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Adaptive evolution without natural selection

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A mechanism of evolution that ensures adaptive changes without the obligatory role of natural selection is described. According to this mechanism, the first event is a plastic adaptive change (change of phenotype), followed by stochastic genetic change which makes the transformation irreversible. This mechanism is similar to the organic selection mechanism as proposed by Baldwin, Lloyd Morgan and Osborn in the 1890s and later developed by Waddington, but considerably updated according to contemporary knowledge to demonstrate its independence from natural selection. Conversely, in the neo-Darwinian mechanism, the first event is random genetic change, followed by a new phenotype and natural selection or differential reproduction of genotypes. Due to the role of semiosis in the decisive first step of the mechanism described here (the ontogenic adaptation, or rearrangement of gene expression patterns and profile), it could be called a semiotic mechanism of evolution. *Biological Journal of the Linnean Society*, 2014, **112**, 287–294.

ADDITIONAL KEYWORDS: Baldwin effect – epigenetic turn – ontogenetic adaptation – organic selection – semiotic theory of evolution.

INTRODUCTION

On 13 April 1896, H. F. Osborn gave a lecture at the meeting of the Section of Biology of the New York Academy of Sciences, titled 'A mode of evolution requiring neither natural selection nor the inheritance of acquired characteristics' (Kemp, 1896: 148). According to the neo-Darwinian theory of evolution, such a mode is possible only as neutral evolution. Adaptive evolution has been explained in neo-Darwinism exclusively via the obligatory role of natural selection.

However, it can be demonstrated that such an adaptive mechanism of evolution without natural selection is theoretically possible, and may play a significant role in evolution. Evidence for this can be found particularly in recent studies in developmental biology, where there have been attempts to make the 'epigenetic turn' (Jablonka & Lamb, 2009; and already Ho & Saunders, 1979) integral to the biological worldview (analogously to the former epigenetic turn of the early 19th century that owed much to the work of K. E. von Baer). [Cf. Muller-Sievers (1997).] The current approach has also been referred to as the post-Darwinian view (see, for example, Kull, 1999a, b).

For example, West-Eberhard (2003: 526) writes: 'By the plasticity hypothesis, divergence, in the form of alternative phenotypes, life-stage differences, and contrasting traits such as those expressed under extreme or novel conditions, arises first; then particular variants are fixed in particular subpopulations due to assortative mating, environmentally mediated change in expression, or selection. . . . Extreme plasticity such as learning can produce exceedingly rapid (abrupt) speciation.'

The recent literature on evolution provides many examples of a similar kind (e.g. Weingarten, 1993; Maturana & Mpodozis, 1999; Margulis & Sagan, 2002; Speybroeck, Vijver & De Waele, 2002; Weber & Depew, 2003; Bateson, 2004; Jablonka & Lamb, 2005, 2008; Noble, 2006; Pigliucci & Müller, 2010). Analogous conclusions have been made on the basis of artificial life studies focused on the Baldwin effect (Belew & Mitchell, 1996; Turney, Whitley &

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Anderson, 1996). However, to be certain that we do have here an evolutionary mechanism that is non-neo-Darwinian, it is necessary to demonstrate that a phenotypic change can become genetically fixed via *random* genetic changes and without differential reproduction, i.e. without natural selection, in the strict sense. This is the main point of the current article.

THE MEANING OF 'ADAPTIVE'

'What is it that qualifies a particular dynamical behavior, a change of state, as adaptive?' (Rosen, 1999: 309). This question has not been simple to solve in biology.

Adaptation means making something suitable for a use, or becoming adjusted to something – 'a feature for a particular utility' (Gould, 2002: 1230). Turning a non-suitable situation into a suitable (adaptive) one means solving a certain problem that an organism is facing. Finding a suitable solution in the situation of indeterminacy – this is what an adaptation process does. Thus, we can state that adaptation is primarily a qualitative change or, as a product of this process, a qualitative feature. We call a change adaptive if it solves some problem a living being faces, i.e. if it *turns certain incompatibility into a compatibility*.

