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Sewall Wright meets Artificial Life: the origin and maintenance of evolutionary novelty

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Michael J. Wade

A decade ago, Langton coined the term Artificial Life (A-Life) to identify the new field of research that is attempting to create and characterize open-ended evolving systems using diverse computer-based methods. Fruitful interactions between A-Life research and that of conventional biological sciences (B-Life) are rare. Using the framework of molecular and evolutionary genetics, we discuss some of the reasons for this lack of conceptual cross-pollination between the disciplines and we identify some potential areas for interdisciplinary collaboration.

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'What is life?' and 'what could life be?' are central questions in the interdisciplinary field of Artificial Life (A-Life). It tries to subsume all of the conventional domain of the biological sciences (B-Life) and it can make fundamental contributions to our understanding of B-Life processes[1,2]. However, a decade after the birth of A-Life, there are few reciprocal contributions between the A-Life and B-Life sciences. These two sciences converge most conspicuously in the development of genetic algorithms, which are computer-based models for creating open-ended evolving systems in A-Life in addition to solving complex problems in engineering. Modern texts in evolutionary genetics rarely cite any A-Life publications[3–5] and some biologists have considered these genetic algorithms as computer exercises in pseudo-genetics: *ad hoc* treatments with a fragmentary theoretical basis of B-life genetics. Computer scientists, on the other hand, have found few if any useful theorems in evolutionary genetics that improve the adaptive performance of genetic algorithms or that permit the solution of complex problems (cf. Ref. 6).

We discuss the general structure and findings of genetic algorithms (see definition in Box 1) in the context of classical evolutionary and population genetics. The lack of reciprocal exchange between the fields results from the different questions each is trying to address. These differences in central questions lead the fields to differ in the emphasis placed upon the evolutionary forces that consume or generate genetic and phenotypic variations. Finally, we discuss the conceptual convergence between some A-Life models and the evolutionary genetic theories of Sewall Wright, and indicate some unexplored avenues for two-way interactions between genetic algorithms and population genetics.

Evolutionary and population genetics

The goal of population genetics theory is to characterize and to discriminate among the several evolutionary forces that operate simultaneously in natural populations. The mathematical tools of the theory vary from simple linear algebra to statistical and stochastic diffusion models. A major focus is the balance among evolutionary forces that maintains genetic variation in natural populations. The signature of natural selection is detected by comparison with the predictions of null models where only the neutral forces (mutation and random drift) are operating. Two extreme views coexist within formal evolutionary theory[7].

The fisherian worldview

R.A. Fisher proposed that adaptation typically takes place in large and randomly mating populations[3–5,7,8]. Random drift is negligible in this context and mutation and selection are sufficient to explain patterns of genetic variation. Fisher's fundamental theorem of natural selection (FFT)[8] predicts that the rate of adaptive evolution of a population is determined

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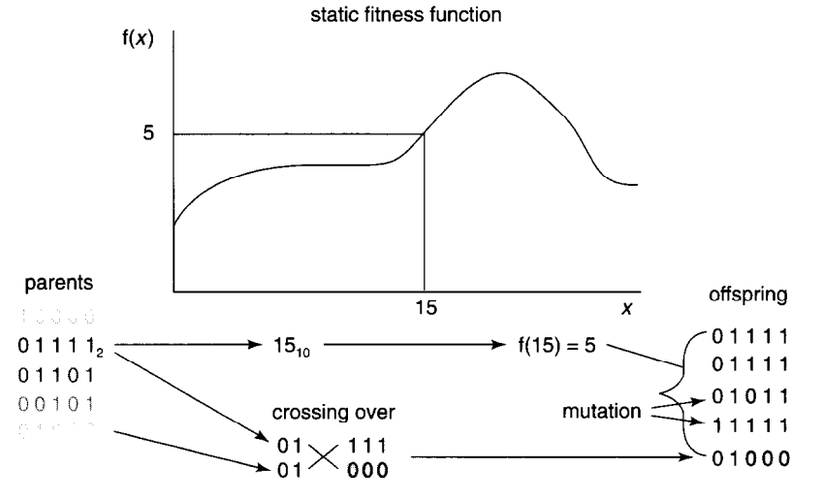
Box 1. Genetic algorithms

There are four major genetic algorithms: Holland's genetic algorithm (GA)[26,27], Koza's genetic programming[28], Fogel's evolutionary programming[29] and Rechenberg's evolutionary strategies[30]. They differ from each other in the mechanisms of genetic coding, the selection procedures, and the genetic operations permitted in reproduction[31–33]. The latter two methods adopt continuous variables rather than discrete bit-string coding, and have the greatest similarity to the quantitative genetics in evolutionary biology.

