\infty = T(\Sigma_\infty^{-1} + \Gamma^{-1})^{-1}\Sigma_\infty^{-1}$. *If the spectral radius of* P_∞ *satisfies* $\rho(P_\infty) < 1$, *then the existence of* $\lim_{t\to\infty} \mu_t = \mu_\infty$ *prevails with*

$$\mu_\infty = (I - P_\infty)^{-1}[T(\Sigma_\infty^{-1} + \Gamma^{-1})^{-1}\,\Gamma^{-1}\gamma + \varphi]. \tag{11.5}$$

If $\Gamma > 0$ *and* $\rho(TT') \leqslant 1$ *then* $\rho(P_\infty) < 1$.

Remark 11.1. (i) The result of (11.5) clearly holds if the norm of T obeys $\| T \| \leqslant 1$ and $\Gamma > 0$. (ii) The condition $TT' \leqslant I$ is present, for example, if T is a doubly stochastic matrix. (iii) Refinements on the convergence results will be elaborated in Sections 13 and 14.

Observe that we need to impose growth restrictions on the norm of T in order to guarantee the convergence of μ_t which was unnecessary in establishing the convergence of the of covariance matrices Σ_t.

Where $F = 0$ such that the mutation and environmental influences are deterministic (or nonexistent) and $T = I$ (e.g., hapoid reproduction or uniparental transmission), then Σ_t converges to 0, signifying that the limiting phenotype value behaves deterministically. Further study of the progress of μ_t in this case reveals often $\| \mu_t \| \to \infty$, indicating that the deterministic environmental influence when extant overwhelms the stabilizing fitness effects.

It is of interest to determine the population dynamics when no random-environmental (residual) is involved. Accordingly, we consider the reduced dynamic relation of (11.2) when $F = 0$, yielding

$$\Sigma_{t+1} = T(\Sigma_t^{-1} + \Gamma^{-1})^{-1} T' \tag{11.6}$$

(assuming for ease of exposition T nonsingular). This is equivalent to the linear recursion in $\Sigma_t^{-1} = W_t$ of the form

$$W_{t+1} = T'^{-1} W_t T^{-1} + \hat{\Gamma}, \tag{11.7}$$

where $\hat{\Gamma} = (T'^{-1}\Gamma^{-1}T^{-1})$. The analysis of (11.7) is classical and we find that

W_t converges to a finite nonzero covariance if and only if $\rho(T'^{-1}T^{-1}) < 1$ *or equivalently if and only if all the eigenvalues of* $T'T$ *exceed* 1. (11.8)

We can sum up the preceding in the following result.

RESULT III. *With no random-environmental-mutational variance,* $F = 0$, *the equilibrium population covariance* $\Sigma_\infty = 0$ *unless all the eigenvalues of* $T'T$ *exceed* 1. *In the latter event, i.e., when the principal eigenvalue* $\rho((TT')^{-1}) < 1$, *a balance between selection and transmission is established entailing* $\Sigma_\infty > 0$ *as the unique solution of the equation* $\Sigma_\infty = T(\Sigma_\infty^{-1} + \Gamma^{-1})^{-1}T'$.

12. The Deviation of the Mean Population Phenotype from the "Optimal" Phenotype

It is of some interest to assess the departure of the equilibrium phenotypic mode μ_∞ from the optimal selection mode located at γ (see (4.1)). Recall the explicit formula (11.5):

$$\mu_\infty = (I - P_\infty)^{-1}[T(\Sigma_\infty^{-1} + \Gamma^{-1})\Gamma^{-1}\gamma + \varphi], \tag{12.1}$$

where $P_\infty = T(\Sigma_\infty^{-1} + \Gamma^{-1})^{-1}\Sigma_\infty^{-1}$ We are now concerned with the magnitude of

$$\| \mu_\infty - \gamma \| \tag{12.2}$$

evaluated in any suitable metric.

In the context of preferential mating expressed by fertility selection acting on certain mating types (see (5.2)) such that

$$\phi(\mathbf{z}) = \exp[-(1/2)((\mathbf{z} - \gamma, \Psi(\mathbf{z} - \gamma)))],$$

the mode γ can be interpreted as the socially optimal couple phenotype.

