Flegr, J. (2022). Adaptations, By-products, and Spandrels. In T. Shackelford (Ed.), The Cambridge Handbook of Evolutionary Perspectives on Sexual Psychology (Cambridge Handbooks in Psychology, pp. 87-113). Cambridge: Cambridge University Press.

5. Adaptations, Byproducts, and Spandrels

Jaroslav Flegr

Department of Philosophy and History of Science, Faculty of Science, Charles University, Prague 2, Viničná 7, CZ-128 44, Czech Republic. email: flegr@cesnet.cz

Abstract

Organisms carry a large number of adaptive traits, i.e., traits that enable them to obtain resources and acquire sex partners from their social, biotic, and abiotic environments, and escape the negative factors of these environments. When we recognize an adaptive trait, we typically assume that it is a product of some form of selection, either of natural selection *sensu stricto* (environmental selection, as, for example, legs and eyes), sexual selection (e.g., antlers and peacock tail), or parental selection (e.g., the colorful interior of the beak of altricial birds' nestlings). In many cases, the attribution of a biological function to the trait is simple and straightforward. However, even in such cases, we can be wrong – a particular trait could be an *exaptation* rather than an adaptation or it could be a *by-product* of processes other than selection. Sometimes, we are not able to recognize what function a trait has for its bearer. The trait, including a behavioral pattern, can be a product of the manipulative activity of a biological entity other than the entity we suspect, usually another member of its species or a parasite. Besides, many traits are products of the organism's own genes but help to spread their own copies at the expense of the biological fitness, viability, or fecundity of their carrier. A trait can also be a product of a different selection process. A trait might be a product of group selection or species selection, for example. Certain complex traits evolved only to keep an old biological adaptation in a functional state, not to evolve a new useful adaptation. And, finally, a trait can fulfill a function that was useful for the ancestors of the present organism but is not useful for the organisms that we study now. Similarly, a particular trait can be useful under special and rare conditions that are unknown to or otherwise not taken into consideration by the researcher.

Keywords: Adaptations; preadaptation; exaptations; postadaptations; spandrels; altruism; spite behavior; stability-based sorting.

5.1. Adaptive traits that are not adaptations in a narrow sense

Many complex traits that fulfill a certain useful biological function and therefore seem to be biological adaptations are not biological adaptations in a narrow sense. Stephen J. Gould (Gould, 2002; Gould & Lewontin, 1979) described two categories of such adaptive traits: spandrels and exaptations.

5.1.1. Spandrels

Spandrels are structures, sometimes rather complex structures, that do not come into existence by any sort of selection or sorting, but because of the inherent laws of physics, chemistry, or geometry. Gould borrowed the term spandrel from architecture (Gould & Lewontin, 1979), where it describes an architectural element that develops not through the intention of the architect, but as a consequence of objective, in this case topological, laws. Gould and Lewontin discussed a special case of spandrels, the pendentives. These are spherical, triangular areas permitting the placing of a circular dome over a square room. The best-known case (at least for evolutionary biologists who read the seminal article of Gould and Lewontin) consists of the pendentives in the Basilica of St. Mark in Venice. Today, these pendentives bear pictures of the four evangelists and thus seem to be an essential and intentionally created element of the artistic decoration of the cathedral. However, they were not created to bear these paintings, but because this was the most rational structural solution for joining a four-walled base with its cupola ceiling. In short, the pendentives in the Basilica of St. Mark originated as a structural necessity and "became the subject of selection", i.e. were covered with the mosaics of four evangelists, long after the creation of the basilica.

Columella in the axis of a snail shell is an example of a biological spandrel (Fig. 5.1). It could be considered as a biological adaptation that was built up by natural selection to increase the rigidity of the shell. However, the central rod must arise automatically due to laws of topology in any shell that develops by coiling a growing chamber of an aging animal into a spiral during the life of a snail. Therefore, the columella is not an adaptation, but a spandrel. Of course, the morphology of columella can be later modified by natural selection to fulfill a certain biological function. In such a case, the columella becomes an exaptation (see below). Similarly, it is useful to

have body mass for keeping attached on the surface of Earth, where most resources are available. However, body mass is not an adaptation built up by natural selection to anchor us to the surface of Earth; it is a spandrel that arises due to laws of physics.

Fig. 5.1 Columella represents a spandrel, not an adaptation.

5.1.2. Exaptations

Exaptations are traits (structures or behavioral patterns) that originated as spandrels or by means of selection but now fulfill different functions than those for which they were originally selected. For example, the feathers of birds probably evolved as an adaption for thermoregulation in certain groups of reptiles (Thanukos, 2009). The existence of these structures later enabled the evolution of wings, the organs of gliding, and eventually active flight. The feather was therefore a "preadaptation" for flying (the structure originally evolved to insulate animals, but was later modified by selection for flying). When it started to fulfill its new function, it became an exaptation (not an adaptation) for flying.

The difference between an adaptation and an exaptation is not just a question of terminology; it has also important practical implications. The current state of various exaptations carries not only the traces of the selection for their present function but also the vestiges of their past function or functions. Previous functions act as historical constraints playing an important role in the past evolution and current structure and function of the exaptation. Often, the structure of an organ or the nature of some behavioral pattern is suboptimal in the context of its current biological function. The reason for this could be that it is not a biological adaptation designed for its current function but instead an exaptation for this function. It is no exception that the exaptation must fulfill both new and old functions. Because of this, its current form cannot reach the optimum state because there is a trade-off between the optimization for the purpose of old and new functions. The feather best suited for thermoregulation differs from the feather best suited for flying. Similarly, the structure of the seal paw cannot be optimal both for swimming and for walking. In some cases (e.g., feathers), the two functions may separate with further evolution. In other cases (e.g., seal paws), such separation is complicated or even effectively unattainable, which leads to a prolonged state of suboptimal exaptation.

In the real world, we see a ladder of adaptations, preadaptations, and exaptations. All traits originally come into existence as products of accidents – random mutations. The resulting evolutionary novelty usually decreases the viability of its carrier. Sometimes, however, the mutation has a positive effect on the biological fitness of the mutant individual and improves some biological function (A) of the organism (or the probability of transmission of a particular allele to the next generation; see Dawkins' model of inter-allelic competition (Dawkins, 1982)). In the first case, the new trait can be considered as a preadaptation for function A. The efficiency of the function is improved by the accumulation of further mutations and the corresponding structure (or, e.g., a behavioral pattern) will become an adaptation for A. Trait A can be the preadaptation for a trait B, or sometimes traits B, C, D, etc. These new traits are the exaptations for other biological functions and some of them can be at the same time preadaptations for other distinct and different functions. Taken to the extreme, all adaptations are exaptations because evolution does not start from nil. It modifies already existing structures or functions. Still, the term and concept of exaptation are useful when we analyze the evolution of particular traits and search for developmental constraints that played role in their formation.

An important role in the process of transforming a preadaptation, spandrel, or nonadaptive trait originating by a random mutation into an adaptation is often taken by the so-called Baldwin effect (Baldwin, 1896). Usually, the Baldwin effect is considered to come into action when the evolution of certain behavioral patterns is studied. However, the role of the Baldwin effect is more universal and could also concern the evolution of morphological or physiological traits. The Baldwin effect stresses the role of learning in evolution and implies that a change in behavior may precede a change in gene frequency. Shortly and simply, it states that an animal "cannot evolve fins without starting to swim". The animal must at first start to swim (using, e.g., limbs) to allow selection for, and consequently, accumulation of, mutations that modify the structure of limbs in a way that increases the efficiency of swimming and, in the end, turns the feet of a terrestrial animal into the fin of an aquatic one. Some mutations also modify the neural system of the animal in a such way that the behavioral patterns needed for using the new morphological traits for swimming become instinctive despite the fact they were originally learned and transmitted from generation to generation culturally. J. M. Baldwin was a psychologist and therefore the genetic assimilation of originally learned behavior was the main subject of his interests. However, the principle, in this context often called *organic selection*, is nearly universal and may occur in some form in the evolution of any trait.

5.1.3. Byproducts

Many traits have a nature of preadaptation without clear adaptive value. Some of them originated as spandrels, some as adaptations for certain (known or unknown) functions. They could fulfill some function at the present; however, often their adaptedness is at least a subject of doubt. Often, such traits are byproducts of the evolution of other biological functions. Scientists, and especially editors of scientific journals, like adaptations. Therefore, there is temptation for the authors of a scientific paper to attribute some function to the trait under study. Sometimes, such attribution is correct; however, sometimes it is wrong. For example, several hundred papers exist showing the behavioral effects of latent *Toxoplasma* infection on the behavior of intermediate hosts, including on the behavior and personality of humans. These effects are mostly considered as products of the manipulative activity of the parasite aimed to increase the efficiency of its transmission from infected intermediate hosts (any warm-blooded animal) to a definitive host (a cat) by predation. Some effects are specific, e.g., the change from fear associated with a cat's smell to attraction to this smell in infected rodents (Berdoy, Webster, & Macdonald, 1995, 2000), chimpanzees (Poirotte et al., 2016), and probably also humans (Flegr, Lenochová, Hodný, & Vondrová, 2011; Flegr et al., 2018). Such specific effects have a high probability of being the products of parasite manipulation.