This definition is applicable both for ontogenic (reversible) and for phylogenic (irreversible) adaptation. In other words, inheritance and reversibility can be analysed separately from the adaptive change itself. Therefore, it is sufficient to use the qualitative concept of adaptation when describing evolution.

The quantitative study of adaptiveness has led to the concept of fitness as measured by comparing relative reproductive rates. Since Sewall Wright's work (Wright, 1932), the concept of the fitness landscape has been widely used in such studies. However, when using a common quantitative measure alone and thus reducing the change exclusively into the change of reproduction rate (i.e. if replacing adaptation by fitness), we inevitably eliminate the functional content of adaptation, and thus its essential meaning. Here we can observe an analogy with the concept of information – when measured in bits, the semantic aspect becomes lost.

Strictly speaking, adaptation is never just a matter of number. Meaning of a trait is not a derivative from its frequency of occurrence. When speaking about adaptation, biologists almost always had in mind one or other concrete functional relationship. These functional relationships, being local and concrete, do not have any common quantitative measure – and they really cannot have such, due to their qualitative relational nature. Still wanting to find a common measure, however, the concept of measuring fitness has been worked out and widely applied – thus forgetting about proper adaptation as such. Adaptation is a meaningful relation for a living system, which is defined independently from the process of evolution. An adaptation is evolutionary only if it is irreversible, as evolution is defined as an irreversible transformation, in concordance with Dollo's rule.

Nevertheless, our further analysis will not depend much on whether the concept of adaptation used is qualitative or quantitative. We need this concept mainly to distinguish between adaptive and neutral modes of evolution.

ORGANIC SELECTION, ORGANIC CHOICE, PLASTICITY

In 1897, H. F. Osborn published an article with the title 'Organic selection', in which we read: 'Organic selection is the term proposed by Professor Baldwin and adopted by Professor Morgan and myself for this process in nature which is believed to be one of the true causes of definite or determinate variation. The hypothesis is briefly as follows: That ontogenic adaptation is of a very profound character. It enables animals and plants to survive very critical changes in their environment. Thus all the individuals of a race are similarly modified over such long periods of time that very gradually congenital or phylogenetic variations, which happen to coincide with the ontogenetic adaptive variations, are selected. Thus there would result an apparent but not real transmission, of acquired characters. This hypothesis, if it has no limitations, brings about a very unexpected harmony between the Lamarckian and Darwinian aspects of evolution, by mutual concessions upon the part of the essential positions of both theories. While it abandons the transmission of acquired characters, it places individual adaptation first, and fortuitous variations second, as Lamarckians have always contended, instead of placing survival conditions by fortuitous variations first and foremost, as selectionists have contended' (Osborn, 1897: 584).

Organic plasticity – the ability to solve unexpected problems, to accommodate one's behaviour during the lifetime, according to circumstances – is a universal feature of all living beings. This feature does not require an additional evolutionary explanation because it is as universal as life itself (West-Eberhard, 2003: 34). Once alive, organisms cannot avoid fulfilling their organic needs and, by doing so, they cannot completely avoid learning. [Thus, learning can be seen as a means for achieving adaptation. There exist several different mechanisms of learning. One may assume that ontogenetic learning is based on a mechanism that is analogous to

natural selection at the intraorganismal or histological level; however, this is not generally the case (see, for example, Watson *et al*., 2010; Kirby, 2000).] Organic plasticity (as different from transformations in non-living systems) should be understood as a change that has alternatives – it should be possible also to behave in the ways that do not meet the needs, it should be possible to make errors. In this case we can say that organic selection – or rather, organic choice made by organisms – is inevitable. Where a population of organisms is facing a shared change of conditions, all organisms in the population may respond simultaneously and in a similar way. However, the role of organic plasticity in evolution depends on the mechanisms that may make the results of organic choice irreversible.

