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Xenoadaptations

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Synonyms

Adaptations without adaptive value;
Aloadaptations

Definition

Xenoadaptation is an evolutionary adaptation that
involves the development of a trait or characteris-
tic in an individual, which provides advantages to
another biological entity, including individuals
of different species, rather than the individual
exhibiting the trait.

Xenoadaptations: Beyond Individual Fitness

The traditional Darwinian model of evolution
operates on the premise that adaptations enhanc-
ing the biological fitness of an individual relative
to others within the same population arise
throughout evolutionary history. In classical the-
ory, the potential role of group selection, which
could create adaptations that benefit all members
of a specific population against members of other
populations of the same species, is also debated at
most. It was not until the 1960s that studies began
challenging this paradigm, suggesting that adap-
tations could emerge throughout biological evo-
lution, such as morphological structures or
behavioral patterns, which do not necessarily ben-
efit their bearer but instead aid another biological
entity. Prime examples of these kinds of adapta-
tions are morphological structures on a host's
body, whose formation is often attributed to a
parasite.

Xenoadaptations Focused on Allele Interests: The Outlaw Alleles Phenomenon

In the broadest sense, all traits can be considered
xenoadaptations. They collectively form an indi-
vidual's phenotype and typically increase biolog-
ical fitness of their carriers. However, as
demonstrated by William Hamilton (Hamilton,
1964a, b) and further elaborated and popularized
by Richard Dawkins (Dawkins, 1976, 1982),
these traits actually serve the interests of the spe-
cific allele responsible for their creation. What
appears outwardly as competition between indi-
viduals carrying different forms of a trait for the
highest fitness is, in reality, competition between
various alleles at individual loci for the greatest
number of self-copies transferred to subsequent
generations. The vast majority of alleles achieve
this goal by contributing to the creation of traits
that increase the fitness of their carrier. It is more

than debatable to classify this type of trait as a xenoadaptation, as the term adaptation would be completely emptied. Or, more precisely, it could only be used for adaptations in asexually reproducing organisms, where the evolution of adaptive traits is not driven by intra-locus competition, but by Darwinian individual selection.

Some alleles, however, choose different paths to achieve their ultimate goal of increasing the number of self-copies. This category includes, for example, ultra-selfish alleles (outlaw alleles), which directly reduce the biological fitness of the individual whose genome they are in, while at the same time increasing the chances of their own transfer to the next generation. One of the longest known outlaw alleles is the t-allele in the house mouse (Ardlie, 1998; Vanboven et al., 1996). In the heterozygous state, its carriers have shorter tails. In addition, however, it causes the males to have half the number of viable sperms, which in the mouse, where the female mates with a larger number of males in a short time frame and where sperm competition occurs in the female reproductive tract, necessarily represents a significant decrease in biological fitness. Nevertheless, all viable sperms carry the t-allele, not the wild allele found on the sister chromosome. The t-allele spreads in the mouse population, and its fixation is mainly prevented by the fact that, in the diploid state, it is lethal to its carrier. It is quite possible that the promiscuity of female mice (mating with several males during estrus) is a defensive strategy against the spread of t-alleles.

In nature, there are many cases where outlaw alleles are found on sex chromosomes. In such instances, their presence in the population typically results in a drastic shift in the sex ratio, leading these alleles to be referred to as Sex Ratio Distorters (SRD) (Carvalho & Vaz, 1999). In extreme cases, members of a particular sex may disappear entirely from the population, causing the population to become extinct. It is possible that outlaw alleles are similarly common on other chromosomes, but their presence is not indicated by the striking phenomenon of a shift in the sex ratio.

Some alleles, in their "effort" to achieve the most effective spread, go even further. In the heterozygous state, they use the mechanism of gene conversion and overwrite the allele located on the homologous chromosome to themselves. Alternatively, using meiotic drive, they deceive the mechanism of meiosis during the formation of female gametes, ensuring that they end up in the nucleus of the oocyte, while the other allele ends up in the polar body. Current data show that these manifestations of selfish or ultra-selfish alleles are surprisingly numerous in nature.

Before the emergence of sexual reproduction based on amphimixis (the restoration of ploidy after cell division by fusion of two haploid gametes or their nuclei), the evolution of adaptive traits proceeded in the classical Darwinian manner. Individuals competed with each other for maximum biological fitness, usually for the maximum number of offspring that would reach reproductive age and reproduce. When studying a certain adaptive trait in asexually reproducing species, the question to be asked is how this trait contributes to the biological fitness of its carrier. In sexually reproducing species, the researcher must ask the same question differently. The trait may not increase the biological fitness of its carrier at all and may even decrease it. A biologist investigating the function of a specific trait and the cause of its emergence must ask how the trait increases the number of copies of the allele responsible for its formation, transmitted to the next generation. In many cases, the same answer will be obtained for both questions, but in some cases, the correct answer to the first question will not exist.