There are also many hybrids and variants of these four genetic algorithms. In this article, we use the term 'genetic algorithm' to refer to this class of models as opposed to any specific model or variety. These genetic algorithms were invented for the common purpose of solving optimization problems in engineering. Because Holland's GA (Box 2) is the most commonly used, we will discuss its central features, which are representative of the entire class of genetic algorithms, and compare these features with those of real biological systems.

Box 2. Holland's GA

The figure shows the simplest example of Holland's genetic algorithm (GA)[26,27]. Parent bit-strings are decoded into decimal numbers, and the individual '01111' is assigned a reference number of 15. This number is used to obtain a fitness value by referring to the static fitness function [f(*x*)] (shown above). The assigned fitness value is five, hence, the individual produces five offspring. Basically, the offspring are copies of their parents. However, the mutation operation allows bit-flips, and, in our example, two out of five offspring differ from their parents by one bit. The crossing-over operation changes the bit-string downstream of the crossing point. As a result, the bottom-most offspring differs from its parents by three bits. In the next generation, these offspring are decoded and evaluated with respect to the same fitness function as their parents.



by its additive genetic variance for fitness. Mathematical expressions of FFT permit analytical solutions at the expense of some of the flavor of living organisms. Complex adaptations involving the coordinated action of many genes are assembled one gene at a time, by natural selection acting on the average additive effects of alleles[8].

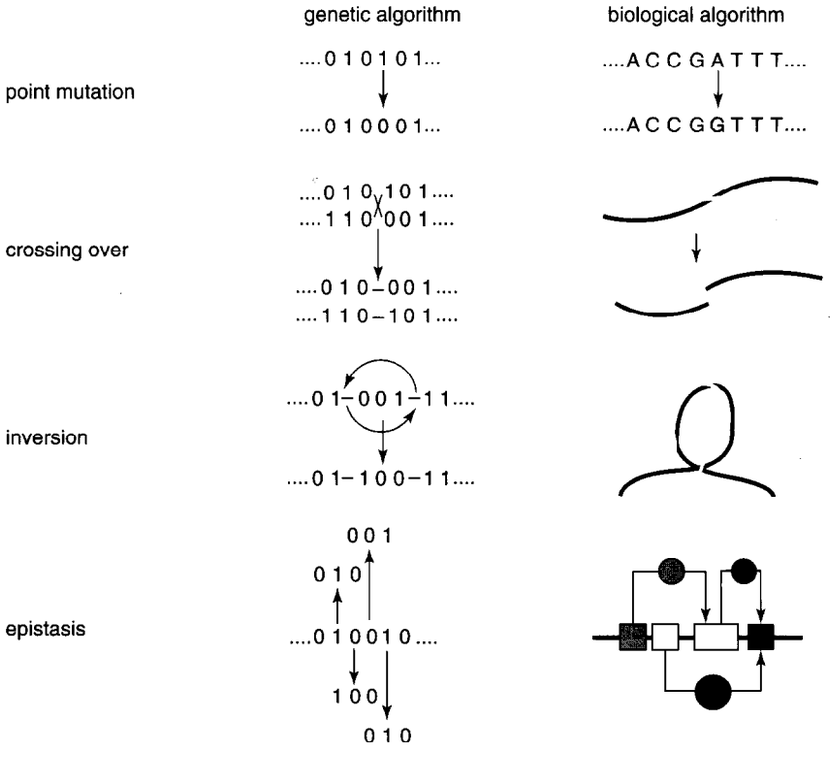
The wrightian worldview

The other extreme view is Wright's shifting balance theory (SBT)[9] describing the origins of evolutionary novelties and speciation in subdivided populations. Finite local demes and epistasis between genes complicate the evolutionary trajectory of a species[8,9]. The scope of Wright's theory is somewhat wider than that of Fisher because random genetic drift is considered an essential feature and there is the additional adaptive force of interdemic selection.