In fact, Osborn, in the article about organic selection, continues: 'This hypothesis has been endorsed by Alfred Wallace. It appears to me, however, that it is subject to limitations and exceptions which go far to nullify its universal application. This is especially seen in the fact that the law of determinate variation is observed to operate with equal force in certain structures, such as the teeth, which are not improved by individual use or exercise, as in structures which are so improved' (Osborn, 1897: 584–585). To understand this hesitation (which has been continuously used in the interpretations of the Baldwin effect), I should point out that there was almost no knowledge about the dynamics of gene expression patterns until recent decades.

Regardless, we should distinguish here between two statements:

1. The organic selection mechanism is a mechanism that is different from the natural selection mechanism. They are probably both at work in evolution, but their relative roles have to be discovered by empirical studies. If so, then it is theoretically possible that on some occasions an adaptive evolutionary change can take place without natural selection.
2. Organic selection is a possible constituent part of the evolutionary mechanism, the other part of the same mechanism being natural selection. In this case it may be that natural selection is never absent in an adaptive evolutionary change.

Indeed, the advocates of organic selection mostly could not completely avoid involvement of natural selection in the mechanism they proposed. This is true for most interpretations of organic selection, or the Baldwin effect, by H. F. Osborn and his contemporaries (see also Bowler, 1983), of genetic assimilation as described by C. H. Waddington (Waddington, 1953a, b, 1956), of niche-construction as described by Odling-Smee, Laland & Feldman (2003), or of epigenetic evolution by Jablonka & Lamb (2005). Thus, they all seem to accept (2), but not (1). [However, there exists some work that makes the radical claim, similar to the view expressed here, that natural selection is not necessary for adaptation – see, for example, Jablonka & Lamb (2008), Margulis & Sagan (2002), and Watson *et al*. (2010).] Let me now argue for the possibility of accepting (1).

NATURAL SELECTION

To make a strong conclusion about natural selection, we require a very strict definition of this term. The term has often been used rather loosely, which has inhibited the possibility of analysing logical alternatives to the mechanism of natural selection.

I use here the most traditional definition: natural selection is the *differential reproduction of genotypes*. 'Natural selection [. . .] is the differential and non-random reproduction of different alternative alleles in a population' (Grant, 1985: 88); 'population genetics and modern evolutionary theory equate natural selection with differential reproduction of alternative forms of genes, genotypes, or other reproducible units' (Grant, 1985: 91). Thus, natural selection is the gradual, non-random process by which genetically inherited traits (alleles) become either more or less common in a population due to non-random differences in the effective reproductive rate of bearers of these genetic traits.

Thus, to check the existence of natural selection, we should (1) divide a population into subpopulations on the basis of alleles of a certain gene, i.e. by the genotypes; (2) measure the reproduction rates for each subpopulation (i.e. the number of offspring per capita for each genotype); (3) test the significance of the differences in reproduction rates between subpopulations (i.e. genotypes). If and only if the average reproduction rates between the genotypes are statistically significantly different, then there is natural selection. If the differences between the reproduction rates of the genotypes are statistically insignificant (i.e. random), then natural selection is not in operation.

It is important to note that the death of a single genetically unique individual due to any reason is not a case of natural selection. This is because the definition tells about *genotypes*, not *genotokens*, i.e. about classes, not individuals. We can assign natural selection as a reason for deaths only if the deaths occur as a regular result of a certain genetically inheritable trait (allelic difference), but this requires several individuals with this trait. Natural selection requires a statistically significant decrease or increase in the number of individuals with a particular genetic trait.

The definition of natural selection as given above is a little narrower than the concept used in popular literature about evolution; however, the definition given here undoubtedly embraces the core neo-Darwinian understanding of the process.

THE SEMIOTIC MECHANISM OF EVOLUTION

Supplied with these definitions, I can now describe the mechanism that is alternative to or, as it may be, more general than, the mechanism of adaptive evolution by means of natural selection. It can be more general in the sense that we assume the adaptive relation is always of a certain qualitative and communicative kind, and, as a special case, natural selection may also be involved in its distribution; however, in general the latter is not necessary.