Xenoadaptations Serving Other Members of the Same Species

In many instances, an individual exhibits a behavior pattern that benefits members of the same species at the expense of themselves. This behavior is often interpreted as altruistic and it is assumed that it evolved due to kin selection, group selection, or that it is so-called reciprocal altruism. However, in several cases, it may not be genuine altruism but rather the result of manipulation by another member of the same species. Whether to label such "involuntary" behavior as altruistic or not remains a matter of terminology.

One example of this involuntary altruistic behavior is found in some forms of eusociality (Hamilton, 1964a, b). In eusocial Hymenoptera species, typically only two individuals reproduce within a nest, while the other nest members, usually sterile females, assist in producing their offspring. In many cases, an individual can achieve higher inclusive fitness by adopting this behavior rather than reproducing themselves. This is observed in monogamous haplodiploid species, where a female passes more of her genes to the next generation through her sister than through her daughter. However, evidence suggests that this may not be an extreme form of altruism involving the individual's resignation from reproduction but rather succumbing to manipulation by the queen. When a queen is removed from a nest, many workers begin to lay eggs. In some instances, workers attempt to lay their eggs even when the original queen is still present. Even under normal circumstances, the collapse of the eusocial reproductive system is largely prevented by workers monitoring each other and removing eggs laid by their fellow workers.

Manipulation by parents or siblings may also occur, albeit not as extreme, in many other species and is highly likely to be present in our own species. One evolutionary enigma is male homosexuality (Ruse, 1988; Trivers, 1974). Numerous studies show that male homosexuals have significantly lower biological fitness than heterosexuals. At the same time, many studies have demonstrated that the probability of a man having homosexual preferences and behavior significantly increases with the number of older brothers (Slater, 1962). One possibility is that homosexuality arises from manipulation by the maternal organism, aiming for a more efficient distribution of family resources. In a highly stratified, polygamous society, only the wealthiest men have the opportunity to reproduce. In this situation, it is more beneficial for a family to invest resources in one son rather than several. Homosexuality among later-born sons facilitates this distribution of family resources. The maternal organism may alter a son's sexual preferences during embryonic development, for example, through hormonal action on the developing nervous system. The mother's information about her previous sons can be obtained through the presence of antibodies against male embryo-specific antigens. A 2017 study showed that the maternal immunological response to the Y-chromosome-linked protein neuroligin plays a crucial role in the development of male sexuality. Neuroligin is a cell adhesion molecule thought to play an essential role in specific cell-cell interactions in brain development (Bogaert et al., 2018).

A related hypothesis that offers another possible explanation for homosexuality is the sibling manipulation hypothesis (Flegr, 2022). According to this hypothesis, older brothers program the maternal organism during their own embryogenesis so that later-born brothers have a higher probability of being born homosexual. By orchestrating this, older brothers serve their own interests by lessening the competition for material resources and, most significantly, mating partners. In doing so, they compromise an essential component of their inclusive fitness. Nevertheless, according to Hamilton's rule, resources invested by the older brother in his offspring have twice the value as the same amount of resources invested in younger brothers' offspring (Hamilton, 1964a, b). As a result, the sibling manipulation hypothesis offers a more plausible explanation for the fraternal birth order effect on male homosexuality than the parental manipulation hypothesis.

More frequent instances of manipulation in humans, primates, and possibly other animals likely occur between unrelated individuals of the same species and manifest at the ethological rather than physiological level. In humans, the personality trait of Machiavellianism is recognized, characterized by an increased tendency to manipulate others (Davis et al., 2019). Some studies suggest that men exhibiting higher Machiavellian traits have increased fecundity (Carter, Lyons, & Brewer, 2018) and women preferring men with higher Machiavellianism traits have lower fecundity than their same-age peers (Marcinkowska et al., 2016). Additionally, men expressing high Machiavellianism have a higher number of sexual partners (Jonason et al., 2009), a modern-day marker of reproductive success (Kanazawa, 2003).

Parasite-Induced Xenoadaptations in Hosts

Important and numerous groups of xenoadaptations are traits induced by parasites in their hosts. The aim of these traits, such as an increase in body size, can be to redirect resources from host reproduction to the growth and multiplication of the parasite. Often, the purpose of these xenoadaptations, especially when it involves changes in host behavior, is to increase the probability of parasite transmission to uninfected individuals of the host species or between intermediate and definitive hosts.

For instance, when infected by the parasitic fluke *Leucochloridium paradoxum*, the amber snail *Succinea putris* experiences a transformation of one of its tentacles into a large, conspicuous, pulsating organ. This "organ" is an adaptation of the fluke, not the snail. The fluke's evolution has optimized the transformed tentacle's morphology and appearance to attract insectivorous birds, its definitive hosts. Birds then consume the transformed tentacle, which contains hundreds of microscopic fluke larvae that develop into sexually reproducing adults within the bird's body (Lewis, 1974).

