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On the "Origin" of Natural Selection by Means of Speciation

Key words. Evolution, selection, heritability, fitness, punctuated equilibrium.

Abstract. *A new model of evolution of adaptive traits in sexual species is suggested. As the expression of most phenetic traits is codetermined by many genes and the influence of a gene on the phenotypic trait is often gene-context specific, the heritability of phenetic traits in outbreeding populations is often low. Also the effect of expression of a single trait on biological fitness is often context specific being determined by the presence and level of expression of other phenetic traits. Because of the low heritability of phenetic traits and of biological fitness the potential for adaptive evolutionary change by means of natural selection is very limited in sexual species. However, most mechanisms of speciation lead to drastic reduction of genetic variance in the new species. In the absence of genetic variance genetic context for a new mutation is always the same and the heritability of phenetic traits and of biological fitness increases. After a renewing of genetic variance by mutation processes the heritability of traits and fitness again decreases. In the history of any sexual species a transient period of evolutionary plasticity in which the species can respond to natural selection and evolve new phenetic traits is followed by a long period of evolutionary stasis in which the species accumulates changes mostly by genetic drift and molecular or meiotic drive.*

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1. DARWIN'S MODEL OF EVOLUTION

Darwinian evolutionary theory supposes that complex adaptations, which are so typical for living organisms, originate and evolve by means of natural selection. Different variants (individuals with hereditary modifications - mutations) randomly occur in any population. The individuals with a beneficial mutation have better chance to survive in intraspecies competition and produce more offspring. The mutations, i.e., the changes in the DNA, are copied in the process of DNA replication and transmitted to next generation in the process of reproduction. As the next-generation individuals with beneficial mutation have also the better chance to survive and reproduce they also produce more offspring. Consequently, the frequency of the beneficial mutation increases from generation to generation. Finally, all individuals bear the new mutation and possess a new beneficial property. The mutation is fixed. New mutations continuously originate in the population and those that are beneficial are being fixed. Therefore, the populations and species continuously evolve and new adaptive properties, organs, processes as well as behavioral patterns are being developed in the history of life.

In Charles Darwin's time this model represented a beautiful hypothesis deduced on the basis of analogy with artificial selection of varieties of domestic animals and cultivated plants and possibly also with competition between economical subjects of developing industrial society. Today the Darwinian model is generally accepted in the whole scientific community. Most of the processes playing role in it were studied into deepest details and their molecular nature was revealed. We understand the mechanisms of storage and transfer of genetic information as well as the processes of the origin of new mutations. On the basis of both an elementary thinking and sophisticated mathematical analyses it seems evident that any systems functioning on the same principles as the biological systems do cannot but evolve.

2. VANISHING INHERITANCE OF QUANTITATIVE TRAITS IN SEXUAL SPECIES

Only a few current biologists (Williams [1975]; Maynard Smith [1978]; Dawkins [1982]) are aware or openly admit that this model of evolution can smoothly work only in the absence of sexual reproduction. In sexual organisms, i.e., in most organisms living in our world, there is a fundamental problem concerning the heritability of genetically coded phenetic traits. This heritability represents a necessary condition for Darwinian evolution. In asexual species, the heritability can be practically absolute. In the absence of environmental (nongenetic) variation, the offspring's phenotypes are often identical with the phenotype of their parent. In the sexual species, a genotype of the offspring represents a unique combination of genes from two parents. Therefore, their phenotypes are in most of characters intermediate between these two parents. If one parent has a new mutation that results in a new quantitative phenetic trait then the level of phenotypical expression of this trait in the offspring is usually different from in its parent, probably somewhere between its expression in the mother and father. It is the most frustrating experience of animal and plant breeders that in the succeeding generations a phenotype of outbred offspring with the new mutation is less and less similar to the original mutant and finally might even return to the population norm. The problem of this 'vanishing' heritability was known to Darwin and was widely discussed by his critiques. The tremendous amount of empirical evidence for this kind of heritability of phenetic traits led him (and others) to accept a hypothesis of blending inheritance. It supposes that the sexual reproduction is accompanied by some sort of fusion rather then mixing of properties (or the properties-determining factors) of the two parents (Mayr [1982]). The logical consequence of the acceptance of blending inheritance is a rapid loss of any heritable variance and therefore a necessity to postulate unrealistically high rate of new variance creation (Fisher [1958]). A rediscovery of Mendel's work and his model of factorial (particulate) inheritance solved this problem. When the heritability of factors (genes) is discrete, there is no inherent tendency for the variance to diminish.