Some effects are, however, less specific and could therefore be byproducts of other activities of the parasite, or could be the product or byproduct of adaptive or maladaptive reactions to the infection by a host. For example, infected humans display several specific shifts in personality traits, e.g., in extroversion and suspiciousness. Typically, the opposite personality shifts are observed in men and women (Flegr, Zitkova, Kodym, & Frynta, 1996). To explain this pattern, Lindová et al. suggested the stress-coping hypothesis (Lindová et al., 2010; Lindová et al., 2006). It posits that the observed personality changes are not the result of the manipulative activity of *Toxoplasma*, but byproducts of the behavioral reaction of infected hosts on the chronic stress that accompanies the latent infection. Most parasitologists and medical doctors consider latent toxoplasmosis, which affects about one-third of the world's population, harmless in immunocompetent humans. However, recent research inspired by the stress-coping hypothesis showed that this prevailing notion is probably wrong. One study showed that differences in the prevalence of toxoplasmosis in 88 countries positively correlate with the incidence of many diseases and disease categories after potential confounding variables (e.g., Gross Domestic Product per capita (GDP), latitude, and humidity) are controlled (Flegr, Prandota, Sovickova, & Israili, 2014). In fact, the separate analysis performed for Europe showed that the differences in prevalence of toxoplasmosis

explain 23% of the total variability in disease burden in 28 European countries. In a cross-sectional study performed on 1488 nonclinical volunteers showed that 333 *Toxoplasma* infected subjects scored worse in 28 of 29 health-related variables and reported a higher incidence of 77 of 134 disorders under study (Flegr & Escudero, 2016).

It is highly probable that some latent toxoplasmosis-associated personality changes are the byproducts of the impaired health of infected subjects. For example, *Toxoplasma* infected subjects, both men and women, score lower on the personality trait novelty seeking than uninfected subjects (Flegr et al., 2003; Novotná et al., 2005). This personality trait is known to be associated with an increased level of the neurotransmitter dopamine (Cloninger, 1998). The overproduction of dopamine can explain about a 2.7 times higher risk of schizophrenia in *Toxoplasma* infected subjects (Torrey, Bartko, Lun, & Yolken, 2007; Torrey, Bartko, & Yolken, 2012). It was originally suggested that this neurotransmitter is produced by activated leukocytes in the inflammation loci in an infected brain (Flegr et al., 2003). Later, however, genes for two essential enzymes for the dopamine synthesis were identified in the genome of *Toxoplasma gondii* (Gaskell, Smith, Pinney, Westhead, & McConkey, 2009) and the expression of these genes, as well as large concentration of dopamine, were observed in cysts of *Toxoplasma* in the infected brain tissue (Prandovszky et al., 2011). It is, therefore, possible that increased production of dopamine and the resulting behavioral changes were originally byproducts of pathological processes in an infected brain. Later, however, they were reinforced by acquiring corresponding genes, possibly by horizontal transfer, and production of dopamine in the infected cells.

Some byproducts can be important and adaptive. For example, the evolution of our brain could be originally fueled by selection for other functions (Gould, 1997). A good candidate is processing visual stimuli in the complex tridimensional environment of canopies where our primate ancestors lived. Later, the same "hardware" was adopted for other functions that started the processes of sapientation in our more recent ancestors. Corresponding brain structures, therefore, could be considered both as a byproduct and a preadaptation.

5.2. "Adaptations" without adaptive values for their carriers

5.2.1. Selfish alleles

Since Darwin, most biologists studying an unknown complex trait automatically ask the same questions: "What is the biological purpose of this trait? How does it increase the biological fitness of its carrier or its chances to win in the competition for resources, including sex partners?" However, the situation is not so simple. Often, this old question is principally wrong and misleading.

As was shown by William D. Hamilton in his seminal papers in the 1960s (Hamilton, 1964a, 1964b) and described in detail by Richard Dawkins in his books *The Selfish Gene* and *The Extended Phenotype* (Dawkins, 1976, 1983), the question should, in fact, be: "How does this trait increase the chance of an allele of the gene responsible for this form of a trait to be transferred to further generations in more copies than other alleles of this gene?" In many cases, the presence of a particular trait, e.g., an extreme form of altruism, decreases the direct biological fitness of its carrier. However, the same gene increases the chance of an individual's survival and consequently also reproduction of relatives who, due to their common descent, probably carry copies of the same allele for altruism. By boosting their chances to reproduce, the allele increases the total number of its copies transmitted to future generations even if the altruistic individual dies before reproducing or leaves a smaller number of offspring.

In the case of ultra-selfish genes (alleles), the presence of a certain allele (and corresponding trait) impairs not only the direct fitness, but also the inclusive fitness of its carriers. For example, the t-allele of mice makes its male carriers subfertile as they have only half the number of the viable sperms. However, all their viable sperms carry the t-allele, not the wild type allele of the gene. The reason is that the sperms with the wild type allele are incapacitated during spermatogenesis. The t-allele, which also codes for a shorter tail, spreads quickly in a population without providing any advantage to its carriers (Ardlie, 1998;

Vanboven, Weissing, Heg, & Huisman, 1996). Such blue-beard alleles (do not confuse with the green-beard effect, which is also described in Dawkins' books) can be responsible for many traits without apparent biological "purpose."

5.2.2. Adaptation (and for the benefit) of other members of a species

Sometimes, the phenetic trait can bring an advantage to another organism, either of the same or different species. In eusocial species, the useful and therefore adaptive strategy of the members of the completely or nearly sterile caste is a resignation of their own reproduction in favor of helping their relatives reproduce. It can be shown that in this way they achieve higher inclusive fitness than when they try to reproduce themselves (Skutch, 1935). In most other cases, however, the resignation of an organism's own reproduction is a product of manipulation by parents or nestmates (González-Forero, 2015). When the manipulation ceases to exist, for example, when the queen dies or is removed from the nest, some "sterile" individuals turn into a new fertile queen.

We can theorize, for example, that a gene in the developing embryo of an older son can, by manipulation with the mother's physiology, affect the ontogeny of the brain of future younger brothers. This could increase the probability that the younger brothers become homosexuals, handicapping them in the future competition for sexual partners or, later, for parental resources, see BOX 1.

Box 1. The sibling manipulation hypothesis of homosexuality

Male homosexual behavior and especially its psychological background – homosexual sexual preferences – are an old evolutionary enigma. It remains common in human (as well as animal) populations despite the fact that homosexuals have significantly lower fecundity than heterosexuals (Ciani, Battaglia, & Zanzotto, 2015). Several principally different explanations have been proposed. A group of related hypotheses posits that homosexuality is a byproduct of bisexuality (bisexuals are hypothesized to have higher fecundity than heterosexuals) (Dewar, 2003). Another group of hypotheses suggest that homosexuality is a result of pleiotropy – basically, an undesirable side-effect (from the point of view of fitness) of some otherwise useful gene (Camperio-Ciani, Corna, & Capiluppi, 2004; Miller, 2000). However, the hypotheses of both groups are only weakly supported by empirical data.

Another hypothesis suggests that homosexuality serves some function unrelated to reproduction, most probably maintenance of long-term alliances of men (Kirkpatrick, 2000). Another category of hypotheses suggests that male homosexuality is a product of kin selection – homosexual males could provide resources to their heterosexual siblings as helpers (E. O. Wilson, 1975). Again, neither of these concepts have been unambiguously supported with empirical data.

Rather, a related hypothesis, the parental manipulation hypothesis, suggests that male homosexuality is a result of the manipulative activity of parents (probably the mother) aimed to optimize the (unequal) distribution of resources among sons (Ruse, 1988; Trivers, 1974). This hypothesis was supported by the discovery of the fraternal birth order effect – the positive effect of older sons (and their number) on the probability of homosexual orientation in younger sons (Slater, 1962). It was shown that each older brother increases the probability of homosexuality of younger brothers by about 33% (regardless whether they lived together or were separated after birth) and that only biological brothers, not stepbrothers (or older or younger sisters) have this effect (Blanchard & Bogaert, 1996), but see (Miller, 2000).

In 2017, a possible mechanism of this manipulation was identified – the maternal immunological response to the Y-chromosome-linked protein neuroligin. Neuroligin is a cell adhesion molecule thought to play an essential role in specific cell-cell interactions in brain development (Bogaert et al. 2017). The nature of this immunological response suggests that the initiators of the manipulation might be older brothers, rather than the mothers. In contrast to the mother, older brothers can directly increase

their fitness by diverting a part of important resources, e.g., future mating partners or material resources of a family, from their closest competitors, the brothers, to themselves. Of course, by doing this, they impair an important component of their inclusive fitness. According to Hamilton's rule, however, the resources invested by the older brother to his own offspring has a two times higher value than the same amount of resources invested in younger brothers' offspring (Hamilton, 1964a, 1964b). Therefore, the sibling manipulation hypothesis offers an alternative and more plausible explanation of the fraternal birth order effect on male homosexuality than the parental manipulation hypothesis does. To test the validity of the hypotheses, it would be necessary to compare the effect of homosexual orientation of a man on the fitness of his older brothers and his mother. Of course, a modern human, with its drastically changed social environment is not an optimal model organism for testing this (and many other) hypotheses..

