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Short Review

The evolution of maladaptation

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This review contains a description of a research program for the study of maladaptation, defined here in terms of deviation from adaptive peaks. Maladaptation has many genetic causes, including mutation, inbreeding, drift, gene flow, heterozygote advantage and pleiotropy. Degrees of maladaptation are determined by genetic architecture and the relationship between the rates of selective, environmental change and the nature and extent of genetic responses to selection. The empirical analysis of maladaptation requires: (1) recognition of putative maladaptation, using methods from phylogenetics, teleonomy, development and genetics, followed by an assessment of the nature and degree of deviation from adaptation, using studies of natural selection and teleonomy; (2) determination of the causes of the deviation, using analyses of genetics, development, or other methods. Conditions for unambiguously identifying maladaptation are considerably more stringent than those for demonstrating adaptation and remarkably few studies have clearly identified and characterised maladaptative traits. A thorough understanding of the nature of phenotypic variation will never be achieved without an analysis of the scope and usual causes of maladaptation.

Keywords: adaptation, adaptive peaks, evolution, maladaptation.

'But by far the most important consideration is that the chief part of the organization of every being is simply due to inheritance; and consequently, though each being assuredly is well fitted for its place in nature, many structures now have no direct relation to the habits of life of each species'. Darwin (1859; p. 199).

Introduction

The study of phenotypic adaptation and its genetic basis is central to evolutionary biology. The term 'adaptation' has accumulated myriad definitions (reviews in Reeve & Sherman, 1993; Rose & Lauder, 1996) but adaptations are forms of traits that are always construed as the result of natural selection, whereas individuals with variant traits that are less well fit to the environment exhibit lower reproductive success. However, the fit of traits to environments epitomized in the term adaptation ('fit for', from the Greek 'ad aptos') can never be perfect, partly because organisms are always adapted to at least one generation in the past. Thus, some degree of deviation from the maximal possible degree of adaptation is always expected. Such deviations have been analysed under a variety of rubrics, using the tools of population and quantitative genetics, developmental biology, and behavioural and evolutionary ecology. The purpose of this review is to synthesize perspectives and information from these disparate disciplines and to analyse the nature and causes of maladaptation. Firstly, I have categorized definitions of 'adaptation', translated them into definitions of 'maladaptation' and discussed the usefulness of these definitions for addressing various questions. Secondly, I have discussed the causes of maladaptation at different levels in the hierarchy of biological information (Arnold, 1992). Thirdly, I have focused on methods for identifying and analysing maladaptation, drawing on approaches that have developed independently in different fields and seeking to reconcile the often-acrimonious opinions of geneticists and ecologists regarding the usefulness of their research methods.

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Defining adaptation and maladaptation

Our primary criterion for choosing a definition should be its utility for answering questions of interest. Definitions of adaptation can be categorized into four main types: (1) teleonomic; (2) phylogenetic; (3) population genetic; (4) quantitative genetic.

Teleonomic definitions of adaptation developed in evolutionary and behavioural ecology (Thornhill, 1990) and focus on the functional design of phenotypic traits – how they have been 'designed' by the blind watchmaker of selection to 'function' in some environmental context. These definitions emphasize the selective maintenance of traits, and involve identification and quantification of the fit between form and function (Reeve & Sherman, 1993). The implementation of teleonomic definitions into a research program requires specification of a strategy set (a range of possible trait forms), an application of some fitness criterion (what is maximized, such as performance of some task or a component of fitness) and deliniation of constraints (fixed parameters that bound the analyses and link the strategy set with the fitness criterion). This research program is based on the premise

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that a history of natural selection leads to forms of traits (adaptations) that are optimal in a given environmental context, within the range of the strategy set. Quantification of current selection is not necessary under this program, because current selection need not reflect the selective pressures during the trait's evolution (Thornhill, 1990). This optimization approach to adaptation has been useful in characterising the nature and causes of associations between traits, and between traits and environments, especially for aspects of behaviour and life history.