Although FFT acts locally within the SBT, describing selection within every deme, there is also a genetic nonlinearity constituent to individual phenotypes and their interactions. Thus, in the SBT, natural selection is not immediately accorded the dominant role as in FFT. Instead, the importance of the different evolutionary forces depends upon the situation as strong selection at one locus reduces effective size and enhances the probability of loss of alleles at other selected loci[10]. In this sense, the SBT provides only a framework within which evolution occurs as opposed to the better defined, deterministic theory of Fisher. Ordinal mathematical expressions for the nonlinear genetic systems of the SBT can be rendered but are complex. Furthermore, Wright himself considered his widely used method for

Box 3. Comparison of genetic operations between a simple genetic algorithm and biological systems

Point mutation: in Holland's genetic algorithm (GA)[26,27], the mutation operation flips a bit in a bit-string from 1 to 0, or *vice versa*. In biological systems, point mutation changes one nucleotide base sequence into another.
Crossing over: bit-strings and chromosome sequence exchange bits or bases, respectively, downstream of the cross-over point.
Inversion: in Holland's GA[26,27], inversion causes reverse transcription of the inversed part, but the inversion causes the formation of a loop in biological systems that does not tend to be reverse transcribed.
Epistasis: in Kauffman's genetic algorithm[13], each locus refers to other loci when decoded into phenotype. In biological systems, products encoded upstream regulate the transcription of the downstream genes.



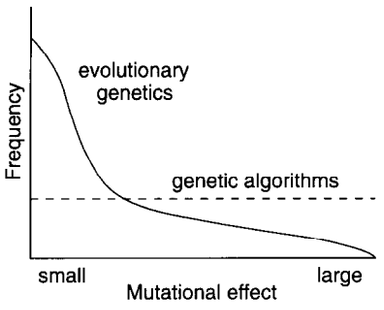


Fig. 1. Mutational effect. In evolutionary genetics most mutation events have minor effects on fitness. In contrast, the mutational effects are uniformly distributed in the rugged landscape of genetic algorithms applied in A-Life.

quantifying population genetic structure (F_{st}) as 'wholly inadequate', since it described only single genes and not gene combinations[9].

Genetic algorithms

Genetic algorithms (Boxes 1 and 2) were developed as a computational device, wherein computer scientists adopted concepts and mechanisms from genetic biology and used them as rules for replication and change in evolving computer life systems. An iterative genetic process might find optimal solution(s) for complex problems more efficiently than other computer algorithms. In A-Life studies, genetic algorithms are used as 'darwinian random generators' in place of optimization techniques. In the simplest genetic algorithms, genotypes are bit-strings consisting of 1's and 0's. These bit-strings undergo operations analogous to the processes that affect haploid mendelian chromosomes like mutation and crossing-over (Boxes 2 and 3). Diploidy and multiple chromosomes are modeled by increasing the number of bit-strings per individual[11].

Sources of variation

Heritable variation in fitness is necessary for darwinian evolution, and the bit-string populations of genetic algorithms have three sources of it. The first resides in the initial distribution of founder bit-strings, which are randomly generated. This is analogous to the 'infinite-sites infinite alleles' models, where each initial allele is assumed to be unique[12]. A second source lies in the genetic operators (Box 3) that create variation at bit-string loci, although this variation is mapped to phenotype in a manner slightly different from the analogous processes in B-Life genetics (Box 3). Lastly, genetic algorithms create new variation by elongation of existing bit-strings, where each one-bit increase doubles the number of possible states. In B-Life models, gene copy numbers are unlikely to increase or decrease by fractional parts of the whole.

Mapping genotype to phenotype

There is wide variation among A-Life genetic models in mapping bit-string to phenotype (Box 1). In engineering applications, one-to-one mappings are common, similar to those of additive quantitative genetics. However, the one-to-one mapping restricts the range of possible phenotypes and limits the power of solving complex problems. To compress larger numbers of phenotypes into a finite bit-string, a variety of nonlinear mappings have been considered. Kauffman's NK-model[13], for example, uses each bit many times to decode phenotypes and realizes highly epistatic and pleiotropic interactions (Box 3). In such nonlinear systems, there is no continuity in the map: very small changes in bit-string result in very large jumps in phenotype space. This differs from B-Life genetics in two ways. First, the distribution of mutational effects on phenotypes is centered near zero, because most mutations are believed to have small effects and are neutral or nearly so (Fig. 1). Second, mutations of large effect are mostly deleterious.