5.2.3. Adaptations for parasites

Certain behavioral patterns, physiological traits, or morphological traits might be also products of parasite manipulation. For example, upon infection, parasitic flukes *Leucochloridium paradoxum* can turn a tentacle of amber snail *Succinea putris* into a large conspicuous pulsating organ. This "organ" is a biological adaptation of the fluke, not the snail. The evolution of the fluke optimized the morphology and appearance of the transformed tentacle to attract insectivorous birds, the definitive hosts of the fluke. The bird harvests and eats the transformed tentacle with hundreds of microscopic larvae of the fluke that transform in its body into adult, sexually reproducing flukes (Lewis, 1974).

Many examples of complex morphological structures whose morphogeneses are programmed by genes of parasites are known in plants. The resulting structures on the leaves and other organs of a plant, galls, serve as microhabitats for phytoparasites from many phylogenetically unrelated taxa of insects and mites, but also fungi, bacteria, and viruses. The galls are formed from the plant cells; however, they are often genetically or epigenetically modified to produce special chemicals that serve as nutrients for the parasitic organisms that programmed their formation.

Various zooparasites manipulate the most flexible part of the host phenotype, its behavior, to increase their chance of transmission from infected to non-infected hosts. For example, the protozoan parasite *Toxoplasma gondii* can reprogram its intermediate host, usually a rodent, to lose its innate fear of the smell of a cat, or rather transform this fear to an attraction to this smell (Berdoy et al., 2000). It seems now that epigenetic modification (probably demethylation) plays an important role in this reprogramming of regulatory elements of specific genes in neurons of the medial amygdala in the host's brain (Dass & Vyas, 2014; Flegr & Markos, 2014). Causing this, *Toxoplasma* increases its chance of transmission from the intermediate to the definitive host by predation.

Toxoplasma seems to be able to manipulate also the behavior of apes, including humans (Flegr et al., 2011; Flegr et al., 2018). Both chimpanzees (Poirotte et al., 2016) and humans seem to prefer the odor of diluted cat urine (or leopard urine in chimpanzees) after infection with *Toxoplasma*. This surprising finding might be of major significance. *Toxoplasma* causes a lifelong infection in about one-third of people on Earth. It has been suggested that its manipulative activity results in a change in personality and behavior, including specific sexual behavior (Flegr, 2017; Flegr & Kuba, 2016). Now, of course, the induced behavioral changes that include the prolongation of reaction times are not adaptive, as the probability of being eaten by a feline predator is very small in modern humans. However, the situation was probably rather different in our not-so-distant evolutionary history.

BOX 2. Is *Toxoplasma* responsible for masochism?

Latent toxoplasmosis affects the sexual behavior of humans. A cross-sectional study of 5,087 *Toxoplasma*-free and 741 *Toxoplasma*-infected subjects showed that *Toxoplasma*-infected men are more often sexually aroused by fear-, violence-, and danger-related stimuli than non-infected men (Flegr & Kuba, 2016). It was suggested that the manipulative activity of *Toxoplasma*, specifically the change of mice's fear

of the smell of a cat to attraction to this smell, is based on the observed modification (demethylation) of regulatory elements of specific genes associated with the hypothalamus (Dass & Vyas, 2014). This masterpiece of epigenetic engineering could cause the strong stimuli that normally stimulate fear-related circuits to stimulate adjacent sexual arousal-related circuits (Flegr & Markos, 2014).

Of course, toxoplasmosis is not the cause of sexual masochism. In fact, toxoplasmosis is responsible for only a small part of the variability in this personality trait (about 5%) and many *Toxoplasma*-free subjects, especially women, express this trait to a very high degree. Rather, *Toxoplasma* (in a similar way to producers of horror films) exploits the fact that fear- and sex-related circuits are localized in adjacent regions of the hypothalamus and strong stimuli could spillover from the former to the latter. *Toxoplasma* evolved a way to intensify this spillover.

More detailed analyses showed that *Toxoplasma*-infected subjects express lower intensity and a narrower spectrum of sexual activities compared to *Toxoplasma*-free subjects (Flegr, 2017). Less usual types of sexual behavior, including those related to sexual dominance (sadism) and sexual submission (masochism) were especially affected. However, the activities related to sexual submission were affected much less than those related to sexual dominance, leading to the observed relation between latent toxoplasmosis and masochism.

Toxoplasma is a eukaryotic organism with many thousands of genes. It is no wonder that it could have evolved sophisticated biological adaptations to increase the efficiency of its transmission in its host populations. It is highly probable, however, that many viruses, which typically carry only few genes (or a few tens of genes) are also able to manipulate the behavior of their hosts. For example, *Rabies lyssavirus* can modify the behavior of an infected animal in a specific way that is useful for spreading the virus by biting other animals of the same or different species (Hueffer et al., 2017). A less conspicuous (and, fortunately, also less fatal) case is probably the manipulation activity of many respiratory viruses that induce coughing and sneezing, which effectively enhances the spreading of the infection by respiratory droplets or aerosol and possibly by even more sophisticated behavioral manipulation (Bouayed & Bohn, 2020).

5.3. Adaptations that evolved not by individual selection, but by other types of selection

Another problem with adaptive traits is that they could be adaptive on various levels. We have already seen trait adaptation for individuals (those originated by individual selection) and for close family (those originated by kin selection. However, certain traits might be adaptive for a group of only loosely related individuals (group or interdemic selection), whole species (interspecies selection/competition), or even for evolutionary lines/clades (species selection).

Individual selection is probably the most famous evolutionary mechanism that was discovered by Charles Darwin (Darwin, 1860). It provides a simple and understandable explanation of the origin of the purposeful traits of living beings. It can be easily explained and understood due to its analogy with artificial selection (i.e., breeding). Therefore, when a biologist studies a certain structure or behavior of an animal, including a human, he or she automatically begins by asking how it maximizes biological fitness in the competition among the members of the same population.

It is a rational behavior because it is highly probable that most of the adaptive traits originated by this mechanism, or that it at least plays a role in sustaining these traits in current species. The traits that bring no advantages in intrapopulation competition, or even bring some disadvantage, are usually quickly eliminated by individual selection, drift, or draft even if they are useful for a population or a species. However, that is not always the case.

5.3.1. Kin selection

Many traits including parental behavior and many (but not all) forms of altruistic behavior aimed at genetic relatives are the well-known products of kin selection, i.e., the competition for maximal inclusive fitness (Hamilton, 1964a, 1964b). Kin

selection is often used to explain eusociality – the existence of sterile castes in many insect species and even some species of mammals, or the existence of helpers – the individuals who temporarily help their parents to care for their siblings instead of beginning their own reproduction in some species of birds (E. O. Wilson, 1975). Kin selection is sometimes also used to explain the existence of menopause in humans and some other species (Shanley, Sear, Mace, & Kirkwood, 2007; Sherman, 1998).

However, the product of this form of selection includes also spite behavior aimed at nonrelatives. Spite behavior decreases the fitness of the victims of such behavior while it usually has negative impacts also on the individual that expresses it. The individual can increase its relative direct fitness either by increasing its own direct fitness or by decreasing the direct fitness of other members of the population. In the terms of (more important) inclusive fitness, an individual can increase its relative fitness by decreasing the fitness of individuals that are not its genetic relatives. For many species, it is not easy to estimate the relatedness of other members of the population. However, for some species, this is possible to achieve, for example, by using olfactory clues. In other species, an individual can achieve the same effect by moving from its native population to a geographically distant population. It has been suggested that the tendency of individuals infected by certain parasites to travel to distant populations is not the result of the manipulative activity of the parasite (aimed to infect new populations), but the adaptive spite behavior of an infected host aimed to increase its relative inclusive fitness by infecting unrelated individuals (Rozsa, 1999, 2000).

5.3.2. Group selection

Group selection, sometimes also called interdemic selection, is encountered wherever a species forms a large number of more or less independent social groups, i.e., herds, flocks, or bands, and when the survival or reproduction of an individual is closely connected with the survival and success of its social group. If group selection is in operation in a given species, then its action can lead to a preference for those properties of organisms that are advantageous for the group as a whole, but need not provide any advantage for the bearer or can even be harmful to the bearer. The pattern of altruistic behavior is a typical example of traits that are useful for a group as a whole but harmful to their carriers.