Under a teleonomic research program, 'maladaptation' can be defined as prevalence in a population of a 'strategy' (a form of a phenotype), that does not lead to the highest relative fitness of the strategies in the allowed set. This viewpoint has often been difficult to implement, due to the difficulties involved in defining a complete and accurate set of strategies and constraints, the complex selective pressures on many traits and the challenge of measuring fitness in an evolutionarily meaningful way (Lewontin, 1979). Moreover, if a formerly neutral trait (e.g. red vs. yellow flowers) comes under selection, such that plants with red flowers have higher reproductive success (e.g. their pollen fertilizes more ovules), then red flowers will be called an adaptation under some teleonomic definitions (e.g. Reeve & Sherman, 1993), even before there has been any genetic response to selection.

Phylogenetic definitions of adaptation require the use of a phylogeny to infer the origin of a trait, inference of the 'selective regime' under which the trait arose, and an analysis of performance of the trait under its ancestral and current selective regime (Baum & Larson, 1991 and references therein). If the trait arose under its current selective regime and exhibits higher performance than its antecedent, then it is considered an adaptation. This definition focuses on joint analyses of the origin and maintenance of traits, under the presumption that traits changing in function over the specific time period considered should be categorized differently from traits that do not. Baum & Larson (1991) provide explicit criteria for identifying maladaptation under this phylogenetic perspective; a trait is maladaptive (a.k.a. 'disadapted', in their lexicon) if it exhibits lower utility (performance at some task) than its antecedent state, in its selective regime (environmental context). This research programme for categorizing traits has yet to take hold, apparently due to uncertainties of inferring ancestral states and selective regimes (Leroi *et al*., 1994) and a stronger focus on the importance of testing statistically for adaptation via quantifying convergence (Doughty, 1996).

Population and quantitative genetic perspectives on adaptation involve: (1) relating alleles and genotypes to phenotypes and fitness in current populations; or (2) quantifying current selection, and expected or observed responses to selection, on single-locus and polygenic traits. For population geneticists, adaptation involves gene substitutions driven by selection, or the maintenance of variation via selection. In the case of gene substitution, the degree of adaptation and maladaptation can be quantified as the 'substitutional load' or the 'lag load' (e.g. Maynard Smith, 1976), measures of: (1) the reproductive excess required to prevent extinction of a small population subject to unfavourable environmental change, or (2) the degree to which the fitness of the current genotype lags behind the optimal genotype in a changing environment (see also Kirkpatrick, 1996). Gillespie (1991; p. 63 and p. 305) concludes that such loads are often heavy as populations are usually far from allelic equilibrium, apparently; this is because genotypic adaptive peaks outrun responses to selection. Load-based metrics of maladaptation require estimating the relation between alleles or genotypes and fitness, and where feasible they provide a strong link from maladaptation to its causes.

Quantitative geneticists normally discuss adaptation in the context of phenotypic adaptive topologies, where local and global peaks represent optimal population states (Schluter & Nychka, 1994). For quantitative traits, approaches to these peaks are governed by the form and strength of multivariate selection and response to selection which can be predicted (at least in the short term) using the genetic variance-covariance matrix (Shaw *et al*., 1995). By this perspective, maladaptation can be quantified as the distance of a population from the nearest adaptive peak (Loeschcke, 1987; Bjorklund, 1996). This distance is largely a function of the degree to which the population does not exactly track the vector of directional selection, as a result of aspects of genetic response to selection that prevent the largest possible selection-driven step upwards. Stabilizing selection serves to define the optimal, 'adapted' state or peak, though a population may spread more or less widely down its sides. As we can measure selection in the field, construct adaptive surfaces and estimate **G** in the field or laboratory, this measure of maladaptation is feasible to employ. Moreover, it provides a bridge between genetic mechanisms of microevolutionary change, notably additive genetic variance, pleiotropy, and linkage disequilibrium, and aspects of ecology, as depicted in adaptive landscapes. To the extent that adaptive topologies move across generations, or populations are displaced downhill by genetic happenstance, populations will be off their peaks and thus, to some quantifiable degree, maladapted.

I have purposefully avoided the term 'constraint' in the exposition above as most authors use it in a heuristic and general sense to refer to deviations from some expected course of evolution (e.g. Maynard Smith *et al*., 1985; Antonovics & van Tienderen, 1991; Pigliucci & Kaplan, 2000). My goal is to describe a research program for analysing a specific type of such constraint, one that can be estimated and dissected empirically. To do so, one must draw insight into maladaptation from each of the four perspectives above (Stearns, 1984).

