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Mathematical Theory of Phenotypical Selection

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A general concept of phenotypical structure over a genotypical structure is developed. The direct decompositions of multilocus phenotypical structures are considered. Some aspects of phenotypical heredity are described in terms of graph theory. The acyclic phenotypical structures are introduced and studied on this base. The evolutionary equations are adjusted to the phenotypical selection. It is proved that if a phenotypical structure is acyclic then the set of fixed points of the corresponding evolutionary operator is finite except for a proper algebraic subset of the operator space. Some applications of this theorem are given. © 2001 Academic Press

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1. INTRODUCTION

We develop a general mathematical scheme of relations between the genotype (the set of genes of an individual) and the phenotype (the set of observed characteristics), see [4; 5, Sect. 1.1]. Our main goal is to study the evolution of the genotypical probabilities in a population under selection based on a phenotypical structure. The latter is formally defined as

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the triple (Z, Φ, φ), where Z is the set of all considered genotypes in a population, Φ is the set of their phenotypes, and φ is a mapping from Z onto Φ (the *gene control*) associating the phenotypes with the underlying genotypes. The existence of such a mapping is a fundamental principle; see [3, Chap. 1]. A simplest manifestation of that was discovered by Mendel (1866).

In his famous experiments Mendel worked with the alternative colors of peas, Y (yellow) or G (green), and derived some rules of transmission of these characteristics (phenotypes) from parents to offsprings. If both parents are of phenotype G then so are all their offsprings, symbolically, $G \times G \to G$. In fact, there are two sorts of Y, say Y_1 and Y_2 such that $Y_1 \times Y_1 \to Y_1$ but $Y_2 \times Y_2 \to \{Y_1, Y_2, G\}$, i.e., any of the characteristics Y_1, Y_2, G is observed in offsprings. In this sense Y_1 can be called *nonsplitting yellow* in contrast to the *splitting yellow* Y_2. Moreover, stable frequencies (probabilities) $\frac{1}{4}, \frac{1}{2}, \frac{1}{4}$ for the offsprings Y_1, Y_2, G are observed in a large population in which all parents are Y_2. To explain this result Mendel introduced the concept of *constant character* (or *gene* in modern terminology). He supposed that there are two genes, say A and a, such that Y_1, Y_2, and G are their combinations (genotypes) $Y_1 = AA$, $G = aa$, and $Y_2 = Aa \equiv aA$. The offsprings' genotypes are independent combinations of two genes coming together from two parental gene pools in the process of *fertilization*. On the cell level this process results in fertilized eggs (*zygotes*). In turn, the gene pools are formed by the separation of genes from the genotypes of zygotes in the process of *meiosis* which results in the sex cells (*gametes*).

The *Mendel phenotypical structure* is (Z, Φ, φ) where $Z = \{AA, Aa, aa\}$, $\Phi = \{Y, G\}$ and $\varphi(AA) = \varphi(Aa) = Y$, $\varphi(aa) = G$. In Mendel's terms A *dominates* a which just means that the phenotype of AA is the same as of Aa.

The genes carried by a zygote (or any body cell) are located in the same position on two geometrically identical *chromosomes*, which are called *homologous*. The position of a gene is called its *locus*. For example, Menderl's genes A and a are from the same locus or, in other words, they are the *alleles* of this locus. In general, there are more than two alleles for a given locus in the population but each zygote carries exactly two alleles which, maybe, are not distinct. (In this paper we do not consider the so-called polyploids.)

There are many loci on each chromosome and there is a number of pairs of homologous chromosomes in each body cell (in the zygote). The loci are called *linked* if they relate to the same chromosome (hence, to all homologous chromosomes).

A zygote is called a *homozygote* if at every loci the alleles coincide. If for a zygote there is a locus with distinct alleles then it is called a *heterozygote*.

The special chromosomes X and Y determine the sex: XX are females and XY are males. All other chromosomes (as well as the loci therein) are called *autosomal*.

In meiosis the homologous chromosomes may break in some place (or places) and then exchange corresponding parts. These events (*crossing-overs*) are random; their probabilities are important structural parameters of the population.

The present paper is organized as follows.

In Section 2 we describe the genotypical structure in terms of an algebraic language; see [4–6].

In Section 3 we give some general definitions concerning the phenotypical structure and then consider the single-locus situation. In particular, we describe the multiallele dominance in terms of an acyclic directed graph.

Section 4 is devoted to the construction of direct decompositions which, in particular, allows us to describe some multilocus phenotypical structures as the direct products of single-locus phenotypical structures.

In Section 5 we consider a natural concept of phenotypical heredity for the multilocus multiallele population. Actually, this is formulated in terms of a directed graph which shows a relation between the offspring and parental gametes by mean of the homozygote phenotypes (the *graph of phenotypical heredity*). If this graph is acyclic, we say that the *phenotypical structure is acyclic*. A particular case of that is the direct product of single-locus dominant phenotypical structures, the multilocus dominance.

The evolutionary equations of natural selection can be adjusted to the phenotypical structure assuming the selection parameters (the *fitness coefficients*) depending only on phenotypes. This situation is considered in Section 6. Note that, though the dynamical problems are principal in mathematical population genetics after classical works of Fisher, Haldane, and Wright, the phenotypical selection processes are still investigated for some elementary models like Mendel's dominance.

An important aspect of the population dynamics is the "population statics," the subject of which is the equilibria set, i.e., the set of fixed points of the evolutionary operator. By the Brouwer Fixed-Point Theorem the equilibria set is nonempty. This set may be infinite in some realistic situations. For instance, this is true in absence of effective selection, i.e., in the case of equal fitness coefficients when the equilibria set is the famous Hardy–Weinberg parabola (1908) or its generalizations. If the equilibria set is finite then the question about the number of equilibria becomes possible; see [3, Chap. 6] for biological motivation.

In Section 7 we consider the general problem of finiteness of the equilibria set and estimation of its cardinality. Using the classical elimination theory we prove a very useful (cf. [2]) technical lemma concerning the equilibria equations extended to the complex space. (This is possible since the

equations are algebraic.) This lemma does work in Section 8 where we prove our Main Theorem. The latter states that *the equilibria set is finite for any acyclic phenotypical structure and for all fitness vectors (whose coordinates are the fitness coefficients) except for an algebraic proper homogeneous subset of the fitness space.* In this sense the equilibria set is finite generically for any acyclic phenotypical structure. The number of equilibria admits an upper bound which only depends on the number of gamete genotypes under consideration.

One of applications of the Main Theorem is generic finiteness of the equilibria set for the multilocus dominant phenotypical structure. This result is new even in the single-locus multiallele case.

2. GENOTYPICAL STRUCTURES

Consider a set $L = \{1, \dots, l\}$ of autosomal loci with allele genes a_{ik} at the ith locus ($1 \le i \le l$, $1 \le k \le m_i$, $m_i \ge 2$), so that we have the *gene array*

$$\begin{matrix} a_{11} & \cdots & a_{1m_1} \\ \vdots & \ddots & \vdots \\ a_{l1} & \cdots & a_{lm_l}. \end{matrix}$$

The *gamete genotypes* (the *gametes*, for short) are formal commutative monomes

$$g = \prod_{i=1}^{l} a_{ik_i}.$$

The set of all gametes is denoted by Γ. The total number of gametes is $|\Gamma| = m_1 \cdots m_l$. In particular, $|\Gamma| = 2^l$ for l diallele loci.