From careful scrutiny of (12.1) we see that the mean random environmental-mutational translation endowed to the vector φ, if nonzero, compels a deviation of μ_∞ from γ. However, even if $\varphi = \mathbf{0}$, then generally (12.2) is nonzero. More precisely, where T, Γ, and F commute (as in the one-dimensional case or when these effects are properly "synchronized"), note that $(I - P_\infty) = T(\Sigma_\infty^{1-} + \Gamma^{-1})\Gamma^{-1}$ and then formula (12.1) reduces to $\mu_\infty = \gamma + (I - P_\infty)^{-1}\varphi$. Then, as long as $\varphi = \mathbf{0}$ and even in the presence of environmental and natural and/or sexual selection variance, we see that μ_∞ coincides with the optimal fitness mode. *On the other hand, even with* $\varphi = 0$, *but where* T, Γ, *and* F *do not commute* (*i.e., the interactions expressed in these forces are not consonant in phase and direction*) *then* μ_∞ *will generally deviate from* γ. For $T = I$ and $\varphi = \mathbf{0}$ then always $\mu_\infty = \gamma$.

The Existence of a Finite Mean Equilibrium Phenotype

Recall that $\| \mu_\infty \| < \infty$ holds if $\rho(P_\infty) < 1$ (Result II), signifying that the spectral radius of P_∞ is less than 1. In particular, the existence of μ_∞ holds if T is doubly stochastic. However, if the segregation mechanism of T engenders excessive dispersion in parent–offspring phenotypic transmission, then μ_∞ does not exist, yet in sharp contrast Σ_∞ is always uniquely determined. For the contingency of $\| \mu_\infty \| = \infty$, the centered phenotype variable $X_t - \mu_t$ invariably settles as $t \to \infty$ to the normal density $N(\mathbf{0}, \Sigma_\infty)$. Thus, with weak selection and strong parental transmissible overloading, the mean phenotype tends appropriately to ∞, although the accompanying variability (covariance structure) of the process is predictable.

13. The Vector Phenotype Model with Genetic and Environmental Parameters Varying Systematically or Randomly in Time

The changes over successive generations in the phenotypic covariance matrix for the phenotypic model of (Section 5) involving preference mating (sexual selection and/or viability selection), mutation-environmental perturbations, and a parental transmission matrix T is described by the recursion

$$\Sigma_{t+1} = f(\Sigma_t) = T(\Sigma_t^{-1} + \Gamma^{-1})^{-1} T' + F. \tag{13.1}$$

We know by Result I that Σ_t converges to Σ_∞, the unique equilibrium phenotype covariance matrix associated with the vector phenotype trait.

It is relevant to examine the validity of an equilibrium phenotype covariance matrix when the underlying genetic and environmental forces change in time. We report results on three aspects of this problem.

A. *Systematic Variation in the Forces*

Consider the model leading to (11.2) or (4.4) where the preference mating determination $\Phi(\mathbf{x}, \mathbf{y})$ characterized by the matrix Γ, the total environmental variance F, and parental transmission form T vary in time such that

$$\begin{aligned} \Gamma_t &\to \Gamma^* \\ F_t &\to F^* \qquad \text{as} \quad t \to \infty. \\ T_t &\to T^* \end{aligned} \tag{13.2}$$

The relations (13.2) cover the situation of a systematic temporal trend in the basic forces tending to the limits as indicated.

In this context the transformation equation of the population phenotype covariance over successive generations is adjusted to

$$\Sigma_{t+1} = T_t(\Sigma_t^{-1} + \Gamma_t^{-1})^{-1} T_t' + F_t . \tag{13.3}$$

Exploiting the innate monotonicity properties of $f(\Sigma)$ (see Result I and Appendix A) we again can confirm the limit relation

$$\Sigma_t \to \Sigma_\infty , \tag{13.4}$$

where Σ_∞ uniquely satisfies

$$\Sigma = T^*(\Sigma^{-1} + \Gamma^{*-1})^{-1} T^{*\prime} + F^* . \tag{13.5}$$

Accordingly, all the influences operating on the limiting phenotype covariance matrix as determined by the model forces $\{T_t, F_t, \Gamma_t\}$ translate into its dependence on T^*, Γ^* and F^* where $\Sigma_\infty(T^*, \Gamma^*, F^*)$ and its properties and operations are as elaborated previously; see Karlin (VI).