Causes of maladaptation

The ultimate causes of maladaptation are aspects of genetic systems in relation to changes in environments. These include such processes as mutation, drift, inbreeding, selection, pleiotropy, linkage disequilibrium, heterozygote advantage and gene flow. Most mutations are maladaptive or non-adaptive because their effects are independent of adaptive significance and traits of organisms are normally reasonably well adapted

(Orr, 1998). Maladaptation may also be caused by a lack of sufficient genotypic variation for phenotypes to maximally respond to selection. Such paucity of variation may be due to drift, inbreeding, past directional selection, or a low rate of mutation. For example, drift and inbreeding remove populations from genotypic adaptive peaks and thus may lead to phenotypic maladaptation. Although years of artificial and strong selection experiments on single traits attest to high levels of variation maintained in the short term for most traits and species, the relevance of these experiments to nature remains uncertain (Harshman & Hoffmann 2000). This is in part because natural and artificial selection may often differ profoundly in their targets, strength and consequences. Pleiotropy, whereby genes affect multiple traits, is considered a near-universal mode of gene action and it may facilitate maladaptation by impeding the joint optimization of several traits (Barton, 1990). Linkage disequilibrium, due to linkage, drift, or selection, similarly leads to more-or-less severe limitations of genetic effects on phenotypes. Heterozygote advantage provides a third example of intrinsic properties of genetic systems that cause deviations from maximal population adaptation. Finally, gene flow between differently adapted populations may also lead to maladaptation, the degree of which will depend on migration rates and selection intensities (Slatkin, 1985).

Quantitative-genetic methods allow estimation of both additive genetic variances, which probe the expression of genetic variation in a given environment and also genetic correlations, which are due to pleiotropy and linkage disequilibrium. A low level of additive genetic variation, or a substantial genetic correlation, may indicate the possibility of maladaptation with regard to the traits involved (Price & Langen, 1992). Aspects of developmental mechanisms (a.k.a. 'developmental constraints') can be depicted by matrices of genetic variances and covariances (Cheverud, 1984), although **G** is always environment-specific and may not capture essential elements of life-history or other tradeoffs (Clark, 1987; Houle, 1991; Partridge & Sibly, 1991). In our adaptive-peak depiction of maladaptive evolution, mutation moves populations downhill and a lack of variation strands them on a slope, or slows their climb. Drift and inbreeding normally drive populations downhill. Genetic correlations may accelerate uphill movement or cause curved trajectories (Arnold, 1992), and gene flow between multiple local adaptive peaks pulls populations down towards some centre of gravity (see also Fear & Price, 1998). Selection, however, defines the landscape on which populations move, and to the extent that environments and selective pressures change our landscape becomes a sea where waves rise, sink and move like water in a bath. If populations are normally on or near a crest, movements will tend to displace them downward – Fisher's (1958) constant deterioration of the environment, due to both abiotic and biotic causes. The degree of maladaptation of populations thus depends on rates of change in selective surfaces, in relation to rates of genetic and phenotypic change (Kirkpatrick, 1996). But how can one empirically capture this complex scene and put a maladaptationist research program into practice?

Methods to identify and analyse maladaptation

Our maladaptationist research program has two main components: (1) recognition and quantification of putative maladaptation; and (2) determination of maladaptation's causes. As described below, putative maladaptation can be recognized using information from phylogenetics, developmental processes, teleonomy and optimality theory and genetics. Deviations from adaptation can then be quantified directly using studies of selection and selection response, or quantified indirectly via optimality studies, each of which can provide information about directions and distances from adaptive peaks. Finally, to demonstrate maladaptation and exclude adaptive alternative hypotheses, it is essential to determine its causes using information from population genetics, quantitative genetics, developmental mechanisms, or other approaches.