Numerous examples of intricate morphological structures, whose formation is driven by parasitic genes, are found in plants. These structures, known as galls, appear on plant leaves and other organs, serving as microhabitats for various phylogenetically unrelated groups of phytoparasites, including insects, mites, fungi, bacteria, and viruses. Although galls are formed from plant cells, they are often genetically or epigenetically modified to produce specific chemicals that nourish the parasitic organisms responsible for their formation.

Many zooparasites manipulate the most adaptable aspect of their host's phenotype – behavior – to increase transmission chances between infected and non-infected hosts. For example, the protozoan parasite *Toxoplasma gondii* can alter the behavior of its intermediate host, typically a rodent, causing it to lose its instinctive fear of cat odors and instead become attracted to them (Berdoy et al., 2000). Recent research suggests that epigenetic modifications (likely demethylation) play a significant role in reprogramming regulatory elements of specific genes within the host's medial amygdala neurons (Dass & Vyas, 2014; Flegr & Markos, 2014). By inducing this behavioral change, *Toxoplasma* enhances its likelihood of transmission from intermediate to definitive hosts through predation.

Conclusions

The concept of xenoadaptation challenges the traditional Darwinian view of evolution, positing that evolutionary adaptations can arise that benefit entities beyond the individual expressing the trait. This vast landscape of xenoadaptations spans a myriad of phenomena, from alleles directing phenotypic manifestations that are detrimental to their carrier to enforced altruistic behaviors benefiting manipulative individuals of the same species, and extending to the impacts of parasites that induce changes in the host species' phenotype. These changes are adaptive from the parasite's perspective and maladaptive from the host's standpoint.

It's crucial to note that while many xenoadaptations might decrease an individual's fitness, they enhance the dissemination of specific alleles responsible for certain traits within the gene pool of their carrier's species or even within the gene pool of another species. This leads to the understanding that to fully comprehend the origins and consequences of specific traits, it's necessary to consider the interests of individual alleles, rather than simply focusing on the visible organism.

In the realm of intraspecies interactions, xenoadaptation underscores the potential for manipulation among individuals. What might be perceived as altruistic behaviors could indeed be involuntary actions, prompted by manipulation from other members of the same species. This subtle complexity complicates the interpretation of altruistic behaviors, raising certain questions that carry ethical and philosophical implications.

In the context of parasite-host dynamics, xenoadaptations provide an extraordinary showcase of evolutionary ingenuity. By inducing changes in host behavior or physiology, parasites can enhance their own survival and proliferation.

The frequency of this phenomenon in nature remains uncertain, but it's conceivable that it's far more common than currently presumed. It's even plausible that a significant proportion of disease symptoms are, in fact, the result of the manipulative activities of pathogens. This insight could have practical implications in medicine, as at times, it could be the manipulative activity of pathogens, rather than their multiplication, that might serve as the target of therapeutic interventions.

Cross-References

► Altruism
► Dark Triad
► Manipulation
► Sibling Manipulation Hypothesis
► *Toxoplasma* Infection

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.

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*, 1591–1594.

Bogaert, A. F., Skorska, M. N., Wang, C., Gabrie, J., MacNeil, A. J., Hoffarth, M. R., VanderLean, D. P., Zucker, K. J., & Blanchard, R. (2018). Male homosexuality and maternal immune responsivity to the Y-linked protein NLGN4Y. *Proceedings of the National Academy of Sciences of the United States of America, 115*, 302–306.

Carter, G. L., Lyons, M., & Brewer, G. (2018). Lifetime offspring and the dark triad. *Personality and Individual Differences, 132*, 79–83.

Carvalho, A. B., & Vaz, S. C. (1999). Are drosophila SR drive chromosomes always balanced? *Heredity, 83*, 221–228.

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*, 6114–6122.

Davis, A. C., Visser, B. A., Volk, A. A., Vaillancourt, T., & Arnocky, S. (2019). The relations between life history strategy and dark personality traits among young adults. *Evolutionary Psychological Science, 5*, 166–177.

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

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

Flegr, J. (2022). Adaptations, by-products, and spandrels. In T. K. Shackelford (Ed.), *Cambridge handbook of evolutionary perspectives on sexual psychology: volume 1, foundations* (Vol. 1, pp. 87–113). Cambridge University Press.

Flegr, J., & Markos, A. (2014). Masterpiece of epigenetic engineering – how *toxoplasma gondii* reprogrammes host brains to change fear to sexual attraction. *Molecular Ecology, 23*, 5934–5936.

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.

Jonason, P. K., Li, N. P., Webster, G. D., & Schmitt, D. P. (2009). The dark triad: Facilitating a short-term mating strategy in men. *European Journal of Personality, 23*, 5–18.

Kanazawa, S. (2003). Can evolutionary psychology explain reproductive behavior in the contemporary United States? *Sociological Quarterly, 44*, 291–302.

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

Marcinkowska, U. M., Lyons, M. T., & Helle, S. (2016). Women's reproductive success and the preference for dark triad in men's faces. *Evolution and Human Behavior, 37*, 287–292.

Ruse, M. (1988). *Homosexuality: A philosophical inquiry* (1st ed.). Blackwell.

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

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