For example, if a predator appears in the vicinity of a flock of jackdaws, the first jackdaw that notices its presence gives a warning cry, and the whole flock tries to escape or defend itself. From the standpoint of the individual, the issuing of the warning cry and participation in the protection of the flock is disadvantageous behavior. The individual would have a much better chance of survival if it were to selfishly use the information about the presence of the predator for itself alone and, according to the circumstances, either crouch down or inconspicuously move to the other side of the flock and leave some other individual, perhaps its potential competitor, to be eaten. But, instead of this, it warns the rest, gives up its advantage, and exposes itself to the same risk that the predator will attack it as any other member of the flock.

However, from the standpoint of the *group*, this *altruistic behavior* is useful because it reduces the probability that the predator will be successful in attacking the flock. Among a great many moving targets, attacking the center of a scattering flock is difficult and frequently unsuccessful. A flock that contains *altruistic individuals* thus has a better chance of deflecting the predator than a flock of the same size that does not contain *altruists.* Therefore, at the end of a certain period, for example, a season, the group will be more numerous, and it is thus more probable that it will split off a greater number of daughter flocks.

Until the 1960s, there was a strong position for a major role of group selection in evolution. This completely changed after the contributions of John Maynard Smith and George C. Williams (Maynard Smith, 1964; Williams, 1966) who demonstrated that, in most cases, *individual selection* is much stronger than *group selection.* Consequently, until the 1980s, biologists were mostly of the opinion that *group selection* is rarely an important factor in nature. However, new results of the analysis of theoretical models indicate that, under conditions where individual subpopulations regularly emerge and disappear in the framework of the population as a whole, group selection can be an important

factor and, within a certain range of population parameters, can even predominate over individual selection (Alexander & Borgia, 1978; Shanahan, 1998). It is highly probable that many phenetic traits, and especially many conspicuous behavioral patterns, originated through group, not the individual, selection (D. S. Wilson & Sober, 1994).

Box 3 – Altruistic and malicious punishment revealed by the Dictatorship game

A typical example of human altruistic behavior is the punishment of non-cooperative individuals in variants of the dictatorship game. In one variant of this experimental game (Kubena, Houdek, Lindova, Priplatova, & Flegr, 2014), a computer (or a researcher) randomly assembles 12 anonymous players into pairs consisting of a "dictator" and a "slave". The dictator receives a certain amount of money, for example 20 CZK (about $1 US), and he or she can send any part of it to the slave. After that, the computer reassembles the players into new pairs and a new run of the game starts. Nobody plays more than once with the same co-player and nobody knows who is or was his co-player. In the role of a dictator, Czech students sent on average 6.11 CZK to their (unknown) slaves. However, the sum quickly decreased from 11 CZK to 3 CZK during six runs of the game.

In the second variant of the game, the sums sent to all slaves were made public after each run and the players got the option to buy a right to punish any player. If a player paid, for example, 9 CZK, 50% of the punished player's gain in this particular run was subtracted. Note that the punishing was an act of pure altruism, as the prize was paid by an individual and the reward (correction of a selfish player) was obtained by all players. Regardless, players altruistically punished rather frequently and the possibility to punish had a strong effect on the generosity of the players in this variant of the game. The mean contribution in the first run of the game was similar to the standard variant of the dictatorship game (11.3 CZK), but it did not decrease throughout the game. Instead, it increased to 12.4 CZK in the last run. Therefore, the tendency to altruistically punish selfish individuals increased the cooperativeness among players and was therefore advantageous for the group as a whole. Surprisingly, this experimental setup revealed also a sort of spite behavior – the *Justine effect* (Kubena et al., 2014). The probability of being punished decreased with an increasing amount of money sent by a dictator to his slave. However, this negative correlation turned positive for very generous dictators. The inflection point was near the border of the upper quartile. In other words, the most generous subjects had a rather high probability of being punished. These results suggest that overly self-sacrificing individuals are favorite subjects of antisocial punishment. They are targets for punishers significantly more often than individuals who contribute slightly above the median. In the last 10 years, many authors have confirmed the existence of antisocial punishers in different societies and have suggested various evolutionary explanations for this phenomenon (Herrmann, Thoni, & Gachter, 2008; Pleasant & Barclay, 2018; Sylwester, Herrmann, & Bryson, 2013). One is that the antisocial punishing, resulting in the *Justine effect*, represents a sort of spite behavior – driven by the effort decrease of relative fitness of competitors.

5.3.3. Interspecific selection

Another type of selection that can, theoretically, be responsible for the origin of certain traits is interspecific selection, which results from the competition between different species. It can result in the elimination of a species that has lower efficiency of acquiring a common resource or which has a lower ability to sustain the activity of a common predator or parasite. The species with favorable combinations of phenetic traits can survive in the interspecies competition. However, conditions in different parts of the geographic areal of species usually vary. For example, in certain parts of the areal, the common parasite is absent. Similarly, in certain parts of the areal of the two competing species, the common limiting resource is so abundant that it stops limiting the reproduction of one or both species (and other resources, different for each species, could start to limit the growth of these two species). Therefore, different species could win the interspecific

competition in different parts of the areal. Moreover, under some conditions, both competing species, which have an identical niche, could coexist in the same place for a very long time (Flegr, 1994).

The interspecific selection resulting in the interspecific competition is, in fact, a sort of stability-based sorting (see below) rather than a sort of selection – the less adapted species will go extinct earlier while better-adapted species will survive longer. They, however, do not necessarily transmit their "adaptations" to the daughter species (see below). The out- competing of the less adapted species is, in the evolutionary time scale, an extremely rapid process. Therefore, the species could hardly evolve complex adaptations by the process of interspecific selection. Because of this, not adaptations but preadaptations – the traits that have evolved in a context of other selection pressures, or spandrels – the traits that evolved without an involvement of any form of selection (see above) play the key role in the process of interspecific selection.

5.3.4. Species selection

Interspecific selection is sometimes confused with another process of a similar name, *species selection*. The criterion of success in species selection is neither direct fitness nor inclusive fitness, but resistance to extinction and the capacity for speciation (Lieberman & Vrba, 2005; E. S. Vrba, 1984). Therefore, the trait which increases the probability of speciation (e.g., loss of wings, as in some clades of insect) could result in the evolutionary success of the clade, regardless of the fact that the absence of wings might be maladaptive in most of the other forms of selection. Similarly, the clades with species that better resist extinction, e.g., benthic species forming many small, isolated populations, better prosper in the species selection than the clades with mostly planktonic species forming one large intermixed population (Emiliani, 1993). In the macroevolutionary time scale, the clades, i.e., the branches of the phylogenetic tree, containing extinction-proof species or species that are frequently subjects of splitting speciation thrive while the clades without such species succumb.

Both extinction proneness and the tendency to speciate can be at least to some degree inherited by daughter species. Therefore, species selection is a type of selection, not a type of sorting. Species selection prevails over other forms of selection in the long-term perspective. However, it is ineffective in constructing complex adaptive traits. The origin of such traits requires many successive steps analogical to the accumulation of many independent mutations, while there is not enough time and probably also not enough individuals (here species or higher taxa) that are subject to species selection. For example, most phyla of multicellular animals originated 500-600 million years ago (Gould, 1989). The duration of a typical metazoan species is several million years. Therefore, the number of "generations" available for the evolution of any trait through species selection is very small in comparison with the number of generations available for the evolution of a trait by individual selection. Still, some important traits, such as active flying (or active swimming) could originate or at least be promoted, by species selection.

It is not clear whether any human traits evolved by species selection. However, the tendency of *Homo sapiens* for splitting its population into many semi-isolated populations (nations), at least partly driven by rapid evolution/diversification of languages, could protect against extinctions by pandemics and could increase the chances of our species in species selection.

5.3.5. Structures evolved by passive selection

Passive natural selection (originally referred to by the already occupied term neutral evolution) does not lead to an increase in the functionality of a structure but does eliminate the consequences of evolutionary changes (mutations) that would otherwise lead to a decrease in its functionality (Stoltzfus, 1999). The mechanism of RNA editing in *Trypanosoma* could be an example of a system that evolved into the present-day complex form through passive natural selection. Trypanasomas belong in the class of kinetoplastida. Many species of this taxon share extraordinarily complicated means of managing genetic information (Benne, 1992). There are a great many genes of their modified mitochondria that are stored in the DNA in a coded form and must be edited at the RNA level before being translated to the relevant

proteins. Based on the instructions contained in the guide RNA (gRNA), the enzymatic apparatus inserts the individual nucleotides into many sites on the immature messenger RNA (pre-mRNA) and removes others from the pre-mRNA. In this process, many gRNAs specific for a particular gene and coded at various sites in the trypanosome genome gradually participate in a certain order in editing one mRNA. It is probable that the entire complicated and energetically demanding system does not have any functional importance for trypanosomes and that its formation was the result of passive selection.

If a mutation occurs in the DNA, for example, deletion of one of the nucleotides, which reduces the biological fitness of its carrier, selection pressure begins to act on the mutant and its progeny. As a consequence, over time either revertants to the original form or mutants that are capable of compensating the negative manifestations of the mutation will predominate in the progeny of the mutant. One of the possibilities consists of the creation of an editing apparatus that is capable of repairing the change at the level of the mRNA. As soon as one such apparatus is formed, it can be modified and used to compensate for an increasingly broad range of mutations and its universal complexity will gradually increase. After a certain time, the editing process will become essential for its bearer. The means of additional repairing of mRNA is especially effective and thus highly probable for genes that are located in the cell in many identical copies, i.e., the genes of organelle DNA.