Fitness landscapes

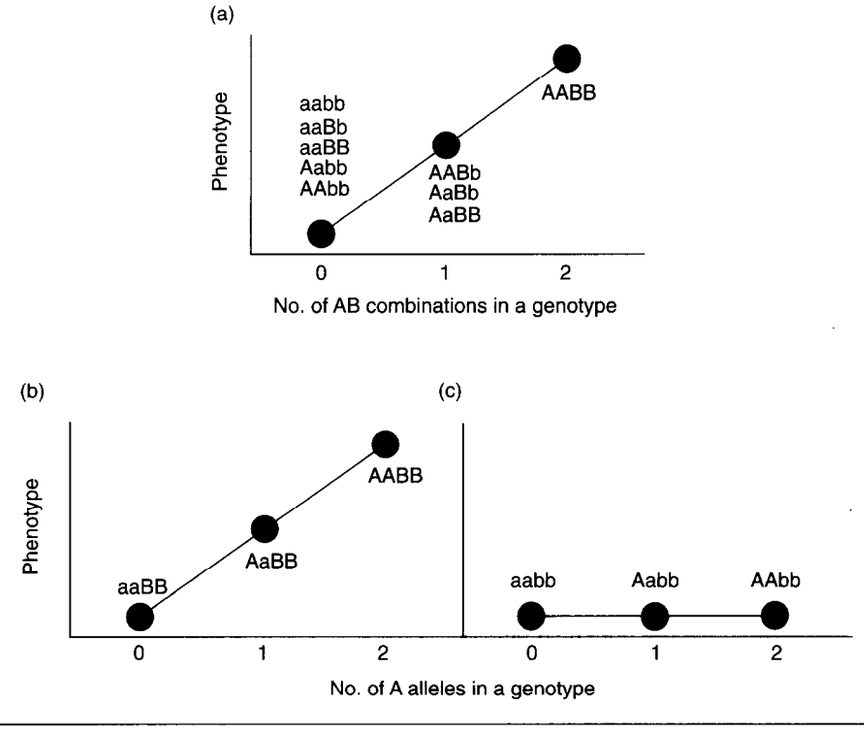
Ray's Tierra[14] model has succeeded in generating open-ended A-Life evolution, despite fixing the number of possible bit-strings because fitness is determined by the interactions among bit-strings, as in real ecosystems, and not an intrinsic property of a bit-string. Thus, the same bit-string can have high or low fitness, depending upon its neighbors. Novel adaptations, such as mutualism and altruism, can occur at the phenotypic level and cooperative or synergistic epistasis can evolve at the genic level. With the fitness scheme of Ray's Tierra, the resulting rugged fitness landscape may permit interdemic selection to affect the adaptive process.

In B-Life genetics, constant gene and genotypic fitnesses may be assumed and restrict dimensionality at the expense of biological reality[15,16]. Even complex gene interactions can be modeled as additive in their fitness and phenotypic effects in the vicinity of an adaptive optimum, where small deviations can be treated as being effectively linear. In contrast, most A-Life systems begin far from the global optimum (whose existence is not guaranteed) and the complexity of the bit-string-to-phenotype map is developed to facilitate

Box 4. Conversion of epistatic to additive genetic variance by random genetic drift

In (a), an individual's phenotypic value increases with the numbers of **AB** gene combinations from small (when the individual lacks either an **A** allele or a **B** allele) to intermediate (for individuals with one **A** and one **B** allele) to large (when the individual has two **A** and two **B** alleles).

When random drift changes allele frequencies at one of two interacting loci, it changes the amount of additive genetic variance segregating at the remaining locus. In (b), **B** is fixed but **A** remains segregating, and the genetic variance appears additive at the **A**-locus. In (c), **b** is fixed but **A** remains segregating, thus no additive variance is associated with the **A**-locus.



finding a solution to the problem at hand, if it exists.

Molecular genetics and evolutionary theory

Bit-strings in genetic algorithms can share four features of the molecular biology of DNA or RNA sequences.

Neutral evolution

Natural selection does not restrict the large majority of sequence substitutions[12,17]. Patterns of synonymous base-pair substitutions, in particular, should be neutral in this sense and tend to support the Neutral Theory of Evolution[12]. More-extended interpretations argue that the pool of neutral genetic variation may permit rapid evolutionary change of phenotype under some circumstances[12].