Given g, for every subset $U \subset L$ one can consider the corresponding *subgamete*

$$g_U = \prod_{i \in U} a_{ik_i}.$$

In particular, $g_i = a_{ik_i}$, $1 \le i \le l$. Obviously,

$$g = g_U g_V, \tag{1}$$

where $V = L \backslash U$, the complement of U in L. The partitions $U|V$ (U runs over all subsets of L) are in a 1-1 correspondence with all possible crossing-overs including the *trivial* one corresponding to $\varnothing|L$; $g_\varnothing$ is the formal symbol such that $g_L g_\varnothing = g_\varnothing g_L = g_L$.

If a crossing-over $U|V$ occurs in meiosis then every gamete pair (g, h) produces the *recombinant gametes* $g_U h_V$ and $h_U g_V$ with equal probabilities $\frac{1}{2}r(U|V)$. The probabilities $r(U|V)$ constitute the *linkage distribution*. Obviously,

$$r(U|V) \geq 0, \qquad \sum_{U|V} r(U|V) = 1. \tag{2}$$

Note that for any nonempty subset $K \subset L$ the linkage distribution r_K is automatically defined; see [5, Eq. 6.1.1].

The loci $1, \ldots, l$ are distributed among the chromosomes according to a partition

$$L = C^1|\cdots|C^q. \tag{3}$$

into the *linkage groups*. Two (or more) loci are *linked* if they belong to the same linkage group. In this case they cannot lie on nonhomologous chromosomes. (The converse may not be true, see Example in [5, p. 238].) Note that the *chromosomal partition* (3) requires a special form of the linkage distribution r. Conversely, (3) can be determined in terms of r; see [5, Lemma 6.1.6].

Each zygote is originated from a pair of gametes (g, h). However, its genotype does not change under the transposition $g \longleftrightarrow h$. Moreover, let

$$g = g^1 \cdots g^q, \qquad h = h^1 \cdots h^q$$

be the decompositions of g and h into the products of subgametes according to (3). Then any transposition $g^j \longleftrightarrow h^j$ does not affect the zygote genotype since any pair of homological chromosomes is not ordered. For a gamete pair (g, h) we denote the corresponding *zygote genotype* (the *zygote*, for short) by $g \circ h$, so that $g \circ h$ is the class of all gamete pairs arising from (g, h) by the above mentioned transpositions. All $g \circ g$ are *homozygotes*; all others are *heterozygotes*. The pair (Z, r) where Z is the set of all zygotes and r is the linkage distribution can be called a *genotypical structure* of the population. For any subset $K \subset L$ we have the *genotypical substructure* (Z_K, r_K) where Z_K is the set of subzygotes $g_K \circ h_K$ for $g \circ h \in Z$. This notion is well defined since the restriction of (3) to K is a chromosomal partition as well; see [5, Lemma 6.1.5].

3. PHENOTYPICAL STRUCTURES

Let $\Phi = \{f_1, \ldots, f_n\}$ be the set of all possible *phenotypes* of zygotes, $1 \leq n \leq |Z|$. We suppose that for every zygote genotype its phenotype is uniquely determined. This means that there is a mapping (the *gene control*)

φ: $Z \to \Phi$ such that the phenotype of a zygote z is $\varphi(z)$. This mapping is supposed to be surjective, otherwise some elements of Φ would not be related to zygotes as their phenotypes. Note that the mapping φ can be lifted to the Cartesian square $\Gamma \times \Gamma$ (which consists of the gamete pairs) by defining $\varphi(g, h) = \varphi(g \circ h)$. Obviously, $\varphi(h, g) = \varphi(g, h)$, moreover, $\varphi(g, h)$ is invariant with respect off all chromosomal transpositions.

It is convenient to identify each phenotype f_k with its preimage $\varphi^{-1} f_k \in Z$ which is actually the class Z_k of zygotes whose phenotype is f_k. The classes Z_k $(1 \le k \le n)$ constitute the *phenotypical partition* of Z. Any partition of Z can be formally considered as a phenotypical one: for any zygote its phenotype can be determined as its class in the partition.

We call the triple (Z, Φ, φ) the *phenotypical structure* of the population. For brevity, we do not mention the linkage distribution r in this definition, though in fact, r is supposed to be given together with Z.

One can identify the phenotypical structures (Z, Φ, φ) and (Z, Ψ, ψ) over the same genotypical structure if there exists a bijective mapping T: $\Phi \to \Psi$ such that $\psi(z) = T(\varphi(z))$, hence, $\varphi(z) = T^{-1}(\psi(z))$. In this situation the structures (Z, Ψ, ψ) and (Z, Φ, φ) are called *isomorphic*. For example, any phenotypical structure is isomorphic to the structure determined by the corresponding phenotypical partition. The latter can be considered as the canonical representative of the family of isomorphic phenotypical structures.

Let (Z, Φ, φ) and (Z, Ψ, ψ) be some phenotypical structures over the same genotypical structure. We say that (Z, Ψ, ψ) is an *enlargement* of (Z, Φ, φ), or (Z, Φ, φ) is a *refinement* of (Z, Ψ, ψ), if the phenotype $\psi(z)$ only depends on $\varphi(z)$, i.e.,

$$\varphi(z) = \varphi(\zeta) \Longrightarrow \psi(z) = \psi(\zeta). \tag{4}$$

In other words, $\psi(z) = \theta(\varphi(z))$ for a mapping θ: $\Phi \to \Psi$. (This mapping is automatically surjective.) In terms of partitions this means that every class of (Z, Ψ, ψ) is the union of some classes of (Z, Φ, φ), so that (Z, Ψ, ψ) is *larger* than (Z, Φ, φ), or (Z, Φ, φ) is *finer* than (Z, Ψ, ψ).

Now we consider some examples of phenotypical structures.

EXAMPLE 3.1. In the simplest situation all zygotes are of the same phenotype, $n = 1$. We call this phenotypical structure *neutral*. This structure is the *largest* one; i.e., it is the enlargement of every phenotypical structure with the same Z.

EXAMPLE 3.2. In the opposite situation the phenotypes of distinct zygotes are distinct, $n = |Z|$. This is just the case of a bijective gene control φ. We call such a phenotypical structure *separative*. This structure is the *finest* one, i.e., any phenotypical structure with the same Z is its enlargement.

EXAMPLE 3.3. At a single locus with two alleles A and a there are exactly four phenotypical structures. Below we use a more standard notation AA instead of $A \circ A$, etc.; note that $aA \equiv Aa$.

(1) The neutral one, where the only class is $\{AA, aa, Aa\}$;

(2) The separative one: $\{AA\}, \{aa\}, \{Aa\}$;

(3) The *Mendel dominant* one: $\{AA, Aa\}$ and $\{aa\}$;

(4) $\{AA, aa\}$ and $\{Aa\}$.

In case (4) the phenotypes can be identified with the numbers of different genes and then $\varphi(AA) = \varphi(aa) = 1$, $\varphi(Aa) = 2$. For this reason we call this phenotypical structure *quantitative*.