B. *Periodic (as Seasonal) Variations in Genetic and Environmental Forces*

Consider the basic model subject to a cycle of forces with parameter specifications as follows

$$\begin{gathered} \Gamma_{2t} = \Gamma_2 , \quad \Gamma_{2t+1} = \Gamma_1 , \qquad F_{2t} = F_2 , \quad F_{2t+1} = F_1 , \\ T_{2t} = T_2 , \quad T_{2t+1} = T_1 , \qquad t = 1, 2, 3, \ldots . \end{gathered} \tag{13.6}$$

Thus, subject to (13.6), every other generation is influenced by the same array of forces (a grandparent–grandchild concurrence). These cyclic possibilities may partly reflect psychological and physiological factors that cause "generation gaps." The flexibility in our analysis, of course, allows us to treat periodicity of any prescribed order.

The analog of (13.1) in the context of (13.6) connects the population phenotype covariance over two generations in the manner that

$$g(\Sigma) = f_2(f_1(\Sigma)), \tag{13.7}$$

where

$$f_i(\Sigma) = T_i(\Sigma^{-1} + \Gamma_i^{-1})^{-1} T_i' + F_i, \qquad i = 1, 2.$$

Again, we deduce for $t \to \infty$, convergence to the pair of equilibrium covariance matrices

$$\Sigma_{2t} \to \Sigma^*, \Sigma_{2t+1} \to \Sigma^{**}, \qquad t \to \infty$$

effective with period 2.

C. *Random Temporal Fluctuations of the Parameters*

To ease the discussion we consider the one-dimensional (numerical trait) model. The recursion of (13.1) becomes, with $T = \alpha^{1/2}$,

$$\begin{aligned} v_{t+1} &= \frac{\alpha}{((1/v_t) + (1/\gamma_t))} + f_t \\ v_{t+1} &= \frac{v_t(\alpha\gamma_t + f_t) + \gamma_t f_t}{v_t + \gamma_t}. \end{aligned} \tag{13.8}$$

It is convenient to introduce the auxiliary variable w_t, writing

$$\frac{v_{t+1}}{w_{t+1}} = \frac{(v_t/w_t)(\alpha\gamma_t + f_t) + \gamma_t f_t}{(v_t/w_t) + \gamma_t}. \tag{13.9}$$

The recursion (13.9) can be equivalently expressed by the linear system

$$\begin{aligned} \tilde{v}_{t+1} &= (\alpha\gamma_t + f_t)\,\tilde{v}_t + \gamma_t f_t \tilde{w}_t \\ \tilde{w}_{t+1} &= \tilde{v}_t + \gamma_t \tilde{w}_t \end{aligned} \tag{13.10}$$

and consequently in the vector-matrix form

$$\begin{pmatrix} \tilde{v}_{t+1} \\ \tilde{w}_{t+1} \end{pmatrix} = \begin{pmatrix} \alpha\gamma_t + f_t, \gamma_t f_t \\ 1, \gamma_t \end{pmatrix} \begin{pmatrix} \tilde{v}_t \\ \tilde{w}_t \end{pmatrix}. \tag{13.11}$$

When the matrices

$$U_t = \begin{pmatrix} \alpha\gamma_t + f_t, \gamma_t f_t \\ 1, \gamma_t \end{pmatrix} \tag{13.12}$$

have random independently distributed entries over successive generations, then the important theory concerning the product of random matrices (e.g., Furstenburg and Kesten, 1960; Furstenburg, 1963; Kingman, 1973) on iteration of random mixing transformations reduces to

$$\begin{pmatrix} \tilde{v}_{t+1} \\ \tilde{w}_{t+1} \end{pmatrix} = (U_t U_{t-1} U_{t-2} \cdots U_1) \begin{pmatrix} \tilde{v}_0 \\ \tilde{w}_0 \end{pmatrix}. \tag{13.13}$$

It is known (loc. cit.) that

$$U_t U_{t-1} \cdots U_1 \sim e^{\pi_t} A \tag{13.14}$$

where π_t is a constant that grows to ∞ and A is a fixed positive random matrix governed by a discernible distribution. It follows on the basis of (13.14) that

$$v_{t+1} = \frac{\tilde{v}_{t+1}}{\tilde{w}_{t+1}} \qquad \text{possesses a limiting distribution.} \tag{13.15}$$

The multidimensional extensions of (13.8) are probably also valid.