As far as I know, no behavioral pattern in animals or humans has been described as the product of passive selection. However, this mechanism was described just recently, and it is a question whether such patterns (or structures) were ever seriously investigated. It might be speculated that modern medical care is (or, in the relatively near future will be) such a complex behavioral pattern. Due to medical care, including assisted reproduction techniques, semi-lethal alleles or alleles for sterility could accumulate in the gene pool of our species. The presence of such alleles might eventually make most humans dependent on the existence of such techniques and will then call for the development of more sophisticated techniques (which again speed up the accumulation of other detrimental alleles). Without the ability (and will) to correct the alleles in the germinal line of cell, this positive feedback could lead to a dead-end in the future.

5.4. Traits formed by stability-based-sorting not selection

A further source of problems is the existence of an evolutionary process related to selection: stability-based sorting (SBS) (Toman & Flegr, 2017; S. Vrba & Gould, 1986). The role of SBS is often neglected. However, this process might be even more important than any form of selection. The main difference between selection and SBS is the central role of heredity in the process of selection and the absence of it in systems subjected solely to SBS. To become the subject of selection, the entities must vary in the trait, the carriers of different forms of the trait must differ in the probability of surviving and/or reproducing, and offspring must inherit the form of the trait carried by its parent(s). To become the subject of SBS, entities in a system need not exhibit any heritability or even a parent-offspring relationship. Only a small fraction of entities (organisms, genes, memes, etc.) are subject to the process of selection. On the other hand, all biotic and abiotic entities are continuously subject to the process of SBS. In this process, the entities which are not stable disappear – are destroyed or change into something else, while those which are stable remain. Therefore, in any system undergoing evolution, at all times, more and more stable/durable entities accumulate. In abiotic systems, we can observe, for example, the accumulation of stable elementary particles, stable chemical elements, or stable types of stars. In biotic systems, we can see a continuous increase of representation of traits that assure the long-term survival of the entities, e.g., developmental pathways, populations or species. Selection is a more effective and more rapid process than SBS as the selected entities accumulate (inherit) useful innovations in genealogical lines. In systems undergoing only SBS, evolution must wait for a rare event of co-occurrence of useful (stability-ensuring) innovations in the same entity.

SBS has, however, two main advantages when compared to any form of selection. It has

always the final decisive word and it can (seemingly) see ahead – it can evolve the traits that will be adaptive from the viewpoint of future sustainability. Selection can result, for example, in the origin of such a sophisticated organ such as the human brain. This organ has enabled one species of apes to conquer the Earth and possibly also other parts of the solar system or the universe. However, the same brain has allowed the ape to construct hydrogen bombs that could destroy a large part of the Earth's biosphere, including its own species. Due to SBS, the winners of the evolutionary battle could be resilient undemanding organisms without such "progressive" brains, e.g. tardigrades, and such organisms might prevail on the Earth and most planets in the universe.

The second important advantage of SBS is that it often acts as if it could predict the requirements of the future environment. Selection is opportunistic, evolving the traits that are useful in the current situation even if the same trait could lead to the extinction of the population or even the species in the future. In contrast, SBS simulates predictive behavior – it promotes the accumulation of traits and properties that objectively ensure the long-term stability of the evolving entities.

For example, the ability of animals, e.g. birds, but possibly also humans, to slow down the rate of reproduction when some important resource is going to run out, might be useful for the populations or species in the long term. However, such a useful property cannot evolve by individual or group selection because of the universality of the 'tragedy of the commons' principle (Hardin, 1968). Lack of resources will make all members of a population suffer. However, the 'altruistic' individuals who 'voluntarily' decrease the rate of reproduction transfer fewer copies of their genes to the next generation than those individuals who continue reproducing at the maximum possible rate until resources (both for them and for the altruists) are depleted. Therefore, 'altruists' vanish in a few generations, leaving only 'selfish' individuals.

In contrast, SBS can promote the fixation of traits, sorts of safety catches, that ensure the reduction or stopping of reproduction before the resource is exhausted. For example, individuals in an overcrowded population might be stressed and therefore lose the ability to reproduce. From the viewpoint of individual selection, this property is maladaptive and its carriers are penalized by lower fitness. However, it is useful for the long-term stability of the species. Traits that prevent its loss to individual selection should accumulate by SBS. All extant species, the winners of the SBS contest, should therefore carry such safe catches that protect them from famine and the resulting extinction of the population. Of cause, some mutants can inactivate their safe catch and the descendants of such mutants might prevail in the population or species due to individual selection. However, in the long term, the species with stable enough safe catches remain due to the action of SBS.

5.5. Postadaptations, the adaptations that are not adaptive any more

Some traits that came into the existence as adaptations in the past lose their adaptiveness in current changed conditions. It is clear that particular adaptations were built up by a selection to tackle specific demands of an environment. When the conditions change, the traits lose their potential to increase the fitness of their carriers. Therefore, whenever we search for the possible function of a trait, we must take into account not only the functions and importance of the trait under present conditions, but also under past conditions. In species whose environment or style of life changed dramatically, e.g. in humans, the function of a trait under past conditions can be more important than its function, or the absence of function, in present conditions.

It is not easy to decide which conditions should be considered preferentially. For example, the style of life of a modern human has dramatically changed even during the past 100 years, mostly because of our victory over nearly all infectious diseases and dramatic technological advances resulting in even more dramatic changes in our social environment. Therefore, many traits or behavioral patterns that were adaptive less than 100 years ago have lost their adaptiveness in the modern world. Similarly, some traits that are adaptive now were not adaptive in the past and can be therefore considered as exaptations or spandrels.

It is not clear what reference time-frame we should use when we think about what is and what

is not an adaptation. Definitively, the traits that started to be adaptive only recently, e.g., our ability to text on a cell phone or drive a car, cannot be biological adaptations as there was not enough time for the spreading of corresponding alleles in our genepool by selection. Some adaptive traits, however, could have already arisen by fixation of favorable alleles by selection after the origin of pastoralism (e.g., the allele for digestion of lactose that stays active in adulthood (Gerbault et al., 2011)), probably other alleles later after the origin of agriculture, and others after the advent of an urban lifestyle. The latter resulted in the spreading of many alleles for resistance to infectious diseases. However, behavioral immunity is in some respects even more important than physiological immunity. We can therefore expect that some alleles for behavioral patterns useful in the protection of an individual or a population against infectious diseases, e.g., the alleles for social phobia, or xenophobia, or obsessive-compulsive disorder, to spread in response to the transition to life in crowded urban populations. On the other hand, other alleles lost their selective advantage in the changing environment. Corresponding traits lost their adaptiveness and were either eliminated from the population or remained there as vestiges of their past functions – the postadaptions.

BOX 4 Does Rh negativity spread in Europe because of the absence of toxoplasmosis?

About 16% of Europeans have Rh-negative blood type, i.e., they are carriers of mutated alleles of the RHD gene with a large deletion (Wagner & Flegel, 2000). This polymorphism is an old evolutionary riddle as the carriers of a less numerous allele, namely Rh-negative women in predominantly Rh-positive populations and Rh-positive men in predominantly Rh-negative populations, were systematically penalized by natural selection. Before the advent of modern prophylactic methods, Rh-positive children of Rh-negative mothers often died due to hemolytic disease of newborns (Bowman, 1997; Filbey, Hanson, & Wesstrom, 1995). In such a situation, RHD polymorphism should be unstable and only one type of RHD gene should remain in a population.

It has been theorized for a long time (Feldman, Nabholz, & Bodmer, 1969; Fisher, Race, & Taylor, 1944; Haldane, 1922), and recently also supported in several studies (Flegr, 2016; Flegr, Hoffmann, & Dammann, 2015; Flegr, Kuba, & Kopecký, 2020; Kaňková, Flegr, Toman, & Calda, 2019), that RHD polymorphism is sustained in human populations by heterozygote advantage (Flegr, Toman, Hula, & Kankova, 2020). It was shown that heterozygotes express higher viability (i.e., better health) than both Rh-negative and Rh-positive homozygotes. It turns out that the strong effect of Rh phenotype on physical and mental health was escaping the attention of researchers for about 90 years because Rh-positive homozygotes have even worse health than Rh-negative homozygotes and widely used serological techniques of Rh assessment do not discriminate between Rh-positive homozygotes and heterozygotes (Flegr, Toman, et al., 2020).

The frequency of Rh-negative individuals is highly variable across countries (Mourant, 1954). It is much higher in Europe and in people of European origin in other parts of the world (16%) than in Africa (5%) and Asia (1%). The high frequency of the allele for Rh-negativity, e.g. about 27% in Basks, was traditionally explained by founder effect and drift. Some results, however, suggest that the spreading of Rh-negative allele was driven by natural section.