3. MECHANISM OF THE EVOLUTIONARY STASIS

A victory of the particulate inheritance model solved the problem of the loss of heritable variance. However, the second aspect of the problem of vanishing inheritance, namely the question of low heritability of quantitative phenetic characters remains. The fact that the factors (genes) are being transmitted between generations in an unchanged form does not imply that the same is also true for phenetic characters. Virtually no phenetic trait is determined by only a single gene (Lander & Schork [1994]). Most of phenetic traits are codetermined by many genes, and the typical quantitative traits by a huge number of genes (Summers [1996]). The tens of thousands of genes in a genome of a modern organism are directly or indirectly interconnected into one functional network and therefore the ontogeny of any phenetic trait can be influenced by any gene (Wagner [1996]). Of course, most of the traits are determined mainly by a few genes of large effect that can explain for example 75% of population variability of the trait. The remaining part of the variability could be determined by hundreds of genes of small effect (Rasmuson [1996]) which themselves are engaged as genes of large effect in the ontogeny of other traits (Thompson [1975]). To make things worse, the effects of genes are not always additive. It is probably common that the same allele of a gene A in the presence of one allele of a gene B influences the expression of the trait positively while in the presence of another allele of the gene B influences the expression of the same trait negatively (Cheverud & Routman [1996]). Because of such epistatic interactions, the influence of a gene on phenotypic traits is sensitive to the context of other genes in a genome. Moreover, even the influence of the phenetic trait on the fitness of organism is context sensitive. The presence of the same phenetic trait in the context of one phenetic trait could be beneficial while in the presence of another phenetic trait it could deteriorate the chance of the organism to survive in intraspecies competition (Wainscoat *et al.* [1983]). The problem of context sensitivity of gene expression was described by the classic of modern population genetics R.A. Fisher [1958]. He discussed the problem of genes modificators which usually rapidly mask any effect of new and sharply distinct

mutations in laboratory stocks of *Drosophila, Nasturtium* or *Gammarus.* A mating of modified mutants to unrelated wild stock and extracting the original sharply distinct form of mutant from hybrids by inbreeding shows that the modification has not been due to any change in the mutant gene, but due to a change in the genetic complex of the organism with which it reacts. The universality of the phenomenon of genetic context sensitivity of the gene effects has been confirmed by results of genetic canalization studies (Waddington [1942], [1959]; Wilkins [1997]) as well as by modern experience with transgenic or knock-outed organisms (Allen *et al.* [1990]; Schuster-Gossler *et al.* [1996]) and genetic diseases of man (Rasmuson [1996]; Summers [1996]).

When a phenetic trait is determined not by a single gene but rather by many genes of small effect then the inheritance of the trait must be not only blending but also vanishing. The descendant of two outcrossing individuals has only 50% genes identical-by-descent with its parent. Therefore the mutation that is beneficial for the parent occurs in a drastically new genetic context and the probability of it having an identical effect here as in the original context is highly reduced. In the next generation, the proportion of identical-by-descent genes decreases down to 25% and similarly decreases also the chance for identical phenotypical effect of the mutation. Within a few generations the unique combination of genes, which determines the unique phenetic trait, is outdiluted and the unique trait can disappear from the population. The mutation, which was responsible for the original occurrence of the trait, however, still persists there and can reveal its presence whenever it occurs in the genomes of possibly rather unrelated individuals with proper combination of alleles in their genomes.