14. APPENDIX A: The Monotonicity and Concavity of $f(\Sigma)$ Defined in (11.2) and Proof of Result I of Section 11

The following analysis extends to a wide range of matrix-valued transformations including

$$f(\Sigma) = ((R\Sigma R' + S\Sigma S')^{-1} + \Gamma^{-1})^{-1} + F$$

and

$$f(\Sigma) = ((T(\Sigma^{-1} + \Phi^{-1})^{-1}T' + F)^{-1} + \Gamma^{-1})^{-1},$$

defined for Σ positive definite where Γ, F, and Φ are positive definite. Recall the function $f(\Sigma)$ from (11.2)

$$f(\Sigma) = T(\Sigma^{-1} + \Gamma^{-1})^{-1}T' + F.$$

It is a familiar property that if

$$0 < \Sigma_1 \leqslant \Sigma_2 \text{ , i.e., } \Sigma_2 - \Sigma_1 \text{ is positive semidefinite and } \Sigma_1 \text{ is positive definite} \tag{A.1}$$

then

$$\Sigma_1^{-1} \geqslant \Sigma_2^{-1} \qquad \text{and strict inequality holds unless} \quad \Sigma_1 = \Sigma_2 .$$

Applying this fact twice we deduce straightforwardly that $f(\Sigma)$ is strictly increasing.

For A a symmetric matrix and small (in any norm), we have

$$f(\Sigma + A) - f(\Sigma) = f'(A) + \tfrac{1}{2}f''(A) + O(\| A \|^3), \tag{A.2}$$

where $f'(A)$ is a linear matrix valued mapping and $f''(A)$ is a quadratic matrix map, viz.,

$$\begin{aligned} f'(A) &= TLAL'T' \qquad \text{where} \quad L = (\Sigma^{-1} + \Gamma^{-1})^{-1}\Sigma^{-1} \\ f''(A) &= 2T[(-1)LA\Sigma^{-1}AL' + LA\Sigma^{-1}LAL']\,T' \end{aligned} \tag{A.3}$$

and $O(\| A \|^3)$ is an anlytical matrix function in A of the order $\| A \|^3$.

We establish now the Taylor expansion as displayed in (A.2). Let A be a symmetric matrix of small norm. Consider

$$(\Sigma + A)^{-1} = \Sigma^{-1/2}(I + \Sigma^{-1/2}A\Sigma^{-1/2})^{-1}\Sigma^{-1/2}.$$

Set $U = \Sigma^{-1/2}A\Sigma^{-1/2}$ and writing the Neumann series for $(I + U)^{-1}$ gives

$$\begin{aligned} (\Sigma + A)^{-1} &= \Sigma^{-1/2}\left(\sum_{n=0}^{\infty} U^n(-1)^n\right)\Sigma^{-1/2} \\ &= \Sigma^{-1} - \Sigma^{-1}A\Sigma^{-1} + \Sigma^{-1}A\Sigma^{-1}A\Sigma^{-1} + O(\| A \|^3) \\ &= \Sigma^{-1} + Z + O(\| A \|^3) \end{aligned}$$

where $O(\| A \|^3)$ represents a matrix of the order $\| A \|^3$. (We have simplified the notation with $Z = -\Sigma^{-1}A\Sigma^{-1} + \Sigma^{-1}A\Sigma^{-1}A\Sigma^{-1}$.) A completely analogous expansion for $(\Sigma^{-1} + \Gamma^{-1} + Z)^{-1}$, setting $K = \Sigma^{-1} + \Gamma^{-1}$, produces

$$(\Sigma^{-1} + \Gamma^{-1} + Z)^{-1} = K^{-1} - K^{-1}ZK^{-1} + K^{-1}ZK^{-1}ZK^{-1} + O(\| Z \|^3).$$

Substituting for Z yields the expansion (A.3).

We claim that

$$f''(A) < 0, \qquad \text{i.e., } f''(A) \text{ is strictly negative definite if } A \text{ is nonsingular symmetric.} \tag{A.4}$$

Manifestly the validation of the foregoing assertion reduces to proving

$$\Sigma^{-1} > \Sigma^{-1}L \qquad \text{where} \quad L = (\Sigma^{-1} + \Gamma^{-1})^{-1}\Sigma^{-1}. \tag{A.5}$$

Observe that $\Sigma^{-1}L = \Sigma^{-1}(\Sigma^{-1} + \Gamma^{-1})^{-1}\Sigma^{-1}$ is positive definite so that the comparison (A.5) is well defined. We next consider (A.5). Let O be orthogonal

such that $O'\Sigma^{-1}O = \Lambda =$ a positive diagonal matrix having $(\lambda_1, ..., \lambda_n)$ down the diagonal. Then for any nontrivial vector $\mathbf{x}$, we have the inner product value

$$((O'\Sigma^{-1}O\mathbf{x}, \mathbf{x})) = ((\Lambda\mathbf{x}, \mathbf{x})) = \sum_{i=1}^{n} \lambda_i x_i^2.$$