One possible cause is the variable prevalence of toxoplasmosis (Novotná et al., 2008). Definitive hosts of the parasite *Toxoplasma gondii* are representatives of feline species. Various species of large and (especially) small cats are numerous and abundant in most habitable parts of the world. One important exception was Europe before the rather recent introduction of domestic cats (Torrey & Yolken, 1995). It is probable that 2000 years ago, the prevalence of toxoplasmosis in European populations was low in comparison to other parts of the world. Currently, the prevalence of toxoplasmosis is about 30% in Europe and it is decreasing at a rate of about 1% per year in most countries, probably due to recently increased hygienic standards and changes in alimentary habits (cooking from

cooled or frozen meat) (Tenter, Heckeroth, & Weiss, 2000).

Several studies have shown that Rh-positive subjects, especially heterozygotes, are protected against negative effects of latent toxoplasmosis. It has been shown, for example, that *Toxoplasma*-free, Rh-negative subjects have the shortest reaction times in simple reaction times tests. However, the psychomotor performance of Rh-negative subjects radically decreases when they are *Toxoplasma* infected, while the performance of Rh-positive subjects remains unchanged. Among *Toxoplasma*-infected subjects, those who are Rh-negative have the longest reaction times, while Rh-positive heterozygotes have the shortest reaction times (Flegr, Novotná, Lindová, & Havlíček, 2008; Novotná et al., 2008). In fact, the reaction times of *Toxoplasma*-positive heterozygotes are even better than those of non-infected heterozygotes. Such positive effect of toxoplasmosis (together with more recent results showing the better health of *Toxoplasma*-infected than *Toxoplasma*-free heterozygotes, Flegr 2020b) even suggests that our species is adapted to being *Toxoplasma*-infected – which was probably a natural status of our ancestors in Africa.

5.5.1. What is the environment of evolutionary adaptedness of our species?

It is widely discussed in cultural anthropology what ancestral environment has shaped our phenotype, including the mind. It is widely believed that for hundreds of thousands years, this environment was located in the African savannas where humans lived in small groups as hunters and gatherers (Heerwagen & Orians, 1993). Therefore, the African savanna and mixed woodland habitats is considered to be the environment of our evolutionary adaptedness (Tooby & Cosmides, 1990) by most anthropologists, and the adaptedness of particular human traits is judged in this context.

However, this opinion could be wrong for two reasons. First, the savanna might be the biotope in which human remnants have a higher chance to fossilize (especially in comparison with the African tropical forest), rather than the biotope in which our ancestors lived for the largest part of our species history (Roberts, Boivin, Lee-Thorp, Petraglia, & Stock, 2016). Second, sexually reproducing species can be expected to gather most of their adaptations in the environment in which they lived immediately after they originated by splitting from their ancestral species rather than in the environment in which they spent the largest part of their evolutionary history. According to several genetic theories, sexually reproducing species are evolutionarily plastic only for a short time (10-20 thousand years) after their origin by a specific type of speciation (usually peripatric speciation), later becoming macro-evolutionarily frozen and micro-evolutionarily elastic. For the rest of their existence, they cannot effectively respond to selection pressures (see the Box 5). In this macro-evolutionarily frozen state they could only passively wait for such changes of their environment that will cause their extinction, or for another speciation event that will turn the new species evolutionarily plastic, which allows them to adapt to the changed environment. Our closest relatives, chimpanzees and gorillas, live in tropical forests. It is therefore probable that our species originated in the tropical forest and we thus could be adapted to this biome. If this is true, then our adaptations for life in the savanna are in fact exaptations and we bear many postadaptations – traits that are not useful for the biotopes in which most of the present cultures live or lived until recently.

BOX 5. What can be the mechanism of the punctuated evolution?

In apparent contrast to the gradualistic view of modern evolutionary synthesis, Eldredge and Gould (Eldredge & Gould, 1972) argued that the typical mode of evolution of multicellular organisms is punctuated. Short periods of rapid phenotypical (i.e., anagenetic) change, which are mostly untraceable in the paleontological record, are followed by long periods of evolutionary stasis in which the phenotype of a species remained stable. The stasis typically covers 98-99% of the time of the existence of a species.

Several explanations for this punctuated nature of the evolution have been suggested in

the past 50 years, including four that suggested specific genetic mechanisms (for review see (Flegr, 2010)). Eldredge and Gould's original hypothesis was based on the E. Mayr theory of genetic revolution (Mayr, 1954, 1963) that could occur mainly due to a random shift of in genepool composition due to the sampling effect after splitting-off a small population during peripatric speciation. The funder-flush model (Carson, 1968) is based on the idea of an expansion of the population in an open ecological niche, which relaxes all forms of selection and allows the survival of recombinants and mutants with suboptimal phenotypes (crossing valleys in the adaptive landscape). The genetic transilience model (Templeton, 2008) explains the rise in the responsiveness to selection mainly by increasing the amount of selectable genetic variability due to the transformation of nonadditive (and therefore nonselectable) genetic variability to additive genetic variability in the arising species. The theory of frozen plasticity (Flegr, 1998, 2010, 2013) suggests that evolvability transiently rises when the alleles with frequency-dependent effects on fitness (that normally keeps a sexually reproducing species micro-evolutionarily elastic and therefore macro-evolutionarily frozen) are eliminated by the sampling effect and genetic drift in small split-off populations during peripatric speciation. All four of these theories explain the punctuated pattern of macroevolution, but they differ in the explicative potential regarding other evolutionary and ecological phenomena. They can also explain why most species are obsolete. Residing in an evolutionary stasis for most of their existence, sexual species are usually adapted to the past conditions in the time of their last significant anagenetic change rather than the current conditions.

5.5.2. Rudiments and atavisms

A species may bear not only the adaptations that are or were once useful for it, but also traits that were useful for their ancestral species. These traits (morphological, physiological, or even behavioral) could be divided into rudiments and atavisms. Rudiments are present in some form in all individuals of the species (e.g., the appendix of humans) while atavisms are present only in a fraction, sometimes a very small fraction, of them, namely in the individuals with special and often rare combinations of alleles (e.g., tail in humans). The rudiments and atavisms could be adaptive, i.e., could fulfill their biological functions, in the immediate ancestor of the species as well as in some distant ancestor. Again, when we search for the biological function of a trait, including a behavioral pattern, we should always keep in mind that the trait could be a rudiment or atavism and could have no biological function in the species under study.

5.6. Summary

Not all traits that seem like adaptations and have a biological function are adaptations in their narrow sense. Some can be classified as exaptations – the traits that evolved to serve another function than that they serve now. Some could be spandrels – the structures that come into existence because of the inherent laws of physics, chemistry, or geometry. Some biological adaptations come into existence by other processes than Darwinian selection – for example, by stability-based sorting or species selection. Some adaptations serve good not to an individual but allele, family, group of unrelated organisms, own species, or even foreign species. On the other hand, some traits that are not adaptive under current conditions were adaptive in the past evolutionary history of a species, sometimes even in time of its evolutionary plasticity hundreds of thousand years ago.

References

Ardlie, K. G. (1998). Putting the brake on drive: meiotic drive of t haplotypes in natural populations of mice. *Trends in Genetics, 14*, 189-193.

Baldwin, J. M. (1896). A new factor in evolution. *American Naturalist, 30*, 441-451.

Benne, R. (1992). Review - RNA editing in trypanosomes - The us(e) of guide RNAs. *Molecular Biology Reports, 16*, 217-227.

Berdoy, M., Webster, J. P., & Macdonald, D. W. (1995). The manipulation of rat behaviour by *Toxoplasma gondii Mammalia, 59*(4), 605-613.

Berdoy, M., Webster, J. P., & Macdonald, D. W. (2000). Fatal attraction in rats infected with *Toxoplasma gondii*. *Proceedings of the Royal Society B-Biological Sciences, 267*(1452), 1591-1594. doi: DOI 10.1098/rspb.2000.1182

Blanchard, R., & Bogaert, A. F. (1996). Homosexuality in men and number of older brothers. *American Journal of Psychiatry, 153*(1), 27-31.

Bouayed, J., & Bohn, T. (2020). Behavioral manipulation - key to the successful global spread of the new coronavirus SARS-CoV-2? *Journal of Medical Virology*. doi: 10.1002/jmv.26446

Bowman, J. (1997). The management of hemolytic disease in the fetus and newborn. [Review]. *Semin Perinatol, 21*(1), 39-44.

Camperio-Ciani, A., Corna, F., & Capiluppi, C. (2004). Evidence for maternally inherited factors favouring male homosexuality and promoting female fecundity. *Proceedings of the Royal Society B-Biological Sciences, 271*, 2217-2221.

Carson, H. L. (1968). The population flush and its genetic consequences. In R. C. Lewontin (Ed.), *Population Biology and Evolution* (pp. 123-137). Syracuse: Syracuse University Press. (Reprinted from: IN FILE).