Methods from teleonomics and optimality, phylogenetics, genetics, and development play different roles in the analysis of maladaptation. Teleonomic and optimality approaches usually make quantitative predictions regarding phenotypes and these predictions usually more or less succeed and more or less fail. When faced with deviation from optimality predictions we can either hold tight to our optimality paradigm, inspect the nature of the deviations and question our strategy set, constraints, and fitness criterion, or we can allow for the possibility that our phenotype has not been optimized (Orzack & Sober, 1994). A strategy set can, however, always be expanded to include formerly 'maladaptive' phenotypes, which in effect negates the possibility of maladaptation and equates natural selection with adaptation (Rose *et al*., 1987). The main usefulness of teleonomy and optimality approaches is firstly, that they can generate the trade-off curves that describe the relationships between complexly integrated traits and fitness, which no other approach, including quantitative genetics, can do (Partridge & Sibly, 1991). Secondly, cycles of observation, modelling and experiments can identify the causes and objects of selection and their adaptive (environmental) significance. Deviations from optimality predictions may then direct us towards maladaptation. The main objection of many researchers to optimality is that this latter road is not taken; genetically based causes of deviation from optimality are seldom considered, as the space between prediction and observation can always be filled with ad hoc explanations. This is a valid complaint but it does not vitiate the tremendous successes of teleonomy and optimality methods.

Phylogenetic approaches to analysing adaptation provide a vital long-term temporal dimension to data via analysis of convergence between traits and between traits and aspects of their environments, or also via analysis of evolutionary trajectories along specific lineages. Convergence analyses (e.g. independent contrasts, see Doughty, 1996) entail macroevolutionary tests for functional relations and strong deviations from expected relationships, or invariance in traits in particular clades (Stearns, 1984), may be indicative of maladaptation. Inference of evolutionary trajectories may be used to identify apparent temporal lags between the appearance of a selective pressure and evolutionary response

Table 1 Examples of apparent and putative maladaptations. 'Apparent causes' include both ultimate and proximate (genetic) factors

Trait	Description	Apparent cause	Reference
Large fruits, poorly dispersed	Large fruits are not dispersed, rot under parent	Large mammalian dispersers went extinct	Janzen & Martin (1982)
Wood duck egg dumping frequency	Wood ducks engage in extremely high levels of egg dumping when using obvious nest boxes	Novel environment (nest sites much less hidden)	Reeve & Sherman (1993)
Fig wasps sex ratios	Wasp species under weaker sex-ratio selection show larger deviations from optimality	Weak selection	Herre (1987)
Spider behaviour	Spiders in riparian zone exhibit nonoptimal behaviour	Gene flow from surrounding desert	Riechert (1993)
Lack of paternal care	Duck species exhibit lack of male care where it is expected	Lag in response to selection?	Johnston *et al.* (1999)
Tit clutch sizes	Clutch sizes are maladapted to local habitat	Gene flow	Dhondt *et al.* (1990)
Transference	Females exhibit aspects of male secondary sexual traits	High positive genetic correlation between sexes	Muma & Weatherhead (1989)
Vestigial traits	Nonfunctional traits, present in functional form in ancestors	High genetic correlations?	Fong *et al.* (1995)
Lack of disease resistance	Selection for disease resistance but no response	High levels of selfing prevent independent evolution of traits	Parker (1991)
Gall form	Some species exhibit gall form of recent ancestors, and mismatch between gall form and life history	Lag in response to selection?	Crespi & Worobey (1998)
Thorax morphology	Large thoraces in wingless males that developed in environment where winged males developed	High genetic correlation?	Crespi (1988)
Toes in dogs	Large breeds of dogs often have an extra toe	Genetic correlation?	Alberch & Gale (1985)

in traits (e.g. Crespi & Worobey, 1998; Johnston *et al.*, 1999) (Table 1). Phylogenies can also be used to infer evolutionary change in the aspects of genetic systems that can produce maladaptation. For example, phylogenetic approaches can help determine if, and how, **G** changes in the short and long-term (Shaw *et al.*, 1995). They can also help determine whether any observed changes are due to selection, drift, or both, if drift causes only proportional

changes in **G**, and to what extent the short and long-term evolution of species is directed by the main axis of multitrait variation (Schluter, 1996). Phylogenies are thus primarily useful in that they may direct us towards putative maladaptations, which are then dissected using other means.