Moreover,

$$O'\Sigma^{-1}(\Sigma^{-1} + \Gamma^{-1})^{-1}\Sigma^{-1}O = \Lambda(\Lambda + O'\Gamma^{-1}O)^{-1}\Lambda,$$

and therefore, since $O'\Gamma^{-1}O$ is positive definite, we have

$$((O'\Sigma^{-1}LO\mathbf{x}, \mathbf{x})) = ((\Lambda(\Lambda + O'\Gamma^{-1}O)^{-1}\Lambda\mathbf{x}, \mathbf{x})) < \sum_{i=1}^{n} \lambda_i x_i^2 \qquad \text{(A.6)}$$

and (A.5) ensues and thereby (A.4) is established.

Since the second variation of $f(A)$ is strictly negative for A nonsingular, it follows that $f(A)$ is *strictly concave*.

By appeal to the theory of concave monotone operators (Krasnoselskii, 1964), the assertions of Result I, part (a), follow.

The proof of convergence when $T = I$ (or a multiple of the identity as with midparental transmission) of the recursion

$$\Sigma_{t+1} = (\Sigma_t^{-1} + \Gamma^{-1})^{-1} + F \qquad \text{(A.7)}$$

is more accessible employing a simultaneous diagonalization of the positive definite matrices Γ and F. Actually, the procedure described leads to an explicit representation to the extent that eigenvalues and eigenvectors are computable.

Define $V_t = B^{-1}\Sigma_t B^{-1}$ where $B = \Gamma^{1/2}$ is the positive square root of Γ. By multiplying on the right and left by B^{-1}, the relation (A.7) is converted into

$$V_{t+1} = (V_t^{-1} + I)^{-1} + G, \qquad t = 0, 1, 2, ... \qquad \text{(A.8)}$$

where $G = B^{-1}FB^{-1}$. We will prove that V_t converges to

$$V_\infty = \frac{G + (G^2 + 4G)^{1/2}}{2}$$

for any initial $V_0 > 0$. There exists $\epsilon > 0$ sufficiently small and $k > 0$ sufficiently large such that $\epsilon I < V_0 < kI$. (It suffices to take $\epsilon <$ min$\{\lambda \mid \lambda$ an eigenvalue of $V_0\}$ and $k >$ maximum of such eigenvalues. By virtue of the monotonicity of $f(A)$ the ordering $V_t \leqslant V_t^*$ entails $V_{t+1} \leqslant V_{t+1}^*$. The iteration of (A.8) can be performed if $V_0^* = cI$ as only matrices commuting with G are involved and then (A.8) can be considered in its diagonal canonical form. We find that the limit is V_∞ independent of the constant c. Since V_t is

captured in the sense of the ordering of positive definite matrices between two sequences converging to V_∞ of (A.9) it follows that $V_t \to V_\infty$ as claimed. The convergence of Σ_t to Σ_∞ follows directly from that of V_t converging to V_∞.

15. APPENDIX B: Discussion of Result II of Section 11

It is necessary to analyze the recursion formula (4.5) which we write in the compact notation

$$\mu_{t+1} = P_t\mu_t + Q_t\gamma + \phi = P_t\mu_t + \epsilon_t, \tag{B.1}$$

where

$$\begin{aligned} P_t &= T(\Sigma_t^{-1} + \Gamma^{-1})^{-1}\Sigma_t^{-1}, \\ Q_t &= T(\Sigma_t^{-1} + \Gamma^{-1})^{-1}\Gamma^{-1}, \end{aligned} \tag{B.2}$$

and

$$\epsilon_t = Q_t\gamma + \phi.$$

Obviously $P_t \to P_\infty$ and $Q_t \to Q_\infty$ since $\Sigma_t \to \Sigma_\infty$ by Result I (see Appendix A). Moreover, the spectral radius of P_∞ is less than 1 by assumption.

We need the following lemma.