Accepting the facts that because of the genetic context sensitivity of a gene effect the heritability of phenetic trait is low, and that because of whole-phenotype context sensitivity of effect of the phenetic trait on the organism's fitness the heritability of fitness is very low, if any, we have to abandon the classical model of neodarwinian evolution in sexual organisms. To allow the fixation of a context sensitive trait in an outcrossing population by means of natural selection its effect on the fitness would need to be unrealistically high. Natural selection can fix an unconditionally benefi-

cial trait coded by a single gene or by a few genes with strictly additive effects. It is not clear what role such genes of major effects can play in biological evolution. The mutations with severe phenotypical effect are easily obtained and studied by approaches of classical and molecular genetics. However, the nature of such mutations including those studied by Mendel [1966] is mostly an inactivation of some gene or of its product (Bhattacharyya [1990], Burrell [1997]). It is highly questionable what role this type of mutation can play in evolution, for a discussion see ([Bishop [1996]). On the other hand, most new traits that evidently played a role in the evolution of sexual organisms, i.e., increase or decrease of the size, changes of shape or proportions of organs are usually determined by many genes of a small effect. The evolution of biochemical or cellular functions probably occurred before the advent of multicellular organisms and in the absence of sexual processes. Therefore, these functions could evolve by standard neodarwinian mechanism. However, most of multicellular organisms' evolution should proceed by a different mechanism.

4. MECHANISM OF THE EVOLUTIONARY PLASTICITY

One solution of the paradox of rapid evolution of multicellular organisms under conditions of strongly restricted heritability of fitness and consequently highly restricted effectiveness of natural selection has been suggested by Dawkins [1976]. According to his model of the selfish gene the criterion for an ultimate fixation of a new allele is not its beneficence for the fitness of the organisms but its ability to outcompete the other alleles of the same gene in the respect of a rate of transmission into new generations. The competition for higher fitness between the organisms is substituted by a similar competition between different alleles of one locus. As the individual gene (in the Dawkins definition) is a short piece of DNA, it is usually transmitted unchanged by process of recombination even during the sexual reproduction. The model could work under the conditions of absence of any nonadditive epistatic effects of genes. Under the actual situation when the same allele is favored in one and handicapped in another genetic context the

heritability of the 'fitness' of individual recombination-resistant allele is similarly as low as the heritability of the fitness of the recombination-sensitive organisms.

Finding conditions under which the heritability of phenotypic traits and heritability of fitness is sufficiently high can solve the paradox. Such conditions do not exist in outcrossing populations with the natural level of genetic variance. However, they do exist in populations with low level of genetic variance. Under natural conditions, such populations can originate due to colonization of a new geographic area or drastic reduction of population size (bottleneck effect) or by extremely assortative mating (Goodnight [1987]; Whitlock *et al.* [1993]). In these populations, the genetic variance of the original population is drastically reduced by the founder effect and the subsequent effects of inbreeding. In asexual species with reduced genetic variance the effectiveness of natural selection and the rate of evolution is decreased as the selection has only limited reservoir of variants to select from. In sexual organisms the final effect of the variance decrease is very opposite, i.e., an increase of the rate of evolution. In the absence of genetic variance, the genomes of all individuals are same. Therefore, any new mutation occurs in offspring in the same (or very similar) genetic context as in the parent organism and the phenotypical effect of the mutation, as well as its effect on the fitness of any individual are the same. This means that the heritability of phenotypic traits and fitness of organism is high and the mechanism of neodarwinian evolution can operate generating evolutionary changes. Eventually, the natural level of genetic variance is renewed by mutation processes and the heritability of phenetic traits and of fitness decreases down to its original level.

It is well known, of course, that even the polymorphic populations can respond to selection pressure in an experiment. However, the response is known to be only transient and often reversible. Within a large, genetic-equilibrium population the frequency of various alleles is the result of dynamic competition of alleles with frequency-dependent selection coefficients (which can be predicted by an Evolutionarily Stable Strategies (ESS) approach). When we are putting a new selection pressure, we are in fact changing the pay-off matrix of the evolutionary game. The popu-

lation must respond to such changes by shifting the equilibrium frequencies of particular alleles. After the end of the experiment (after several generations without the selection pressure and without any breeding program) the frequencies of alleles slowly return toward their original values. Of course, in small populations some alleles have been lost during the selection period, therefore the return cannot be perfect. In contrasts, the selection-induced changes in the large panmictic populations can be truly reversible.