EXAMPLE 3.4. Consider a single locus with any number $m \geq 2$ of alleles $a_1, \ldots, a_m$. The zygote genotype $a_i a_k$ is the gamete pair (a_i, a_k) up to transposition, so that $|Z| = m(m+1)/2$.

The principal biological mechanism for forming single-locus phenotypes is *dominance*. We will describe this in terms of a directed graph (a *dominance graph*) with vertices $a_1, \ldots, a_m$ and arcs $a_i \to a_k$ corresponding to the sentences "a_i dominated a_k." For instance, in the Mendel dominance case with alleles $\{A, a\}$ the dominance graph is $A \to a$.

For the classical blood group system there are three alleles A, B, O with the dominance graph $A \to O \leftarrow B$; in particular, there is no dominance relation between A and B. The corresponding phenotypes are $\{OO\}$, $\{AA, AO\}$, $\{BB, BO\}$, $\{AB\}$.

There is no dominance relation at all in the locus with two alleles M, N, controlling another blood group system: $\{MM\}$, $\{NN\}$, $\{MN\}$. The dominance graph has no arcs in this phenotypical structure.

Conversely, let a directed graph D with vertices $a_1, \ldots, a_m$ be given. Suppose that D is *acyclic*; i.e., there are no cycles in D. The corresponding phenotypical structure is determined as follows.

First of all, irrespective of the graph, we attribute some pairwise different phenotypes f_i to the homozygotes $a_i a_i$, $1 \leq i \leq m$. The same f_i will be the phenotype of the heterozygote $a_i a_k$ $(i \neq k)$ if $a_i \to a_k$ in D (then $a_k \to a_i$ is forbidden since D is acyclic). Finally, in the absence of arcs between a_i and a_k, $i \neq k$, the heterozygote $a_i a_k$ has a specific phenotype $f_{ik} \equiv f_{ki}$.

Obviously, if there are no arcs in D then the corresponding phenotypical structure is separative. But the neutral phenotypical structure cannot be determined by a dominance graph, otherwise the homozygote phenotypes would be distinct.

The quantitative phenotypical structure at a single diallele locus cannot be described in dominance terms.

4. DIRECT DECOMPOSITIONS

Consider a phenotypical structure (Z, Φ, φ). Suppose that for a nontrivial partition $L = L_1|L_2$ and for some phenotypical structures $(Z_{L_1}, \Phi_1, \varphi_1)$, $(Z_{L_2}, \Phi_2, \varphi_2)$ (with the linkage distributions r_{L_1} and r_{L_2}, respectively) we have $\Phi = \Phi_1 \times \Phi_2$ and $\varphi = \varphi_1 \times \varphi_2$ in the sense

$$\varphi(g \circ h) = \big(\varphi_1(g_{L_1} \circ h_{L_1}), \varphi_2(g_{L_2} \circ h_{L_2})\big). \tag{5}$$

This means that there are two phenotypical partitions

$$Z_{L_1} = Z_{11} \cup \cdots \cup Z_{1n_1}, \qquad Z_{L_2} = Z_{21} \cup \cdots \cup Z_{2n_2}$$

such that the initial phenotypical partition is

$$Z = \bigcup \big\{ Z_{1k} Z_{2j} | 1 \le k \le n_1, 1 \le j \le n_2 \big\}, \tag{6}$$

where

$$Z_{1k} Z_{2j} = \big\{ g \circ h \in Z | g_{L_1} \circ h_{L_1} \in Z_{1k},\ g_{L_2} \circ h_{L_2} \in Z_{2j} \big\}.$$

In this situation we say that (Z, Φ, φ) is the *direct product* of the *constituents* $(Z_{L_1}, \Phi_1, \varphi_1)$ and $(Z_{L_2}, \Phi_2, \varphi_2)$, and we write

$$(Z, \Phi, \varphi) = \big(Z_{L_1}, \Phi_1, \varphi_1\big) \times \big(Z_{L_2}, \Phi_2, \varphi_2\big).$$

A phenotypical structure which is isomorphic to a direct product is called *decomposable*. The number of phenotypes in this case is $n_1 n_2$ as (6) shows. The gene control φ of form (5) can be interpreted as a result of independently acting gene controls φ_1 and φ_2.

If a phenotypical structure is not decomposable then it is called *indecomposable*. Biologically, this is the case of *epistasis* between the groups of loci L_1 and L_2.

Likewise, one can consider the direct decomposition

$$\big(Z, \Phi, \varphi\big) = \big(Z_1, \Phi_1, \varphi_1\big) \times \cdots \times \big(Z_s, \Phi_s, \varphi_s\big) \tag{7}$$

for any partition $L = L_1| \cdots |L_s$, $s \ge 2$, with nonempty L_k, $1 \le k \le s$. This is also the situation of *decomposability* but with s constituents. In particular, if all L_k are singletons, $s = l$, then (Z, Φ, φ) is the direct product of some single-locus phenotypical structures. In this case

$$\varphi(g \circ h) = \big(\varphi_1(g_1 \circ h_1), \ldots, \varphi_l(g_l \circ h_l)\big) \tag{8}$$

and we say that (Z, Φ, φ) is *completely decomposable* (or *completely nonepistatic*).

Obviously, any neutral phenotypical structure is completely decomposable with neutral constituents.

PROPOSITION 4.1. *A separative phenotypical structure is completely decomposable if and only if the loci* $1, \ldots, l$ *are pairwise unlinked*.

Proof. If the loci are pairwise unlinked then the structure is the direct product of the single-locus separative structures. Indeed, in this case the zygotes $g \circ h$ can be identified with the noncommutative monomes

$$\prod_{i=1}^{l} a_{ik_i} a_{iq_i} \tag{9}$$

with $1 \le k_i \le q_i \le m_i$, $1 \le i \le l$. The submonomes $a_{ik_i} a_{iq_i}$ are just the zygotes at the ith locus, $1 \le i \le l$. In any separative structure the phenotypes are in a 1-1 correspondence with the zygotes. Hence, (9) yields the desired direct decomposition.

Note that the number of phenotypes above is

$$n = \prod_{i=1}^{l} n_i, \; n_i = \frac{1}{2} m_i (m_i + 1), \qquad 1 \le i \le l.$$

(In particular, $n = 3^l$ for l diallele loci.)

Now let a l-locus separative structure be completely decomposable and let the number of phenotypes at the ith locus be ν_i, $1 \le i \le l$. Then the number of phenotypes in the whole structure is

$$\nu = \prod_{i=1}^{l} \nu_i \le n$$

since $\nu_i \le n_i$, $1 \le i \le l$. On the other hand, the monomes (9) are phenotypically distinguished, so that $\nu \ge n$. Hence, $\nu = n$ and then there is no pair of linked loci, otherwise some more zygotes would appear. For example, if the loci 1 and 2 are linked then

$$a_{11} a_{22} \circ a_{12} a_{21} \ne a_{11} a_{21} \circ a_{12} a_{22} \tag{10}$$

in contrast to the case of unlinked loci. ∎

We see that the direct product of phenotypical structures is well defined only if a chromosomal partition for the whole system of loci $L = \{1, \ldots, l\}$ is given a priori. For this reason the direct decomposition makes sense only inside a given phenotypical structure.