Data on genetics and phenotypic selection are necessary for the analysis of maladaptation under our chosen definition of the term. Whereas teleonomic and optimality approaches help to identify the most selectively relevant aspects of traits and environments (i.e. the causes and objects of selection) and characterize the direction and extent of deviation from predicted optima, quantification of selection and anticipated or observed response to selection are required for an explicitly adaptive-peak perspective on trait evolution. Path-analytic methods allow for the joint analysis of the causes and objects of selection (Crespi, 1990), and multiple regression methods (Lande & Arnold, 1983) and methods for visualizing adaptive surfaces (Schluter & Nychka, 1994) can be used most effectively once the objects and causes of selection are reasonably well understood. Quantitative-genetic analysis of **G** then permits inference of population trajectories on adaptive surfaces and assessment of if, and how, aspects of genetic architecture prevent, delay, or facilitate approach to local peaks (e.g. Björklund, 1996), and study of if, and how, peaks move over time. One of the most important outcomes of such analyses will be whether or not **G** itself can be considered adaptative (Thornhill, 1990; Schluter, 1996); has it been shaped by a history of correlational selection, or does it reflect inexorable, intrinsic relations between traits? Ultimately, we will need to connect **G** with the genes that lead to adaptation (Clark, 1987; Orr & Coyne, 1992). Genes of larger effect may be more likely to exhibit maladaptive pleiotropy and may be more likely to be fixed by selection, at least during the early stages of approach to an optimum (Orr, 1998).

Although quantitative-genetic methods are tremendously powerful for microevolutionary inference, **G** only more or less indirectly reflects the developmental mechanisms that translate between genotypes and phenotypes (Houle, 1991). Empirical analysis and modelling of developmental processes can identify potential maladaptation by demonstrating that aspects of ontogeny that are recalcitrant to modification by selection lead to maladaptive phenotypes (e.g. Slatkin, 1987). Understanding the developmental mechanisms of maladaptation is important because it allows for exclusion of alternative explanations of observed apparent maladaptation, such as failure to identify the correct trait or selective context. For example, multiple lineages of vertebrates have gained and lost digits in concert with the evolution of larger and smaller body sizes (Alberch & Gale, 1985; Alberch, 1985). Is this pattern due to intrinsic inability of developmental systems to produce certain numbers of digits from a certain amount of limb bud tissue (i.e., a lack of appropriate variation), or might fewer digits be adaptive in some unknown way for smaller species? Does 'transference' of secondary sexual characteristics from males to females (Lande, 1987; Muma & Weatherhead, 1989) result from highly conserved hormonal effects on development, or might such traits be selected for in females? Some aspects of allometry appear to be modifiable by short-term selection, but others do not (Wilkinson, 1993; Emlen, 1996). Analysis of developmental mechanisms should allow us to assess if these latter traits are intrinsically resistant to change; alternatively, selection may not be acting upon them at all.

Using our conception of maladaptation, we can generate a list of cases of putatively maladaptive traits (Table 1), that have been subjected to analysis by various of the four approaches described above. The list is short and a surprising proportion of the studies have been published in top journals. Some studies have put two of the approaches together (e.g. Alberch & Gale, 1985) but none have used three or four, perhaps because of the independence of researchers using genetic, developmental, optimality, or phylogenetic perspectives and tools (Lewontin, 1979). A combination of all the approaches allows connection of microevolution with macroevolution, in the context of a trait's genetic architecture and functional significance, or lack thereof. Whereas the analysis of adaptation benefits from integration of multiple disciplines, the study of maladaptation demands it. Such a research program is challenging, but without it we will never thoroughly understand the causes of phenotypic variation.

Conclusion

To the extent that evolution is defined in terms of changes in gene frequencies and phenotypes, and to the extent that 'natural selection is not evolution' (Fisher, 1958; p. 1), maladaptation can evolve. However, I believe that the integrative research needed to analyse it has largely been prevented by the rhetoric of Gould & Lewontin (1979), chaotic definitions of constraint (Antonovics & van Tienderen, 1991), denigrations of optimality (Rose *et al*., 1987), the contrast between a genetic focus on variation and an ecological focus on optimal states, and the contention of some behavioural ecologists that they need seldom inspect Pandora's black box of genetic mechanisms (e.g. Thornhill, 1990). Many authors have called for synthetic studies that combine phylogeny, teleonomy, development, and genetics in the analysis of adaptation and constraint, however defined (e.g. Stearns, 1984; Arnold, 1992; Pigliucci & Kaplan, 2000). I have attempted here to sketch out a research program based on their hopes. An understanding of how and why both adaptation and maladaptation evolve, and how they are linked to population dynamics, is increasingly important as humans rapidly transform the globe into an environment where maladaptation is expected to be more prevalent for all species, including ourselves.

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