It is important to stress that the same allopatric and sympatric isolation mechanisms that are considered to be a cause of cladogenetic processes of speciation are also responsible in sexual organisms for the functioning of anagenetic processes of origin of evolutionary novelties by means of natural selection. The proposed model shows that in sexual species the origin and development of new adaptive properties by means of natural selection are tightly associated with the origin of new species. Most of these anagenetic changes are probably concentrated only within the earliest period of the existence of a new species. After the accumulation of genetic variance the evolutionary plasticity of a species decreases or disappears. The species loses the ability to adaptively respond to changes of biotic and abiotic environment and for the rest of its sometimes long existence only passively waits for its terminal extinction.

5. THE EVIDENCE FOR THE FROZEN PLASTICITY MODEL OF EVOLUTION

Increase of heritability in an inbred population has been theoretically predicted (Robertson [1952]; Templeton [1980]; Goodnight [1987]; Whitlock *et al.* [1993]) and empirically confirmed in several experimental systems (Katz & Young [1975]; Bryant *et al.* [1986]; Bryant, McCommas & Combs [1986]; Lopez-Fanjul & Villaverde [1989]). In many systems, the effect of higher evolutionary potential of population with decreased level of variance was also observed (Bryant *et al.* [1986]; Lopez-Fanjul & Villaverde [1989]; Wade *et al.* [1996]). Sometimes, however, the evolution-

ary potential positively correlated with the amounts of variance (Wade *et al.* [1996]; Mcvean & Hurst [1997]). The decrease of the potential was in an agreement with predictions of neodarwinian model but in contrast with predictions of the model of the vanishing heritability in polymorphic populations. It can be explained by the fact that the size and therefore the amounts of genetic variance of experimental populations are usually limited and therefore the heritability of phenetic traits is higher than under natural conditions (a similar argument also holds for the effectiveness of artificial selection). In short-term experiments only a fixation of old mutations, which are already present in polymorphic but absent in nonpolymorphic population, can be observed. Therefore, only the polymorphic population can respond to selection pressure. To test which model better describes the evolution of real organisms we should introduce the same beneficial mutation into polymorphic and inbred populations and compare the rates of its fixation.

The increase of rate of evolutionary (anagenetic) changes in the isolated populations has been observed in nature. The most divergent subspecies (including the largest and smallest ones) have been usually found on islands (Sondaar [1977]; Angerbjorn [1986]; Corbet [1986]). This contrasts with the predictions of Kimura's model of selection which suggests that the highest effectiveness of natural selection (higher probability of fixation of beneficial mutation) should be expected in subspecies with larger effective population (Kimura [1995], [1983]). This disagreement between the facts and theory is usually explained by postulating that most of the evolutionary changes in small populations were caused by fixation of slightly deleterious mutations by genetic drift (Ohta [1996]). Such mutations are effectively neutral in small populations but are selected against in larger populations. However, many changes observed in isolated populations, such as the increase or decrease of size, seem hardly selectively neutral and the rate of their fixation is unrealistically high for the genetic drift (Sondaar [1977]). Analysis of recently published results of experimental colonization of islands with *Anolis* lizards (Losos *et al.* [1997]) shows that the rate of microevolutionary change correlates negatively with the Founding-population size. It is very important

that the morphological changes observed in this experiment were of different intensity but in the same direction. Therefore, they can be promoted by relatively deterministic process of natural selection, rather than by stochastic process of genetic drift.

The largest amount of evidence for the model of vanishing heritability is accumulated in paleontological research. Paleontological data mostly support a punctualist picture of evolution of multicellular organisms (Gould & Eldredge [1993]) in which morphological changes of species are associated with the moments of speciation and for the rest of their existence an evolutionary stasis, i.e., the absence of anagenetic changes is characteristic. This contradicts gradualistic picture of evolution deduced from the neodarwinian model which supposed that most morphological changes should occur due to intraspecific selection operating during the whole existence of the species. According to this model most of adaptive changes should occur in large populations where the effectiveness of natural selection is the highest (Kimura [1983]). The cladogenesis and anagenesis should be two independent processes. In contrast the vanishing heritability model suggests that the capacity to evolve a new adaptive trait in a large stabilized population with a natural level of variance is highly limited, if any. Most of adaptive changes in sexual species can evolve in small genetically isolated populations under conditions of decreased variance and increased heritability of fitness, i.e., in the earliest period of the existence of a new species.