COROLLARY 4.1. *The separative 2-locus phenotypical structure with linked loci is indecomposable*.

For example, let the alleles be A and a at the first locus and B, b at the second one. Then there are the following zygotes (cf. (9)): (1) the *homozygotes* $AABB$, $AAbb$, $aaBB$, $aabb$; (2) the *simple heterozygotes* $AaBB$, $Aabb$, $AABb$, $aaBb$; (3) the *double heterozygotes* $AaBb$ and $AabB$ which, however, coincide if the loci are unlinked. If the loci are linked then $AaBb \equiv AB \circ ab$, $AabB \equiv Ab \circ aB$, so that $AaBb \neq AabB$. In the latter case the direct product of single-locus separative structures contains the class $\{AaBb, AabB\}$. In the 2-locus separative structure this class splits into the classes $\{AaBb\}$ and $\{AabB\}$.

We say that there is no *position effect* in a phenotypical structure (Z, Φ, φ) if the phenotype $\varphi(g \circ h)$ is invariant for the gene transpositions $g_i \leftrightarrow h_i$, $1 \leq i \leq l$. Equivalently, $\varphi(g \circ h)$ only depends on the set of genes situated in g, h, irrespective of how the genes are placed in the homologous chromosomes. For example, (10) says that there is a position effect in the separative 2-locus structure with linked loci. This effect disappears in the direct product of the single-locus separative structures regardless of the linkage.

PROPOSITION 4.2. *There is no position effect in a phenotypical structure* (Z, Ψ, ψ) *if and only if it is an enlargement of the direct product* (Z, Φ, φ) *of the single-locus separative structures*.

Thus, the latter is the finest phenotypical structure with no position effect.

Proof. Obviously, there is no position effect in (Z, Φ, φ); see (8). A fortiori, the same is true for any of its enlargement. Conversely, let (Z, Ψ, ψ) be a phenotypical structure with no position effect. This means that the phenotype $\Psi(g \circ h)$ is uniquely determined by the monome (9) which, in turn, is a bijective function of $\varphi(g \circ h)$. Thus, we have $\Psi(g \circ h) = \theta(\varphi(g \circ h))$ where θ is a mapping $\Phi \to \Psi$. We see that (Z, Ψ, ψ) is an enlargement of (Z, Φ, φ). ∎

An enlargement of a decomposable phenotypical structure may be indecomposable. Moreover, there are some indecomposable phenotypical structures with no position effect.

EXAMPLE 4.1. For two diallele loci we consider the *quantitative* phenotypical structure. Its classes are $K_1 = \{\text{homozygotes}\}$, $K_2 = \{\text{simple heterozygotes}\}$, $K_3 = \{\text{double heterozygote(s)}\}$. (Respectively, $\varphi|K_1 = 2$, $\varphi|K_2 = 3$, $\varphi|K_3 = 4$.) Obviously, there is no position effect in this structure. It is indecomposable, for otherwise, one of the constituents would be neutral while the other separative. However, their direct product is not the quantitative phenotypical structure.

5. ACYCLIC PHENOTYPICAL STRUCTURES

Let (Z, Φ, φ) be a phenotypical structure. For some gametes g and h we write $h \succeq g$ (or $g \preceq h$) if g is a recombinant gamete of a gamete pair (γ, χ) with the phenotype $\varphi(h, h)$. More formally, $h \succeq g$ means that there exists a gamete pair (γ, χ) and a partition $U|V$ such that

$$\varphi(\gamma, \chi) = \varphi(h, h), \qquad \gamma_U \chi_V = g. \tag{11}$$

LEMMA 5.1. *If $\varphi(\gamma, \chi) = \varphi(h, h)$ then $h \succeq \gamma$ and $h \succeq \chi$.*

Proof. Here (11) is valid with $g = \gamma$, $U = L$, $V = \varnothing$, so $h \succeq \gamma$. Similarly, $h \succeq \chi$. ∎

COROLLARY 5.1. *The binary relation $\succeq$ on the gamete set Γ is reflexive, i.e., $h = g \Rightarrow h \succeq g$.*

Proof. $g = h \Rightarrow \varphi(g, g) = \varphi(h, h) \Rightarrow h \succeq g$. ∎

Now let $h \succ g$ (or $g \prec h$) mean that $(h \succeq g) \& (h \neq g)$. By Corollary 5.1, $h \succeq g$ if and only if $h \succ g$ or $h = g$ and the latter alternative is strict. We will interpret the relation $h \succ g$ as a directed graph G whose set of vertices is Γ and $h \to g$ in G if and only if $h \succ g$. We call G the *graph of phenotypical heredity* and denote it by $G(Z, \Phi, \varphi)$. This is a generalization of the dominance graph at a single locus. Indeed, let in notation of Example 3.4, $g = a_i$ and $h = a_k$, $k \neq i$. Then (11) means that $\varphi(a_i a_j) = \varphi(a_k a_k)$ with some a_j. Here $j \neq i$ since the homozygous phenotypes are pairwise distinct. Then $j = k$ and $a_k \to a_i$; otherwise, the phenotype f_{ij} of the heterozygote $a_i a_j$ would be different from the phenotype f_k of the homozygote $a_k a_k$. Thus, the relation $a_k \succ a_i$ is equivalent to the presence of the arc $a_k \to a_i$ in the dominance graph.

It may happen that the graph of phenotypical heredity is not acyclic. The simplest example of this kind is the neutral phenotypical structure. Indeed, in this case $h \succ g$ as soon as $h \neq g$, so there is the cycle $h \succ g \succ h$.

DEFINITION 5.1. A phenotypical structure is called *acyclic* if the corresponding graph of phenotypical heredity is acyclic.

EXAMPLE 5.1. Any single-locus dominant phenotypical structure is acyclic.

If a graph has no arcs then it is trivially acyclic. For a graph of phenotypical heredity the absence of arcs just means that $h \succeq g \Longrightarrow h = g$. Here is an application of this remark.

PROPOSITION 5.1. *A phenotypical structure is acyclic if the phenotype of every homozygote is specific, i.e., it is different from all other phenotypes.*

Proof. Now the only case in (11) is $h = g$ since $\varphi(\gamma, \chi) = \varphi(h, h)$ implies $\gamma = \chi = h$ by assumption. ∎

COROLLARY 5.2. *All separative phenotypical structures are acyclic.*

Proposition 5.1 can be partially inverted.

PROPOSITION 5.2. *If a phenotypical structure is acyclic then the homozygous phenotypes are pairwise distinct.*

Proof. By Lemma 5.1 if $\varphi(g, g) = \varphi(h, h)$ then $h \succeq g$ and $g \succeq h$. If $h \neq g$ then there is the cycle $h \succ g \succ h$. ∎

In any l-locus quantitative phenotypical structure all homozygotes have the same phenotype (which can be identified with the number l). By Proposition 5.2 this structure is not acyclic.