An adaptive evolutionary mechanism without natural selection can be described by the following sequence of events:

1. A population happens to face new conditions (due to a change of their environment, either locally or due to migration, of food, or symbionts, etc.) and the organisms of this population accommodate to the new conditions via a physiological adjustment (as related to the trophic or behavioural change); this may change their gene expression profile, as it often does (without a change of genome, yet).
2. The new gene expression profile can last for many generations due to reasons which are not genetic. This may be a result of the permanence of new habitat conditions, of the stability of the environmental conditions, or of the continuation of a newly established ecological bond with a symbiont or parasite species, or, as it may often be, with a new main food resource, particularly in almost monophagous populations. The permanence of the new gene expression profile can be supported by the epigenetic inheritance mechanisms.
3. If the new situation with the new gene expression profile lasts long enough, then mutations that are selectively neutral in the new environment would gradually accumulate and eventually individuals in the derived population might be incapable of developing the ancestral phenotype, even in the old environment. More precisely, the accumulation of mutations (particularly in the part of the genome that became non-functional as a result of new conditions) together with mating (which keeps intrapopulation phenotypic variability bounded by certain limits due to some similarity that is required to enable mating, i.e. due to the limitations for the differences between the organisms within a mate-recognition system) can make the change irreversible, i.e. the return to the previous conditions would not reverse the initial gene expression pattern. Thus, the stochastic genetic changes can make the change evolutionary.

THE GENETIC FIXATION OF ADAPTATION WITHOUT DIFFERENTIAL REPRODUCTION

As this is the part of the mechanism that is quite decisive for the argument, it may require a more detailed description. Stochastic (non-selectional) genetic processes that make the adaptations genetically inheritable (irreversible) include at least two aspects (mechanisms) or effects.

1. Accumulation of mutations in the newly non-transcribed or functionally non-obligatory parts of the genome; we can call this process as 'forgetting of un-used' (Kull, 2000). If the gene expression profile is changed in a number of organisms due to a new (non-genetic) adaptation (e.g. in a new environment), and remains so during a number of generations (e.g. via support from epigenetic inheritance *sensu lato*), then the *accumulation of mutations in the non-expressed (but formerly expressed) loci makes the development of the previous phenotype impossible*. (Alternatively, if the previous phenotype is achieved, then it is not on the basis of the same genetic mechanism.)

On the other hand, the mutations occurring in the *expressed* loci cause certain non-specific mortality of the young, which is roughly proportional to the percentage of the expressed part in the genome. One could say that this is a form of natural selection because the 'deficient' individuals are as if removed selectively. However, this should not be called natural selection, because this mortality may have no statistical connection to any particular mutation, as mutations are usually unique. In other words, the stochastic mutations in various parts of the functional genome produce certain non-specific mortality, but this does not imply fitness differences between the co-surviving genotypes; thus, the mechanism does not require natural selection in the sense defined above.

In conclusion, genetic changes that make a plastic adaptation irreversible can occur without natural selection.

2. Intrapopulational crossings that keep the population within certain limits of variability (Gorelick & Heng, 2011). This effect requires biparental reproduction with recombination (which mixes the genomes and increases the genetic similarity between the organisms in a population; inbreeding), resulting in communicative resemblance. In other terms, this is an effect stemming from assortative mating.

Organisms that are not recognized by other individuals will not reproduce biparentally. But this may have no connection to any particular allele – because the *difference* between the partners is what counts, not any individual feature. This is a purely relational process.

Thus, the (genetic) communication itself holds a population (species) together, working against genetic diffusion. Differential reproduction of particular genetic traits is not obligatory here. Thus, this effect, too, is not due to natural selection – it is more general. [Very often the mechanism that holds populations together is characterized as purifying or stabilizing selection. In assortative mating, however, a similar effect can be achieved without the involvement of natural selection as described above. This is because the 'extreme individuals' are seen as extreme in relation to other individuals and not by any particular absolute trait, i.e. not by any specific allele.] In this way we have genetic change (without any *specific* mutation), spreading in the population without differential reproduction. Let me describe this in more general terms again.