Finally, the model also explains the association between major evolutionary changes and mass extinctions (Raup [1994]). In the period of mass extinction, the population size of many species is drastically reduced. This results in the decrease of population variance and following increase of heritability of fitness. After the mass extinction the size of populations returns quickly while the variance slowly toward its original value. Consequently, evolutionary plasticity and potential of the species that passed through the bottleneck of mass extinction is transiently renewed. The phenomenon of the evolutionary burst after the mass extinction is again in contradiction with implications of neodarwinian model of evolution by means of (intraspecies) natural selection. After the end of mass extinction the population sizes of most species probably

grow. In this period the intensity of intraspecific competition and consequently of the selection and evolution would decrease rather than increase.

The present hypothesis represents an alternative model for evolution of adaptive traits in sexual species. It suggests a solution for various biological problems that until now resisted explanation within a framework of neodarwinian theories. A corner stone of the hypothesis is a model of vanishing heritability of phenetic traits and biological fitness in outcrossing populations. From the time of the victory of Modern Synthesis in evolutionary biology, the "hard" heritability of phenetic traits has rarely been doubted and virtually never seriously tested. The current methods of estimation of heritability of phenetic traits measure the correlation between expression of the trait in close relatives, i.e., the expression of the trait in the context of very similar genomes. Therefore, these methods cannot reveal the vanishing nature of biological heritability. The experiments studying the rate of evolutionary change in small and large or inbred and outbred populations do not distinguish between the short term effect of fixation already present genetic variance and the long term effect of higher heritability of fitness and better chance of fixation of new mutations. All these technical problems, however, can be easily avoided and the question of existence and prevalence of vanishing heritability of phenetic traits and biological fitness can be experimentally resolved.

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SULL'ORIGINE DELLA SELEZIONE NATURALE PER MEZZO DELLA SPECIAZIONE

Riassunto

La teoria darwiniana dell'evoluzione suppone che i complessi adattamenti originino e si evolvano a mezzo della selezione naturale. È solo dopo la speciazione, tuttavia, che i meccanismi selettivi possono operare.

Nel presente lavoro è proposto un nuovo modello di evoluzione dei caratteri adattativi nelle specie sessuate. Poiché l'espressione di molti caratteri fenetici è co-determinata da molti geni e l'influenza di un gene su un carattere fenotipico è spesso specifica del contesto genico, l'ereditabilità dei caratteri fenetici in specie eso-incrociate è spesso bassa. Anche l'effetto dell'espressione dei singoli caratteri sull'adattamento biologico è spesso contesto-dipendente, essendo determinato dalla presenza e dal livello di espressione di altri caratteri fenetici. A causa della bassa ereditabilità dei caratteri fenetici e dell'adattamento biologico, la potenzialità per cambiamenti evolutivi adattativi a mezzo della selezione naturale è bassa nelle specie sessuate. Inoltre, molti meccanismi di speciazione con-

ducono ad una drastica riduzione della varianza genetica delle nuove specie. In assenza di variabilità, il contesto genetico per una nuova mutazione è sempre lo stesso e cresce l'ereditabilità dei caratteri fenetici e dell'adattamento biologico. Dopo il rinnovamento della varianza genetica per accumulo di mutazioni, i processi di ereditabilità dei caratteri e di adattamento diminuiscono nuovamente. Nella storia di qualunque specie sessuata si ha un periodo transeunte di plasticità evolutiva in cui la nuova specie può rispondere alla selezione naturale e evolvere nuovi caratteri fenetici; questo è seguito da un lungo periodo di stasi evolutiva in cui la specie accumula cambiamenti soprattutto attraverso la deriva genetica e la spinta meiotica.