Many multilocus acyclic phenotypical structures can be extracted from

PROPOSITION 5.3. *Any decomposable phenotypical structure* (Z, Φ, φ) *with acyclic constituents is acyclic.*

Proof. In situation (7) let us consider the corresponding graphs G_k, $1 \leq k \leq s$, the graphs of phenotypical heredity for the constituents. It is easy to see that if $h \to g$ in $G(Z, \Phi, \varphi)$ then $h_{L_k} \succeq g_{L_k}$ for all k, $1 \leq k \leq s$, and, moreover, $h_{L_k} \neq g_{L_k}$ for some k. Therefore, any cycle in $G(Z, \Phi, \varphi)$ yields a cycle for a constituent. ∎

COROLLARY 5.3. *The direct product of separative phenotypical structures is acyclic.*

Any completely decomposable phenotypical structure whose constituents are determined by some dominance graphs can be called a *multilocus dominance*.

COROLLARY 5.4. *Any multilocus* (*in particular, single-locus*) *dominant phenotypical structure is acyclic.*

PROPOSITION 5.4. *If a phenotypical structure* (Z, Ψ, ψ) *is acyclic then any refinement* (Z, Φ, φ) *is acyclic as well.*

Proof. By (11) and (4) we obtain that if $h \succeq g$ in (Z, Φ, φ) then $h \succeq g$ in (Z, Ψ, ψ). Therefore, any cycle in $G(Z, \Phi, \varphi)$ is also a cycle in $G(Z, \Psi, \psi)$. ∎

For any acyclic directed graph the graph of its paths determines a partial ordering of the vertices. In turn, any partial ordering of a finite set can be extended to a linear ordering. Thus, we have

LEMMA 5.2. *For any acyclic phenotypical structure* (Z, Φ, φ) *the relation* $\succ$ *can be extended to a linear order on the gamete set* Γ.

Later on we preserve the notation $\succ$ for the extended relation.

6. PHENOTYPICAL SELECTION: PARAMETERS AND EVOLUTIONARY EQUATIONS

A *fitness function* λ of a population is a nonzero mapping $Z \to \mathbf{R}_+$ where, as before, Z is the set of zygotes, $\mathbf{R}_+ = \{\xi \in \mathbf{R} : \xi \geq 0\}$. The value $\lambda(z)$ is called the *fitness coefficient* of a zygote $z \in Z$.

Given a phenotypical structure (Z, Φ, φ), we suppose that the *fitness coefficient* $\lambda(z)$ *only depends on the phenotype* $\varphi(z)$, i.e.,

$$\lambda(z) = \Lambda(\varphi(z)), \tag{12}$$

where Λ is a nonzero mapping from Φ into $\mathbf{R}_+$. For any $f \in \Phi$ the value $\Lambda(f)$ is called the *fitness coefficient of the phenotype* f. The n-tuple $(\Lambda(f) : f \in \Phi)$ is called the *phenotype fitness vector*. The set of all fitness vectors is $\mathbf{R}_+^n \backslash \{0\}$, the punctured at 0 coordinate cone in $\mathbf{R}^n$. This cone can be called the *phenotype fitness space*.

Under our assumption (12) the fitness function λ and the phenotypical structure (Z, Φ, φ) are called *compatible* and (Z, Φ, φ) is called λ-*compatible*. For example, any fitness function is compatible with the separative phenotypical structure. However, the latter is too fine in general. In the largest λ-compatible phenotypical structure the classes are just the level sets of λ. This means that some zygotes z and ζ are of the same phenotype if and only if $\lambda(z) = \lambda(\zeta)$. In this case we say that the phenotypical structure is λ-*determined*. The separative phenotypical structure is determined by any bijective fitness function λ.

The neutral phenotypical structure is determined by a constant fitness function. A population with a constant fitness function is called *selection free* or, briefly, *free* [4].

The Mendel dominant phenotypical structure at a single locus with alleles A and a such that $\lambda(AA) = \lambda(Aa) \neq \lambda(aa)$ is λ-determined. This structure is not compatible with any constant fitness function.

Like φ, one can lift λ to $\Gamma \times \Gamma$ by setting $\lambda(g, h) = \lambda(g \circ h)$. Then (12) takes the form

$$\lambda(g, h) = \Lambda\big(\varphi(g, h)\big). \tag{13}$$

Obviously, $\lambda(h, g) = \lambda(g, h)$. Thus, every lifted fitness function is a nonnegative nonzero matrix over $\Gamma \times \Gamma$, the *fitness matrix*; this matrix is symmetric, moreover, $\lambda(g, h) = \lambda(g', h')$ if $g \circ h = g' \circ h'$.

The selection process is governed by the evolutionary equations where the fitness coefficients are parameters. We consider these equations on the gamete level, cf. [5, Sects. 1.2, 1.3]. Then a *state* p of the population is a probability distribution on Γ, $p = (p(g) : g \in \Gamma)$. Thus, the *state space* is

the simplex

$$S = \left\{ (p(g)) : \sum_g p(g) = 1,\ p(g) \geq 0 \right\},$$

where g runs over Γ. This is just the basis simplex in the vector space $\mathbf{R}^{|\Gamma|}$.

If p is a state of the population in a generation then its state in the next generation is $p' = Fp$, where F is a mapping $S \to S$ called the *evolutionary operator* of the population.

The *evolutionary equations* of the population (expressing the mapping F in coordinate form) are

$$p'(g) = \frac{1}{W(p)} \sum_{U|V} r(U|V) \sum_h \lambda(g_U h_V, h_U g_V) p(g_U h_V) p(h_U g_V), \qquad (14)$$

where $W(p)$ is the *mean fitness of the population*

$$W(p) = \sum_{g,\,h} \lambda(g, h) p(g) p(h), \qquad (15)$$

see [5, Eqs. 9.5.1 and 9.5.3]. The range for both g and h in (14) and (15) is Γ. Obviously, $W(p) > 0$ if $p(g) > 0$ for all g, so that (14) makes sense at least for $p \in \mathrm{Int}S$. We suppose that all $\lambda(g, g) > 0$ which provides $W(p) > 0$ everywhere on the simplex S, so that (14) can be extended to the whole S.

It is convenient to rewrite (14) using the substitution $g_U h_V = \gamma$, $h_U g_V = \chi$ (the inverse substitution is $g = \gamma_U \chi_V$, $h = \chi_U \gamma_V$). In this way we obtain

$$p'(g) = \frac{1}{W(p)} \sum_{U|V} r(U|V) \sum_{\gamma_U \chi_V = g} \lambda(\gamma, \chi) p(\gamma) p(\chi). \qquad (16)$$

In order to adjust these evolutionary equations to a given λ-compatible phenotypical structure (Z, Φ, φ) we relate to any gamete g the set

$$\Phi(g) = \left\{ f \in \Phi \,|\, \exists \gamma, \chi, U|V : f = \varphi(\gamma, \chi),\ \gamma_U \chi_V = g \right\}. \qquad (17)$$

Obviously, the pairs (γ, χ) in (16) are just those which appear in (17). It is important that the inclusion $\varphi(h, h) \in \Phi(g)$ is equivalent to $h \succeq g$. In particular, $\varphi(g, g) \in \Phi(g)$.