Ciani, A. C., Battaglia, U., & Zanzotto, G. (2015). Human homosexuality: A paradigmatic arena for sexually antagonistic selection? *Cold Spring Harbor Perspectives in Biology, 7*(4). doi: 10.1101/cshperspect.a017657

Cloninger, C. R. (1998). The genetics and psychobiology of the seven-factor model of personality. In K. R. Silk (Ed.), *Biology of personality disorders* (pp. 63-92). Washington,DC: American Psychiatric Press, Inc. (Reprinted from: IN FILE).

Darwin, C. (1860). *On the origin of species by means of natural selection or the preservation of favoured races in the struggle for life* (Vol. 5th). London: Murray.

Dass, S. A. H., & Vyas, A. (2014). *Toxoplasma gondii* infection reduces predator aversion in rats through epigenetic modulation in the host medial amygdala. *Molecular Ecology, 23*(24), 6114-6122. doi: Doi 10.1111/Mec.12888

Dawkins, R. (1976). *The selfish gene*. Oxford: Oxford University Press.

Dawkins, R. (1982). *The extended phenotype, The gene as the unit of selection* (Vol. 1). Oxford: W.H. Freeman and Comp.

Dawkins, R. (1983). *The extended phenotype. The long reach of the gene*. New York, Oxford: Oxford University Press.

Dewar, C. S. (2003). An association between male homosexuality and reproductive success. *Medical Hypotheses, 60*, 225-232.

Eldredge, N., & Gould, S. J. (1972). Punctuated equilibria: an alternative to phyletic gradualism. In T. J. M. Schopf (Ed.), *Models in Paleontology* (pp. 82-115). San Francisco. (Reprinted from: IN FILE).

Emiliani, C. (1993). Extinction and viruses. *BioSystems, 31*, 155-159.

Feldman, M. W., Nabholz, M., & Bodmer, W. F. (1969). Evolution of the Rh polymorphism: A model for the interaction of incompatibility, reproductive compensation and heterozygote advantage *American Journal of Human Genetics, 21*, 171-193.

Filbey, D., Hanson, U., & Wesstrom, G. (1995). The prevalence of red cell antibodies in pregnancy correlated to the outcome of the newborn: a 12 year study in central Sweden. [Research Support, Non-U.S. Gov't]. *Acta Obstet Gynecol Scand, 74*(9), 687-692. doi: 10.3109/00016349509021175

Fisher, R. A., Race, R. R., & Taylor, G. L. (1944). Mutation and the Rhesus reaction. *Nature, 153*, 106-106.

Flegr, J. (1994). Chemostat-turbidostat discontinuum, r-K continuum and population-size regulating mechanism. *Acta Societatis Zoologica Bohemica, 58*, 143-149.

Flegr, J. (1998). On the "origin" of natural selection by means of speciation. *Rivista di Biologia-Biology Forum, 91*(2), 291-304.

Flegr, J. (2010). Elastic, not plastic species: frozen plasticity theory and the origin of adaptive evolution in sexually reproducing organisms. *Biol. Direct, 5*, 2.

Flegr, J. (2013). Microevolutionary, macroevolutionary, ecological and taxonomical implications of of punctuational theories of adaptive evolution. *Biol. Direct, 8*, 1.

Flegr, J. (2016). Heterozygote advantage probably maintains Rhesus factor blood group polymorphism: Ecological regression study. *PLoS ONE, 11*(1). doi: 10.1371/journal.pone.0147955

Flegr, J. (2017). Does *Toxoplasma* infection increase sexual masochism and submissiveness? Yes and no. *Communicative & Integrative Biology*. doi: 10.1080/19420889.2017.1303590

Flegr, J., & Escudero, D. Q. (2016). Impaired health status and increased incidence of diseases in *Toxoplasma*-seropositive subjects - an explorative cross-sectional study. *Parasitology, 143*(14), 1974-1989. doi: 10.1017/s0031182016001785

Flegr, J., Hoffmann, R., & Dammann, M. (2015). Worse health status and higher incidence of health disorders in Rhesus negative subjects. *PLoS ONE, 10*(10). doi: 10.1371/journal.pone.0141362

Flegr, J., & Kuba, R. (2016). The relation of *Toxoplasma* infection and sexual attraction to fear, danger, pain, and submissiveness. *Evolutionary Psychology, 14*(3). doi: 10.1177/1474704916659746

Flegr, J., Kuba, R., & Kopecký, R. (2020). Rhesus-minus phenotype as a predictor of sexual desire and behavior, wellbeing, mental health, and fecundity. *PLoS One, 15*(7), e0236134. doi: 10.1371/journal.pone.0236134

Flegr, J., Lenochová, P., Hodný, Z., & Vondrová, M. (2011). Fatal attraction phenomenon in humans: cat odour attractiveness increased for *Toxoplasma*-infected men while decreased for infected women. [Research Support, Non-U.S. Gov't]. *PLoS Neglected Tropical Diseases, 5*(11), e1389. doi: 10.1371/journal.pntd.0001389

Flegr, J., & Markos, A. (2014). Masterpiece of epigenetic engineering - how *Toxoplasma gondii* reprogrammes host brains to change fear to sexual attraction. *Molecular Ecology, 23*(24), 5934-5936. doi: Doi 10.1111/Mec.13006

Flegr, J., Milinski, M., Kankova, S., Hula, M., Hlavacova, J., & Sykorova, K. (2018). Latent toxoplasmosis and olfactory functions of Rh positive and Rh negative subjects. *PLoS ONE, 13*(12). doi: 10.1371/journal.pone.0209773

Flegr, J., Novotná, M., Lindová, J., & Havlíček, J. (2008). Neurophysiological effect of the Rh factor. Protective role of the RhD molecule against *Toxoplasma*-induced impairment of reaction times in women. *Neuroendocrinology Letters, 29*, 475-481.

Flegr, J., Prandota, J., Sovickova, M., & Israili, Z. H. (2014). Toxoplasmosis - A global threat. Correlation of latent toxoplasmosis with specific disease burden in a set of 88 countries. *PLoS ONE, 9*(3). doi: 10.1371/journal.pone.0090203

Flegr, J., Preiss, M., Klose, J., Havlíček, J., Vitáková, M., & Kodym, P. (2003). Decreased level of psychobiological factor novelty seeking and lower intelligence in men latently infected with the protozoan parasite *Toxoplasma gondii.* Dopamine, a missing link between schizophrenia and toxoplasmosis? *Biological Psychology, 63*, 253-268.

Flegr, J., Toman, J., Hula, M., & Kankova, S. (2020). The role of balancing selection in maintaining human RhD blood group polymorphism: A preregistered cross-sectional study. *Journal of Evolutionary Biology*. doi: 10.1111/jeb.13745

Flegr, J., Zitkova, S., Kodym, P., & Frynta, D. (1996). Induction of changes in human behaviour by the parasitic protozoan *Toxoplasma gondii*. *Parasitology, 113*, 49-54.

Gaskell, E. A., Smith, J. E., Pinney, J. W., Westhead, D. R., & McConkey, G. A. (2009). A unique dual activity amino acid hydroxylase in *Toxoplasma gondii PloS ONE, 4*, e4801.

Gerbault, P., Liebert, A., Itan, Y., Powell, A., Currat, M., Burger, J., . . . Thomas, M. G. (2011). Evolution of lactase persistence: an example of human niche construction. *Philosophical Transactions of the Royal Society B-Biological Sciences, 366*(1566), 863-877. doi: 10.1098/rstb.2010.0268

González-Forero, M. (2015). Stable eusociality via maternal manipulation when resistance is costless. [https://doi.org/10.1111/jeb.12744]. *Journal of Evolutionary Biology, 28*(12), 2208-2223. doi: 10.1111/jeb.12744

Gould, S. J. (1989). *Wonderful Life*. New York W.W. Norton&Company.

Gould, S. J. (1997). Evolution: The pleasures of pluralism. *The New York Review of Books, 44*(11), 47-52.

Gould, S. J. (2002). *The Structure of Evolutionary Theory*. Camridge: The Belknap Press of Harvard University Press.

Gould, S. J., & Lewontin, R. C. (1979). The spandrels of San Marco and the Panglossian paradigm: a critique of the adaptationist programme. *Proceeding of the Royal Society of London Series B-Biological Sciences, 205*, 581-598.

Haldane, J. B. S. (1922). Sex ratio and unisexual sterility in hybrid animals. *Journal of Genetics, 12*, 101-109.

Hamilton, W. D. (1964a). The genetical evolution of social behaviour. I. *Journal of Theoretical Biology, 7*, 1-16.

Hamilton, W. D. (1964b). The genetical evolution of social behaviour.II. *Journal of Theoretical Biology, 7*, 17-52.

Hardin, G. (1968). The tragedy of the commons. *Science, 162*, 1243-1248.

Heerwagen, J. H., & Orians, G. H. (1993). Humans, habitats and aesthetics. In S. R. Kellert & E. O. Wilson (Eds.), *The Biophilia Hypothesis* (pp. 138-172): Island Press.