Lemma B.1. *Under the conditions of Result II, then for all t large enough, we have the norm estimate*

$$\| P_t'P_t \| \leqslant \lambda^2 < 1, \qquad \textit{for some } \lambda, \quad 0 < \lambda < 1. \tag{B.3}$$

Proof. It suffices to establish (B.3) for $t = \infty$. Recall the notation $L = (\Sigma_\infty^{-1} + \Gamma^{-1})^{-1}\Sigma_\infty^{-1}$ of (A.5). Consider

$$((P_\infty'P_\infty\mathbf{x}, \mathbf{x})) = ((L'T'TL\mathbf{x}, \mathbf{x})) \leqslant ((L'L\mathbf{x}, \mathbf{x}))$$

the last inequality resulting since $I - T'T \geqslant 0$. But the analysis of (A.6) of Appendix A shows that $I - L'L$ is strictly positive definite and therefore $((L'L\mathbf{x}, \mathbf{x})) \leqslant \lambda^2((\mathbf{x}, \mathbf{x}))$ for some $0 < \lambda < 1$.

Corollary B.1. *The spectral radius of P_∞ is less than $\lambda < 1$.*

In fact, let the eigenvalue of the largest magnitude be ρ^2 and a corresponding normalized eigenvector be $\mathbf{z}$, $\| \mathbf{z} \| = 1$. Then

$$\rho^2 = \| P_\infty\mathbf{z} \|^2 = ((P_\infty'P_\infty\mathbf{z}, \mathbf{z})) \leqslant \lambda^2.$$

The first step in the developement of Result II concerns the proof of the fact that $\| \mu_t \|$ is bounded independent of t.

Manifestly, $\| \epsilon_t \| \leqslant C$ since the operators Q_t $(\to Q_\infty)$ are bounded. From (B.1)

$$((\mu_{t+1}, \mu_{t+1})) = \| \mu_{t+1} \|^2 = ((P_t\mu_t, P_t\mu_t)) + 2((P_t\mu_t, \epsilon_t)) + \| \epsilon_t \|^2 \qquad (B.4)$$

and for t large enough, $t \geqslant t_0$, in view of Lemma B.1, we have

$$\begin{aligned} &\leqslant \lambda^2 \| \mu_t \|^2 + 2C\lambda \| \mu_t \| + C^2 \\ &\leqslant (\lambda \| \mu_t \| + C)^2, \end{aligned}$$

or equivalently,

$$\| \mu_{t+1} \| \leqslant \lambda \| \mu_t \| + C, \qquad t \geqslant t_0, \quad 0 < \lambda < 1. \qquad (B.5)$$

We find that $\| \mu_t \| \leqslant C/(1 - \lambda)$, for all $t \geqslant t_0$ and consequently $\| \mu_t \|$ is uniformly bounded.

We are now prepared to establish convergence of μ_t. To this end, it is convenient to rewrite (B.1) in the form

$$\mu_{t+1} = P_\infty\mu_t + Q_\infty\gamma + \phi + \delta_t \qquad (B.6)$$

where $\delta_t = (P_t - P_\infty)\mu_t + (Q_t - Q_\infty)\gamma$.

Since $P_t \to P_\infty$, $Q_t \to Q_\infty$, and $\| \mu_t \|$ is uniformly bounded, it follows that for any prescribed positive $\delta > 0$ there exists t large enough, say $t \geqslant t_0$ with the property $\| \delta_t \| \leqslant \delta$. Then iteration of (B.6) leads to

$$\mu_{t_0+n} = P_\infty{}^n\mu_{t_0} + \sum_{k=0}^{n} P_\infty{}^k(Q_\infty\gamma + \phi) + \sum_{k=0}^{n} P_\infty{}^k\delta_{t_0+n-k-1}. \qquad (B.7)$$

Since the spectral radius $\rho = \rho(P_\infty) < 1$, then $\| P_\infty{}^k \| \leqslant \rho^k$ for all $k \geqslant 0$, and as $\| \delta_t \| \leqslant \delta$ for all $t \geqslant t_0$ and plainly $\sum_{k=0}^{\infty} P_\infty{}^k = (I - P_\infty)^{-1}$, we deduce standardly

$$\overline{\lim_{n\to\infty}} \| \mu_{t_0+n} - (I - P_\infty)^{-1}(Q_\infty\gamma + \phi)\| \leqslant C_1\delta, \qquad C_1 = \sum_{k=0}^{\infty} \| P_\infty{}^k \|. \quad (B.8)$$

As δ can be made arbitrarily small, (11.5) obtains.

The proof of Result II is complete.

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