Using (17) and (13) we can represent (16) in the form

$$p'(g) = \frac{1}{W(p)} \sum_{U|V} r(U|V) \sum_{f \in \Phi(g)} \Lambda(f) \sum_{\substack{\gamma_U \chi_V = g \\ \varphi(\gamma, \chi) = f}} p(\gamma) p(\chi) \qquad (18)$$

or, briefly,

$$p'(g) = Q_g(p)/W(p), \qquad (19)$$

where

$$Q_g(p) = \sum_{f \in \Phi(g)} \Lambda(f) \sum_{\varphi(\gamma, \chi)=f} \pi_{\gamma\chi, g}\, p(\gamma) p(\chi) \tag{20}$$

with

$$\pi_{\gamma\chi, g} = \sum_{\gamma_U \chi_V = g} r(U|V).$$

According to (1) and (2)

$$\pi_{gg, g} = 1. \tag{21}$$

It follows from (19) that

$$W(p) = \sum_g Q_g(p) \tag{22}$$

since $p' \in S$ for all $p \in S$. The mean fitness $W(p)$ can also be rewritten in terms of the phenotype fitness coefficients, namely,

$$W(p) = \sum_{f \in \Phi} \Lambda(f) \sum_{\varphi(g, h)=f} p(g) p(h) \tag{23}$$

because of (15) and (13).

Being quadratic fractional, the evolutionary equations (18) are homogeneous of degree 0 with respect to the phenotype fitness vector $(\Lambda(f) : f \in \Phi)$. Hence, all proportional phenotype fitness vectors determine the same evolutionary operator. This means that the set $\mathbf{R}^n_+ \backslash \{0\}$ of all those vectors can be reduced to $\mathbf{RP}^{n-1}_+$, the nonnegative part of $(n-1)$-dimensional real projective space. Another possible reduction is the normalization with respect to a norm in $\mathbf{R}^n$, for example, $\max(\Lambda(f) : f \in \Phi) = 1$ or $\sum \Lambda(f) = 1$. After this we get the nonnegative part of the $(n-1)$-dimensional unit sphere in the normed space $\mathbf{R}^n$. In any case we can consider a *reduced fitness space* instead of the *initial* one.

Either of the above procedures reduces each subset $X \subset \mathbf{R}^n_+ \backslash \{0\}$. The correspondence between X and its reduction is one-to-one if X is homogeneous, i.e., $x \in X \Rightarrow \alpha \cdot x \in X$ for all $\alpha > 0$. Therefore, *if X is a homogeneous proper subset of the initial fitness space then its reduction is a proper subset of the reduced fitness space*.

7. THE EQUILIBRIA SET

A state p of a population is called an *equilibrium* if p is a fixed point of the evolutionary operator F, i.e., $Fp = p$.

If the population is selection free (the phenotypical structure is neutral) then the equilibria set consists of

$$p(g) = \prod_{i=1}^{l} p^{(i)}(g_i),$$

where $p^{(i)}$ are arbitrary single-locus states; see [5, Theorem 6.3.1]. Obviously, this set is infinite. Note that the evolutionary operator of the free population is purely quadratic; cf. [5, Eq. 6.2.14]. However, in the class of all quadratic mappings $S \to S$ with nonnegative coefficients the fixed point set is finite generically; see [5, Theorem 8.1.3]. The latter follows from the classical elimination theory, see [1], which learns that for any system of homogeneous algebraic equations

$$F_1(\xi_1, \ldots, \xi_n) = 0, \ldots, F_s(\xi_1, \ldots, \xi_n) = 0 \tag{24}$$

with indefinite real coefficients and with complex unknowns $\xi_1, \ldots, \xi_n$ there exists a system $\{R_1, \ldots, R_t\}$ of polynomials of the coefficients such that (24) has a nontrivial complex solution if and only if the coefficients satisfy the equations

$$R_1 = 0, \ldots, R_t = 0. \tag{25}$$

Any specialization of the coefficients preserves the connection between the solvability of (24) and the validity of (25).

The polynomials R_i are the resultants of the system (24). They are homogeneous with respect to the coefficients of every F_i, $1 \le i \le s$. All corresponding homogeneity degrees are nonzero.

It may happen that the system (25) is empty (the case $t = 0$). This just means that the system (24) has a nontrivial complex solution for every set of values of the coefficients.

It follows from (19) that all equilibrium states p satisfy the equations

$$p(g)W(p) - Q_g(p) = 0, \tag{26}$$

where g runs over Γ. In addition,

$$\sum_{\gamma} p(\gamma) - 1 = 0. \tag{27}$$

In order to use the resultants we pass from (26) and (27) to some homogeneous equations with unknown complex vector $\xi = (\xi(\gamma) : \gamma \in \Gamma)$, namely,

$$\xi(g)Q_h(\xi) - \xi(h)Q_g(\xi) = 0, \qquad h \succ g, \tag{28}$$

and

$$\sum_{\gamma} \xi(\gamma) - \tau\xi(l) = 0. \tag{29}$$

Here τ is a real coefficient, l is a fixed element of Γ, the $Q_g(\xi)$ are the quadratic forms (20) but in variables ξ instead of p. From now on we use the linear order on Γ coming from Lemma 5.2. The equations (28) are enumerated by those pairs (h, g) with $h \succ g$. (Obviously, the pairs (h, g) with $g \succeq h$ are redundant.) It is important that each solution of (26) satisfies (28).

Every Eq. (28) is homogeneous of degree 3, but (29) is homogeneous of degree 1. Also note that (28) are homogeneous of degree 1 with respect to the phenotype fitness vector; see (20).

The resultant equations (25) for the system (28) and (29) take the form

$$\sum_{j=0}^{d_{i,l}} c_{ij,l}\Big(\Lambda(f_1), \ldots, \Lambda(f_n)\Big)\tau^j = 0, \qquad 1 \le i \le t_l, \tag{30}$$

where $c_{ij,l}$ are homogeneous polynomials of the real vector variable $\Lambda = (\Lambda_1, \ldots, \Lambda_n)$. The following technical lemma is valid for an arbitrary phenotypical structure.

LEMMA 7.1. *Let for every $l \in \Gamma$ there exists a polynomial $c_{i_l j_l, l} \neq 0$. Consider the algebraic proper homogeneous subset $\widetilde{E} \subset \mathbf{R}^n$ defined by the equation*

$$\prod_{l \in \Gamma} c_{i_l j_l, l}(\Lambda) = 0. \tag{31}$$

If the phenotype fitness vector $\Lambda = (\Lambda(f_1), \ldots, \Lambda(f_n))$ does not belong to $\widetilde{E}$ then the set F_Λ of complex solutions of the system (26) *and* (27) *is finite. In particular, the corresponding equilibria set is finite.*

Proof. Suppose to the contrary. Then there exists $l \in \Gamma$ such that the coordinate $p(l)$ runs over an infinite set $F_{\Lambda,l}$ in the complex plane $\mathbf{C}$ when p runs over F_Λ. Take any point $w \neq 0$ from $F_{\Lambda,l}$ and consider any $p \in F_\Lambda$ such that $p(l) = w$. The vector $\xi = p$ is a nontrivial solution of system (28) and (29) with $\tau = 1/w$. Hence, all resultant equations (30) are fulfilled with this τ, so that each of those equations with unknown τ has infinitely many roots. Therefore, all $c_{ij,l}(\Lambda) = 0$, in particular, $c_{i_l, j_l}(\Lambda) = 0$. A fortiori, Λ satisfies (31), i.e., $\Lambda \in \widetilde{E}$, which contradicts our assumption. ∎

Note that the system (26) and (27) has exactly the same complex solutions as

$$p(g)W(p) - Q_g(p) = 0, \qquad g \neq k \tag{32}$$

jointly with (27). Indeed, the latter system implies Eq. (26) corresponding to $g = k$ by summation of all Eqs. (32) and by taking into account (27) and (22). For this reason Lemma 7.1 is valid for the system (32) and (27)

where the number of unknowns is (in contrast to (26) and (27)) equal to the number of equations. By the Bezout Theorem we obtain the upper bound

$$|F| \le 3^{|\Gamma|-1} \tag{33}$$

for the number of complex solutions of the system (26) and (27).