Evolution by duplication

The discovery of frequent gene duplication and subsequent sequence divergence has contributed to the development of the theory of evolution by duplication[18]. In this theory, a gene(s) may become duplicated and the influence of natural selection on its sequence can either expand or contract. A duplicated gene may sometimes double its enzymatic expression, gaining a selective advantage. Alternatively, one copy may become neutral and temporarily free from selective constraint, with its fate governed for a time by mutations and drift. It may become wholly dysfunctional (a pseudogene) or gain new function. In contrast to neutral theory, gene duplication theory creates free genetic space to explore for novel adaptations.

Quasispecies

Eigen[19] proposed a similar concept for describing evolution from RNA to DNA. He postulated a more or less stable RNA quasispecies consisting of adaptive variants and the vast mutational array around them. A quasispecies was held together by the selective advantage of some coupled pairs over others. A quasispecies of RNA molecules thus persisted until replaced by an adaptively superior constellation of interacting RNA molecules. An expanded information system (DNA) with a reduced error rate evolved from the RNA quasispecies because coupled pairs of variants could achieve what single variants could not. Harvey's species adaptation genetic algorithm (SAGA) is based on this quasispecies hypothesis[20].

Disparity evolution

Between A-Life and B-Life, Furusawa and Doi proposed the idea of disparity evolution[21]. This theory claims that the Y-shaped replication fork in DNA duplication is adaptive because it results in two kinds of strands: (1) the leading strand or masterpiece, duplicated almost without error; and (2) the lagging strand (producing Okazaki fragments), duplicated with a much higher error rate. As a result, after several DNA replications there is a much wider range of mutational variation among offspring strands than is the case when mutations are randomly distributed to both strands. Furthermore, the original, adaptive strand is conserved at the same time that extensive mutational variation is created. This permits the population to explore more widely nearby adaptive space and, at the same time, conserve previously selected gains in the masterpiece. They showed using a genetic algorithm that the rate of adaptive evolution was enhanced by disparity mutations relative to that of a parity mutational process.

Common goals

Figure 2 demonstrates a schematic relationship among evolutionary genetics, molecular biology and genetic algorithms.

Optima: near and far

In A-Life, as opposed to evolutionary or population genetics, the goal is to develop a system that will both find optima and reciprocally change the fitness landscape, making the evolutionary trajectory truly open-ended. In contrast, for the past 40 years, mathematical evolutionary genetics has focused primarily on forces affecting the origin and maintenance of genetic variability within populations that are already at an adaptive optimum. At mutation–selection balance, the dispersive evolutionary forces act as an impediment to adaptation because they move individuals (mutation and segregation) or even whole populations (drift) away from the optimum under natural selection. The method called evolutionarily stable strategy (ESS)[22] is so focused on optimization that it substitutes a non-genetic, game theoretic approach for formal genetic models[23].

A key component of theories in population genetics, molecular genetics, and A-Life genetic algorithms, is the relationship