Herrmann, B., Thoni, C., & Gachter, S. (2008). Antisocial punishment across societies. *Science, 319*(5868), 1362-1367. doi: 10.1126/science.1153808

Hueffer, K., Khatri, S., Rideout, S., Harris, M. B., Papke, R. L., Stokes, C., & Schulte, M. K. (2017). Rabies virus modifies host behaviour through a snake-toxin like region of its glycoprotein that inhibits neurotransmitter receptors in the CNS. *Scientific Reports, 7*. doi: Artn 12818

10.1038/S41598-017-12726-4

Kaňková, Š., Flegr, J., Toman, J., & Calda, P. (2019). Maternal RhD heterozygous genotype is associated with male biased secondary sex ratio. *BioRxiv*, 1-23.

Kirkpatrick, R. C. (2000). The evolution of human homosexual behavior. *Current Anthropology, 41*(3), 385-413. doi: Doi 10.1086/300145

Kubena, A. A., Houdek, P., Lindova, J., Priplatova, L., & Flegr, J. (2014). Justine effect: Punishment of the unduly self-sacrificing cooperative individuals. *PLoS ONE, 9*(3). doi: 10.1371/journal.pone.0092336

Lewis, P. D., Jr. (1974). Helminths of terrestrial molluscs in Nebraska. II. Life cycle of Leucochloridium variae McIntosh, 1932 (Digenea: Leucochloridiidae). *Journal of Parasitology, 60*, 251-255.

Lieberman, B. S., & Vrba, E. S. (2005). Stephen Jay Gould on species selection: 30 years of insight. *Paleobiology, 31*, 113-121.

Lindová, J., Kuběna, A. A., Šturcová, A., Křivohlavá, R., Novotná, M., Rubešová, A., . . . Flegr, J. (2010). Pattern of money allocation in experimental games supports the stress hypothesis of gender differences in *Toxoplasma gondii*-induced behavioural changes. *Folia Parasitologica, 57*, 136-142.

Lindová, J., Novotná, M., Havlíček, J., Jozífková, E., Skallová, A., Kolbeková, P., . . . Flegr, J. (2006). Gender differences in behavioural changes induced by latent toxoplasmosis. *International Journal for Parasitology, 36*, 1485-1492.

Maynard Smith, J. (1964). Group selection and kin selection. *Nature, 201*(4924), 1145-1147. doi: 10.1038/2011145a0

Mayr, E. (1954). Change of genetic environment and evolution. In E. B. Ford, J. Huxley & A. C. Hardy (Eds.), *Evolution as a Process* (pp. 157-180). Princeton: Princeton University Press. (Reprinted from: IN FILE).

Mayr, E. (1963). *Animal species and evolution*. Cambridge: Harvard University Press.

Miller, E. M. (2000). Homosexuality, birth order, and evolution: Toward an equilibrium reproductive economics of homosexuality. *Archives of Sexual Behavior, 29*(1), 1-34. doi: Doi 10.1023/A:1001836320541

Mourant, A. E. (1954). *The distribution of the human blood groups* (2d ed.). Oxford: Blackwell Scientific Publication.

Novotná, M., Hanušová, J., Klose, J., Preiss, M., Havlíček, J., Roubalová, K., & Flegr, J. (2005). Probable neuroimmunological link between *Toxoplasma* and cytomegalovirus infections and personality changes in the human host. *BMC Infectious Diseases, 5*, 54.

Novotná, M., Havlíček, J., Smith, A. P., Kolbeková, P., Skallová, A., Klose, J., . . . Flegr, J. (2008). *Toxoplasma* and reaction time: Role of toxoplasmosis in the origin, preservation and geographical distribution of Rh blood group polymorphism. *Parasitology, 135*, 1253-1261.

Pleasant, A., & Barclay, P. (2018). Why hate the good guy? Antisocial punishment of high cooperators is greater when people compete to be chosen. *Psychological Science, 29*(6), 868-876. doi: 10.1177/0956797617752642

Poirotte, C., Kappeler, P. M., Ngoubangoye, B., Bourgeois, S., Moussodji, M., & Charpentier, M. J. E. (2016). Morbid attraction to leopard urine in *Toxoplasma*-infected chimpanzees. *Current Biology, 26*(3), R98-R99. doi: 10.1016/j.cub.2015.12.020

Prandovszky, E., Gaskell, E., Martin, H., Dubey, J. P., Webster, J. P., & McConkey, G. A. (2011). The neurotropic parasite *Toxoplasma gondii* increases dopamine metabolism. *PLoS ONE, 6*(9), e23866.

Roberts, P., Boivin, N., Lee-Thorp, J., Petraglia, M., & Stock, J. (2016). Tropical forests and the genus *Homo*. *Evolutionary Anthropology, 25*(6), 306-317. doi: 10.1002/evan.21508

Rozsa, L. (1999). Influencing random transmission is a neutral character in hosts. *Journal of Parasitology, 85*(6), 1032-1035.

Rozsa, L. (2000). Spite, xenophobia, and collaboration between hosts and parasites. *OIKOS, 91*(2), 396-400.

Ruse, M. (1988). *Homosexuality : a philosophical inquiry*. New York, NY: Blackwell.

Shanley, D. P., Sear, R., Mace, R., & Kirkwood, T. B. L. (2007). Testing evolutionary theories of menopause. *Proceedings of the Royal Society B-Biological Sciences, 274*, 2943-2949.

Sherman, P. W. (1998). Animal behavior - The evolution of menopause. *Nature, 392*(6678), 759-+.

Skutch, A. F. (1935). Helpers at the nest. *The Auk, 52*(3), 257-273. doi: 10.2307/4077738

Slater, E. (1962). Birth order and maternal age of homosexuals. *Lancet, 1*(7220), 69-71.

Stoltzfus, A. (1999). On the possibility of constructive neutral evolution. *Journal of Molecular Evolution, 49*(2), 169-181.

Sylwester, K., Herrmann, B., & Bryson, J. J. (2013). *Homo homini lupus*? Explaining antisocial punishment. *Journal of Neuroscience, Psychology, and Economics, 6*(3), 167-188. doi: 10.1037/npe0000009

Templeton, A. R. (2008). The reality and importance of founder speciation in evolution. *BioEssays, 30*, 470-479.

Tenter, A. M., Heckeroth, A. R., & Weiss, L. M. (2000). *Toxoplasma gondii*: from animals to humans. *International Journal for Parasitology, 30*(12-13), 1217-1258.

Thanukos, A. (2009). How the adaptation got its start. *Evolution: Education and Outreach, 2*(4), 612-616. doi: 10.1007/s12052-009-0170-z

Toman, J., & Flegr, J. (2017). Stability-based sorting: The forgotten process behind (not only) biological evolution. *Journal of Theoretical Biology, 435*, 29-41. doi: 10.1016/j.jtbi.2017.09.004

Tooby, J., & Cosmides, L. (1990). The past explains the present - emotional adaptations and the structure of ancestral environments. *Ethology and Sociobiology, 11*(4-5), 375-424. doi: Doi 10.1016/0162-3095(90)90017-Z

Torrey, E. F., Bartko, J. J., Lun, Z. R., & Yolken, R. H. (2007). Antibodies to *Toxoplasma gondii* in patients with schizophrenia: A meta-analysis. *Schizophrenia Bulletin, 33*, 729-736.

Torrey, E. F., Bartko, J. J., & Yolken, R. H. (2012). *Toxoplasma gondii* and other risk factors for schizophrenia: An update. *Schizophrenia Bulletin, 38*(3), 642-647. doi: 10.1093/schbul/sbs043

Torrey, E. F., & Yolken, R. H. (1995). Could schizophrenia be a viral zoonosis transmitted from house cats. *Schizophrenia Bulletin, 21*(2), 167-171.

Trivers, R. L. (1974). Parent-offspring conflict. *American Zoologist, 14*(1), 249-264.

Vanboven, M., Weissing, F. J., Heg, D., & Huisman, J. (1996). Competition between segregation distorters: Coexistence of ''superior'' and ''inferior'' haplotypes at the t complex. *Evolution, 50*, 2488-2498.

Vrba, E. S. (1984). What is species selection? *Systematic Zoology, 33*, 318-328.

Vrba, S., & Gould, S. J. (1986). The hierarchical expansion of sorting and selection: Sorting and selection cannot be equated. *Paleobiology, 12*, 217-228.

Wagner, F. F., & Flegel, W. A. (2000). RHD gene deletion occurred in the Rhesus box. *Blood, 95*(12), 3662-3668.

Williams, G. C. (1966). *Adaptation and natural selection*. Princeton: Princeton University Press.

Wilson, D. S., & Sober, E. (1994). Reintroducing group selection to the human behavioral-sciences. *Behavioral and Brain Sciences, 17*(4), 585-608. doi: Doi 10.1017/S0140525x00036104

Wilson, E. O. (1975). *Sociobiology: The new synthesis*. Cambridge: Belknap Press of Harvard University Press.