8. THE MAIN THEOREM

Our Main Theorem is the following

THEOREM 8.1. *Let a phenotypical structure* (Z, Φ, φ) *be acyclic. Then the equilibria set is finite for all phenotype fitness vectors* $(\Lambda(f) : f \in \Phi)$ *except for a proper homogeneous algebraic subset* $E \subset \mathbf{R}^n_+$.

Hence, the *exceptional set* E is of zero Lebesgue measure, and its complement $\mathbf{R}^n \backslash E$ is open and dense, so E is small in any reasonable sense. The same is also true for its reduction by projectivization or normalization. In any case we can say that *the equilibria set is finite generically for any acyclic phenotypical structure.* For the single-locus separative phenotypical structure this fact is elementary; see [5, Corollary 9.1.3].

Proof. There exists $l \in \Gamma$ *such that all polynomials* $c_{ij,\,l}$ *are equal to zero.* We prove that this assumption leads to a contradiction.

Let us start with the following auxiliary system of equations

$$\xi(h)\left[\sum_{\varphi(\gamma,\,\chi)=\varphi(g,\,g)} \pi_{\gamma\chi,\,g}\xi(\gamma)\xi(\chi)\right] = 0, \qquad h \succ g. \tag{34}$$

In (34) we have $g \succeq \gamma$ and $g \succeq \chi$ by Lemma 5.1. Let $(\xi(\gamma) : \gamma \in \Gamma)$ be a nontrivial complex solution of (34) and let $g = \min\{\gamma : \xi(\gamma) \ne 0\}$. Select the equations with this g. Since $\xi(\gamma)\xi(\chi) = 0$ for $g \succ \gamma$ or $g \succ \chi$, the sum in (34) is reduced to $\pi_{gg,\,g}\xi^2(g)$ which actually is $\xi^2(g)$ by (21). Since $\xi(g) \ne 0$ by definition, we obtain $\xi(h) = 0$ for all $h \succ g$ and, as we know, $\xi(h) = 0$ for all $g \succ h$. Thus, the only nonzero coordinate is $\xi(g)$.

Now we consider (34) simultaneously with (29). Show that if $\tau \ne 1$, then the system (34) and (29) has only the trivial complex solution. Indeed, let $(\xi(\gamma) : \gamma \in \Gamma)$ be a nontrivial complex solution. Then $\xi(g) \ne 0$ for some g but $\xi(\gamma) = 0$ for $\gamma \ne g$. Thus, (29) becomes $\xi(g) = \tau\xi(l)$. Hence, $l = g$ and $\tau = 1$, which is a contradiction.

The resultants of the system (34) and (29) are polynomials of τ. For any $\tau \ne 1$ at least one of them, say θ, does not vanish.

Now we slightly complicate (34) using some independent parameters $\varkappa_g$:

$$\varkappa_g \xi(h) \left[\sum_{\varphi(\gamma,\chi)=\varphi(g,g)} \pi_{\gamma\chi,g}\, \xi(\gamma)\xi(\chi) \right] = 0, \qquad h \succ g. \tag{35}$$

The system (35) and (29) turns into (34) and (29) if $\varkappa_g = 1$ for all g. Under this specialization one of the resultants of (35) and (29) turns into θ. Since this resultant is homogeneous with respect to every $\varkappa_g$, it is a monome of the form

$$\theta(\tau) \prod_g \varkappa_g^{\nu_g}. \tag{36}$$

The exponent ν_g is equal to the sum of the homogeneity degrees of the resultant with respect to the coefficients of those polynomials (35) which correspond to h such that $h \succ g$.

As the next step we consider the system

$$\mu \xi(g) Q_h(\xi) - \xi(h) Q_g(\xi; \mu, \varkappa_g) = 0, \qquad h \succ g, \tag{37}$$

where

$$Q_g(\xi; \mu, \varkappa_g) = \mu \sum_{\substack{f \in \Phi(g) \\ f \neq \varphi(g,g)}} \Lambda(f) \sum_{\varphi(\gamma,\chi)=f} \sum \pi_{\gamma\chi,g}\, \xi(\gamma)\xi(\chi) + \varkappa_g \left[\sum_{\varphi(\gamma,\chi)=\varphi(g,g)} \sum \pi_{\gamma\chi,g}\, \xi(\gamma)\xi(\chi) \right], \tag{38}$$

μ is an additional parameter.

Since $\varphi(\gamma, \gamma) \in \Phi(h)$ implies $\gamma \succeq h$, *for every* $\gamma \in \Gamma$ *the term* $\Lambda(\varphi(\gamma, \gamma))$ *cannot appear in* Eq. (37) *with* $g \succeq \gamma$.

The system (37) for $\mu = 0$ turns into (35). Therefore, one of resultants of system (37) and (29) is of the form

$$\widehat{R} = \theta(\tau) \prod_g \varkappa_g^{\nu_g} + R, \tag{39}$$

where R is a polynomial of τ, μ, all $\Lambda(f)$, and all $\varkappa_g$,

$$R|_{\mu=0} = 0. \tag{40}$$

Every monome in $\widehat{R}$ is of the form

$$\tau^\alpha \mu^\sigma \prod_f \Lambda(f)^{\varepsilon_f} \prod_g \varkappa_g^{\omega_g} \tag{41}$$

up to a constant factor. In all monomes from R we have $\sigma > 0$ because of (40). In all remaining monomes $\sigma = 0$ and all $\varepsilon_f = 0$ but $\omega_g = \nu_g$. We prove that

$$\sigma + \sum_g \omega_g = \sum_g \nu_g \tag{42}$$

in every monome (41).

Consider the system of homogeneous equations of third degree with indefinite coefficients,

$$\sum_I a_{h,\,g;\,I}\xi^I = 0, \qquad h \succ g. \tag{43}$$

Here $I = (i_\gamma : \gamma \in \Gamma)$ is the multi-index,

$$i_\gamma \geq 0, \qquad \sum_\gamma i_\gamma = 3,$$

and

$$\xi^I = \prod_\gamma \xi(\gamma)^{i_\gamma}.$$

A resultant of (43) and (29) turns into $\widehat{R}$ by the specialization

$$a_{h,\,g;\,I} = \mu \sum_f \alpha_{h,\,g;\,I,\,f}\Lambda(f) + \beta_{h,\,g;\,I}\varkappa_g, \qquad h \succ g, \tag{44}$$

where $\alpha_{h,\,g;\,I,\,f}$ and $\beta_{h,\,g;\,I}$ are numerical coefficients. Let $\sigma_{h,\,g}$ be the homogeneity degrees of this resultant with respect to the coefficients $a_{h,\,g;\,I}$.