To have a phenotypic change that is genetically fixed, it is usually assumed that there exists a specific genetic marker or a combination of markers that characterizes the phenotype. However, because a particular phenotype can be genetically fixed via many genetic patterns, it is also possible to have a phenotypic change that is genetically fixed, but without a particular marker common to the population.

A simple example can be a case in which a phenotypic change would become irreversible if a certain gene was permanently switched off. There are obviously billions of different mutations that can make a gene inactive – including the mutations of the gene itself, its regulatory region, or the genes of its regulatory factors, such as transcription factors. Therefore, to make the change irreversible at the population level, there is no need for one particular mutation (genotype) to be spread throughout the whole population as a result of its differential reproduction – instead, it is possible simply to have one of these billions of mutations in each individual. For a germline to collect these mutations will be just a matter of time; differential reproduction is not required for this. The process of collecting these mutations is enhanced by recombination, because if an individual has gathered several mutations having the same effect, a recombination process will distribute these to the offspring of a partner that does not yet have any. Such distribution of the trait in the population, again, does not require any differential reproduction.

Thus, if contrasting the two mechanisms of adaptive evolution (neo-Darwinian and semiotic), we may describe these as individual versus relational. [Natural selection can count also for some relational effects, but its core, as follows from its definition, is in the replication of individual genetic traits.]

a. Based on individuals. *Natural selection* – an individual mutant is copying itself and outcompetes the others (mutation first, phenotypic change follows).
b. Based on relations. *Organismic choice* (organic selection) – a collective bond (relation) becomes stable and inherited (phenotypic contact and epigenetic change first, stochastic genetic change follows).

Here a comment on the evolution of plasticity is appropriate. One may argue that the random genetic changes which make the adaptive plastic change irreversible also reduce plasticity. Indeed, genetic changes are not sources of plasticity, they are rather the constraints of plasticity. On the one hand, if in some cases the plasticity itself were to decrease as a result of turning an adaptation irreversible (like an effect of hardening of habit), it cannot nullify adaptation. Adaptation is always a local relation, it is never universal. Therefore, it is possible, for instance, that an adaptation which enables an organism to consume certain food and to specialize on its use may become detrimental due to a later shortage of that resource – still, it remains an adaptation. On the other hand, the irreversibility of an adaptation as an attainment does not mean that plasticity itself has to be decreased as a result of it. This is because the adaptive plastic changes themselves as attainments (and skills) are the sources of potential new attainments, i.e. of increased diversity and plasticity (see also discussion on this issue in West-Eberhard, 2003: 178–180, *passim*).

In brief, the semiotic mechanism of an evolutionary event consists of two steps: (1) the adaptive plastic non-genetic change, and (2) the neutral genetic change. The event is adaptive due to the first step (and it can be called 'semiotic' because it establishes a meaningful relation). Thus, there is no need for the second step to be adaptive for the whole event to be considered adaptive.

TYPOLOGY OF MECHANISMS OF ADAPTIVE EVOLUTION

This analysis leads us to a clear classification of adaptive mechanisms of evolution, as based on two major characteristics: (1) the type of mutations assumed (either random or non-random), and (2) the order of processes (either epigenetic change first or mutations first). Accordingly, we can distinguish four

Table 1. Four main types of adaptive evolutionary mechanisms

	Random mutations	Non-random mutations
Epigenetic change (learning) first	Baldwinian, or semiotic (organic selection)	Lamarckian (exercising)
Genetic change first	Neo-Darwinian (natural selection)	T. H. Morganian (mutationism)

types of mechanisms, which are all represented by some theories in the history of biology (Table 1).