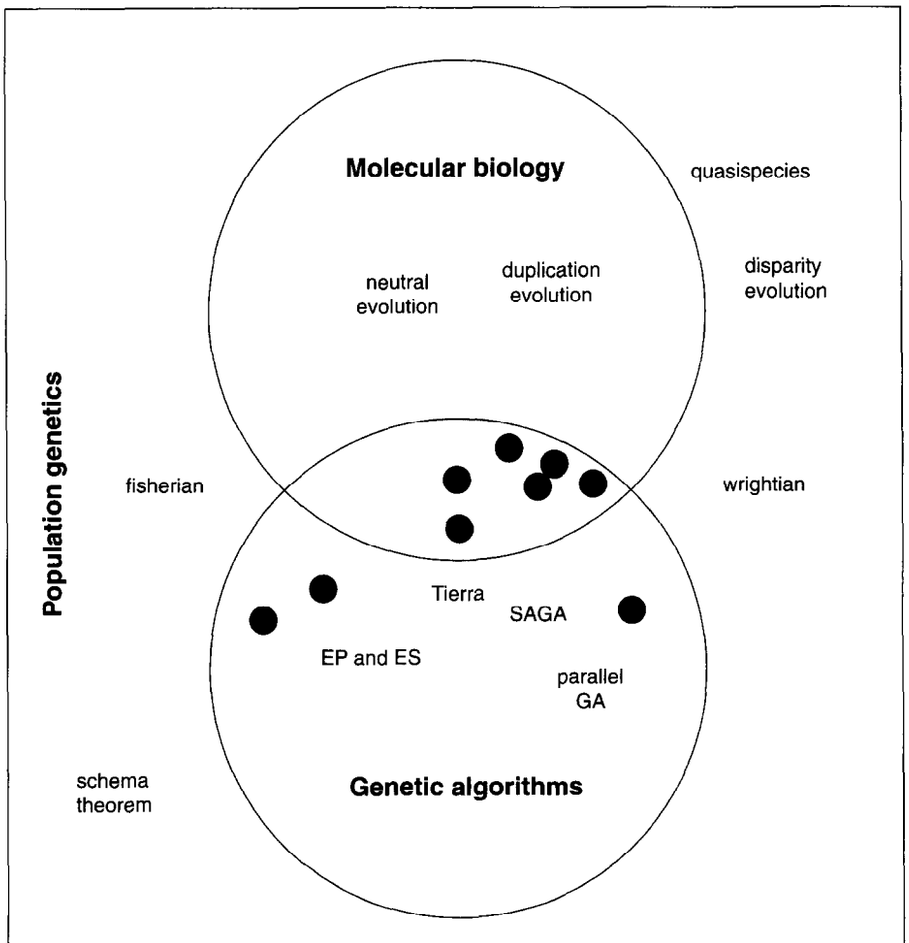


Fig. 2. Schematic relationship among evolutionary genetics, molecular genetics and genetic algorithms. Three domains – population genetics (dumbbell shape), molecular biology (upper circle) and genetic algorithms (lower circle) – are highly overlapping of one another. EP and ES represent Fogel's evolutionary programming and Rechenberg's evolutionary strategies, respectively (Box 1). Parallel GA is a genetic algorithm that has been modified to maximize its performance in a parallel computer architecture. SAGA represents Harvey's species adaptation genetic algorithm[20]. Tierra refers to Ray's Tierra model[14].

between sources of mutational variation and the evolutionary effects of mutations. Fisher and Wright consider classical mutational processes, where novel variants are more likely to reduce adaptation than to enhance it. In these models, there is a balance between (1) purifying natural selection, (2) pushing deleterious mutations out of populations, and (3) mutation reintroducing deleterious variants. There are some differences between FFT and SBT. In FFT, mutational variation is consumed by natural selection, while under SBT it can be transformed by random drift from epistatic to additive variance or from within-deme to among-deme variance[8,24,25] (Box 4). In contrast, in A-Life models, mutations of large effect play an essential creative role in exploring the fitness landscape, which itself can be quite rugged, lacking the continuity of B-Life systems.

Efficient adaptive walks

The strong message for successful ongoing adaptive evolution from the A-Life genetic algorithms is 'Keep a masterpiece and, at the same time, continue to explore adaptive space with mutational variants.' If a better local optimum is found, then change the masterpiece to the variant and restart the procedure again. Some theories emphasize the exploring phase at the expense of selected gains (e.g. Kauffman's NK-model), while in others the reverse is emphasized (Schema Theorem[26,27]).

Wright's SBT is very similar in broad concept to these A-Life theories. In the SBT, random and directed processes give rise to adaptive gene combinations in local demes within a metapopulation. The better the local gene combination for adaptation, the greater the net emigration of individuals out of the local deme and into neighboring demes. Thus, the storage of selected gains is accomplished by protecting the favorable gene combination within a deme from scrambling by recombination and from dilution by immigration, because the deme becomes a net sender as opposed to receiver of migrants. At the same time, the metapopulation continues to prospect for novel gene combinations of higher adaptive value by the local action of random drift, mutation, and selection within demes.

In Fisher's scheme, selection cannot act directly upon interacting systems of cooperatively acting genes because of the opposing effect of recombination. Genes are heritable, while gene combinations are not. In large populations the average effect of a gene on fitness determines its evolutionary fate, and there is no way for the population to pass through a point of lowered fitness in order to reach a favorable gene combination. Fisher's process will climb to a local optimum but will not be able to find some adaptive gene combinations, if the fitness landscape is rugged. The emphasis in Fisher's worldview is on conserving prior adaptations and on discovering them gradually in small increments with optimal mutation and recombination rates as opposed to discovering evolutionary novelties.

Like Wright, Eigen[19] criticized the fisherian process for the origin of life because he felt that competitive natural selection alone could not have developed cooperative molecular pathways from prebiotic systems. The 'interplay of molecular competition and cooperation is necessary to process and utilize the first genetic information' and it is essential for stabilizing the earliest hereditary processes. In almost identical words, Wright viewed gene interaction as essential to complex adaptations and proposed his SBT to permit the direct selection of adaptive gene combinations.

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