If we substitute 2μ and $2\varkappa_g$ instead of μ and $\varkappa_g$, respectively, then all $a_{h,\,g;\,I}$ get the same factor 2 and then $\widehat{R}$ gets the factor 2^ρ with

$$\rho = \sum\{\sigma_{h,\,g} | (h, g) : h \succ g\}.$$

Every monome in $\widehat{R}$ must get the same factor. Looking at (41) we see that

$$\rho = \sigma + \sum_g \omega_g. \tag{45}$$

On the other hand,

$$\rho = \sum_g \nu_g \tag{46}$$

from the first summand of (39). Comparing (45) to (46) we obtain (42).

Another relation we need is

$$\nu_g = \sum_{h:h\succ g} \sigma_{h,\,g}. \tag{47}$$

In order to prove this we note that the considered resultant of (43) and (29) turns into (36) with specialization (44) and $\mu = 0$. After that its homogeneity degree is ν_g with respect to the variable $\varkappa_g$. On the other hand, $\varkappa_g$ in $\widehat{R}$ comes only from Eqs. (37). The homogeneity degree of $\widehat{R}|_{\mu=0}$ with respect to $\varkappa_g$ is exactly the right hand side of (47).

For $\mu = 1$ and $\varkappa_g = \Lambda(\varphi(g, g))$ the system (37) turns into (28). Therefore,

$$\overline{R} = \widehat{R}|_{\mu=1,\, \forall g: \varkappa_g = \Lambda(\varphi(g, g))} \tag{48}$$

is a resultant of system (28) and (29). If remains to prove that $\overline{R} \neq 0$.

By (39) it is sufficient to show that each monome (41) with $\sigma > 0$ cannot turn into a monome of the form

$$\tau^\alpha \prod_g \varkappa_g^{\nu_g}$$

by specialization indicated in (48). Suppose to the contrary that

$$\prod_f \Lambda(f)^{\varepsilon_f} \prod_g \Lambda(\varphi(g,\, g))^{\omega_g} = \prod_g \Lambda(\varphi(g, g))^{\nu_g}. \tag{49}$$

We know that the variables $\Lambda(\varphi(g, g))$ are pairwise distinct (Proposition 5.2). Hence, (49) is equivalent to

$$\forall\, g, \qquad \nu_g = \omega_g + \varepsilon_{\varphi(g, g)} \tag{50}$$

together with

$$(\forall\, g)\ (\varphi(g, g) \neq f) \Rightarrow \varepsilon_f = 0. \tag{51}$$

Since $\sigma > 0$, it follows from (42) that there exists $\bar{g}$ such that $\nu_{\bar{g}} > \omega_{\bar{g}}$. Then $\varepsilon_{\varphi(\bar{g}, \bar{g})} > 0$ by (50). One can assume that $\bar{g}$ with the latter property is maximal with respect to our ordering of Γ. Then $\varepsilon_{\varphi(\gamma, \gamma)} = 0$ for $\gamma \succ \bar{g}$ hence, (50) yields $\nu_\gamma = \omega_\gamma$ if $\gamma \succ \bar{g}$. Thus, the original monome (41) is actually

$$\tau^\alpha \mu^\sigma \prod_{\gamma \preceq \bar{g}} \Lambda(\varphi(\gamma, \gamma))^{\varepsilon_{\varphi(\gamma, \gamma)}} \prod_{g \preceq \bar{g}} \varkappa_g^{\omega_g} \prod_{\gamma \succ \bar{g}} \varkappa_\gamma^{\nu_\gamma}. \tag{52}$$

If we take $\Lambda(f) = 0$ for all f which are different from all $\Lambda(\varphi(g, g))$, this specialization does not affect (52) but at the same time (44) implies

$$a_{h, g; I} = \mu \sum_\gamma \alpha_{h, g; I, \varphi(\gamma, \gamma)} \Lambda(\varphi(\gamma, \gamma)) + \beta_{h, g; I} \varkappa_g, \qquad h \succ g.$$

However, $\alpha_{h, g; I, \varphi(\gamma, \gamma)} = 0$ for $\gamma \preceq g$, as we know from the remark after (38). Therefore,

$$a_{h, g; I} = \mu \sum_{\gamma \succ g} \alpha_{h, g; I, \varphi(\gamma, \gamma)} \Lambda(\varphi(\gamma, \gamma)) + \beta_{h, g; I} \varkappa_g, \qquad h \succ g.$$

Under the substitutions $\Lambda(\varphi(\gamma, \gamma)) \to 2\Lambda(\varphi(\gamma, \gamma))$ $(\gamma \succ \bar{g})$ and $\varkappa_g \to 2\varkappa_g (g \succeq \bar{g})$, we obtain $a_{h,g;I} \to 2a_{h,g;I}$ for $g \succeq \bar{g}$. Then the resultant $\widehat{R}$ gets the factor

$$2^{\sum_{h \succ g \succeq \bar{g}} \sigma_{h,g}} = 2^{\sum_{g \succeq \bar{g}} \nu_g}$$

by (47). At the same time the monome (52) gets

$$2^{\omega_{\bar{g}} + \sum_{\gamma \succ \bar{g}} \nu_\gamma}.$$

Thus,

$$\sum_{g \succeq \bar{g}} \nu_g = \omega_{\bar{g}} + \sum_{\gamma \succ \bar{g}} \nu_\gamma,$$

whence $\nu_{\bar{g}} = \omega_{\bar{g}}$, i.e., $\varepsilon_{\varphi(\bar{g}, \bar{g})} = 0$, which is a contradiction. ∎

COROLLARY 8.1. *For any acyclic phenotypical structure and for all nonexceptional fitness vectors the total number of equilibria does not exceed* $3^{|\Gamma|-1}$.

Now we apply Theorem 8.1 to some more special situations. First of all, combining it with Corollary 5.4 we obtain the following

COROLLARY 8.2. *For any multilocus* (*in particular*, *single-locus*) *dominant phenotypical structure the equilibria set is finite generically.*

Similarly, using Proposition 5.1 we get

COROLLARY 8.3. *If the phenotype of every homozygote is specific* (*in particular*, *if the phenotypical structure is separative*) *then the equilibria set is finite generically.*

For the separative phenotypical structure with l linked loci (the case $q = 1$ in (3)) the phenotypical fitness space consists of all nonnegative nonzero symmetric matrices $[\lambda(g, h)]$. According to Corollary 8.3, *except for a proper homogeneous algebraic subset in the matrix space, the fixed point set of the evolutionary operator* (14) (*or* (16)) *is finite.* The Main Theorem provides the same conclusion even if the matrix space is restricted by the relations

$$\varphi(g, h) = \varphi(g', h') \Longrightarrow \lambda(g, h) = \lambda(g', h'),$$

where φ comes from an arbitrary acyclic phenotypical structure.

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