I. Non-random mutations, genetic change first. This was the view of evolution espoused by T. H. Morgan and some other mutationists. Despite many abandoning this view as a result of the Modern Synthesis of the 1930s, it did not disappear completely. Even more, there exist some recent claims about the existence of this mechanism. For instance, J. A. Shapiro (2011: 143) writes: 'Cells are built to evolve; they have the ability to alter their hereditary characteristics rapidly through well-described natural genetic engineering [. . .]'.
II. Non-random mutations, epigenetic change (learning) first. [The term 'learning' is appropriate here in the general sense, if to define learning as an adaptive plastic change.] This mechanism can be identified as a Lamarckian theory. J. B. Lamarck himself, of course, could not speak directly about mutations due to the level of knowledge at his time, but this is the way the Lamarckian approach – inheritance of acquired characters – has usually been interpreted in the later biological literature (e.g. as explained by Mayr, 1997 [1976]: 314).
III. Random mutations, genetic change first. This is the common neo-Darwinian mechanism, the first event being a random genetic change, followed by a new phenotype and natural selection.
IV. Random mutations, epigenetic change (learning) first. According to the semiotic mechanism described, the first event in an evolutionary transformation is a plastic change (change of phenotype), followed by the stochastic genetic changes. This mechanism can be identified with organic selection (as thought of by Baldwin, 1896; Lloyd Morgan, 1896; Osborn, 1897; see also Kull, 1993; Hoffmeyer & Kull, 2003; Sánchez & Loredo, 2007), with the addition that differential reproduction may even be unnecessary for this mechanism in its pure form.

As the role of non-random mutations is quite limited, the Lamarckian mechanism (II), and evolution via adaptive mutation, often called mutationism (I), are unlikely to have had a dominant role in evolution. Accordingly, the major alternative to the neo-Darwinian mechanism (III) is that of organic selection followed by random mutations (IV).

In addition to these adaptive mechanisms of evolution, there exist mechanisms of neutral or non-adaptive evolution (see Gould, 2002: 1258ff), including self-organization of organic form (see, for example, Depew & Weber, 1997; Corning, 2005), which is also non-adaptive – these can change species, but they do not lead to the formation of new adaptations.

The actual relative role of these different mechanisms in evolution, of course, has to be discovered by means of empirical studies. It seems obvious that in real evolutionary events, in many cases, we can observe the simultaneous operation of two or more different mechanisms of evolution. For a better understanding of these processes, a logically exact distinction between the mechanisms could be very helpful.

CONCLUSIONS

Because what we call 'behaviour' and the corresponding rearrangement of form in the case of living organisms is the way a living being interprets the world – i.e. behaviour is the interpretation process *sensu lato* – and because the study of interpretation processes or semiosis is called 'semiotics', it would be correct to call the mechanism of evolution which is led by the way in which organisms find new solutions as the '*semiotic* apparatus of evolution'.

According to this mechanism, adaptation (which can be seen as a process of acquiring new pieces of knowledge by a living system), strictly speaking, does not require natural selection or its analogue. Instead, the common process that adds new meaningful information to a living system is the process of learning, or abduction, which occurs in the situations of incompatibility, or problem-solving, that a living being has to deal with. In this sense, we may say that life is a more-or-less continuous problem-solving process. Among the common problems, then, we find the biological needs each organism has. In this sense, life can be seen, indeed, as its own designer (Kull, 2000; Markoš *et al*., 2009; cf. Wintrebert, 1962).

If contrasting the neo-Darwinian view on evolution to the one described here (or the biosemiotic

approach, see Hoffmeyer & Kull, 2003), we may formulate it as follows.

(Type III, as above) Evolution is of primary importance. Everything in life is a result of evolution and is based on evolution. (This can be illustrated by Dobzhansky's dictum: Nothing in biology makes sense except in the light of evolution.) The dominant way of explaining how living systems work is through history, i.e. diachronically.

(Type IV) Evolution is of secondary importance. Evolution is not necessary for life to function. Life simply cannot avoid evolution; evolution is rather a side-effect of living processes. (We may use a paraphrase of the dictum above: Nothing in biology makes sense except in the light of sign relations, or meanings and functioning.) The primary way to explain the workings of living systems is via the meanings, i.e. synchronically.

Every living being is polyphenic – it has both potentially and really many shapes and behaviours to be used and discovered for finding its way of life in new situations. The capacity for change in designing itself is dependent on the types of sign processes available to the organism (Kull, 2010). Finally, it can be said: *Evolution is not necessary for living, it just happens.*

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