On fitness

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Abstract. The concept of fitness, central to population genetics and to the synthetic theory of evolution, is discussed. After a historical introduction on the origin of this concept, the current meaning of it in population genetics is examined: a cause of the selective process and its quantification. Several difficulties arise for its exact definition. Three adequacy criteria for such a definition are formulated. It is shown that it is impossible to formulate an adequate definition of fitness respecting these criteria. The propensity definition of fitness is presented and rejected. Finally it is argued that fitness is a conceptual device, a useful tool, only for descriptive purposes of selective processes, changing from case to case, and thus devoid of any substantial physical counterpart. Any attempt to its reification is an apport to the metaphysical load evolutionary theory has inherited from Natural Theology.

Introduction

Fitness is a concept considered of prime importance in population genetics and demography and thus central to the synthetic theory of evolution. Despite its apparently easy understanding, at least *prima facie*, this concept faces several non trivial difficulties. After a brief discussion of the origin of the concept I will offer some adequacy conditions for the definition of fitness in population genetics. I will then formulate the best possible provisional definition and subsequently show how it fails the meet the conditions of adequacy. This is done by two means: a systematic analysis of the constraints imposed by these criteria and also by offering counterexamples. Delayed gene action, for the first time discussed in this respect, renders impossible a correct definition of fitness. The impossibility of a correct definition is further elaborated by considering the difficulties faced when overlapping generations are considered or when the population size increases or decreases. Inclusive fitness is not devoid of these difficulties. Furthermore I will challenge a dispositional account of fitness by unraveling its metaphysical underpinnings. Finally I will support the view that *fitness* is a useful device but devoid of any

general physical counterpart, exactly as the concept of adaptation: it is only a descriptive tool for the study of natural selection.

From origin of the concept of fitness in population genetics. From Darwin through the demographers to Wright

Although Darwin barely ever used the word *fitness* in "The Origin of Species",[1] he clearly had the concept of it. He took it to capture a property of an individual, viz., a physical property of the organism accommodating to its way of living, and thus a cause which explains the success of individuals subjected to the process of natural selection. Fitness might also carry robustness as a nuance, exactly as in ordinary language. Darwin inherited this concept from Natural Theology, and employed *fit* and *fitness* as synonymous to *adapt* and *adapted*, a fitting between the organism and its environment. Even today, Brandon (1990) equates fitness with adaptedness. Herbert Spencer made this term more popular using the unfortunate expression "survival of the fittest" as a synonym of natural selection and, following him, Darwin repeated it.[2]

Population demographers have used *fitness* to express in mathematical formalism the kinematics of population growth. Malthusian fitness is the ratio λ of the number of individuals of one generation (N_{t+1}) to that of its parental generation (N_t). For a detailed exposition of the term in population demographers (Malthus, Verhulst, Lotka) see Gillois (1996) and Krimbas (2000). In this approach all individuals of a population may be considered identical, and thus fitness concept refers to a common individual property no less than it characterizes the entire population.

Fisher was the first to introduce the concept of fitness in population genetics. He used for this purpose mortality and fertility tables (inherited from Lotka). He estimated the value of fitness from that of the Malthusian parameter, m, the difference between the percentage b of the individuals that give birth to one individual during a time interval Δt and the percentage d of those dying at the same time period, that is b–d. The relation between m and λ, the Malthusian fitness, that is according to him the ratio of the number of individuals of one generation (N_{t+1}) to that of its parental generation (N_t), is

$$\lambda = e^m.$$

Furthermore, Fisher considered that the Malthusian parameters, and thus fitnesses, are inherited: different genotypes have different fitnesses. According to him, the course of evolution is to maximize fitness: in an early account he noted that "the fitness of any organism at any time" is maximized

and the rate of increase in fitness is equal to its [additive] genetic variance in fitness at that time. But in a later account he noted that "the average fitness of a population" was to be maximized with a rate equal to the genetic variance of fitness in that population.[3] Since variances are always positive, the change will always be in the direction of an increase in fitness. R. Fisher considered this *fundamental theorem of natural selection* to be a general law, equivalent to the second law of thermodynamics, which stipulates always an increase of a physical quantity, entropy. The generality of Fisher's law was questioned and, in some cases, the law was shown not to hold true.[4]

The problems with Fisherian fitness is that the entire approach, although suited for asexually reproducing organisms, fails if not precautions are taken, to account to complication arising from the Mendelian segregation in sexually reproducing organisms. If the ratio of a certain genotype in two successive generation f_{t+1}/f_t is equated to its fitness, Mendelian segregation is disregarded. As Maynard Smith (1991) remarked in the case of sickle-cell anemia the homozygotes aa for the sickling allele have no offspring, but the number of aa individuals in the next generation arise from the matings between two heterozygotes Aa. Should these heterozygotes increase in frequency (due to the prevalence of malaria, to which they are resistant), aa should also increase in frequency: instead of having a zero fitness, it will be allowed a positive value, greater than one. Otherwise in equilibrium condition it should be equal to one. Thus "the genotype's per capita rate of increase" is not the way population geneticists employ to measure fitness. Another qualm may be found in the way this procedure departs from the intention of the original use of the term "fitness" by population geneticists, i.e., as a mean to provide a cause for the selective process. Since the estimation of fitness is performed a posteriori, that is after the occurrence of selection and no the basis of the selective results, it might be taken that causes and effects are confounded. It could be argued that this critique may also be true regarding Wrightian fitnesses, to be considered below. However, the practice of estimating Wrightian fitnesses by the enumeration of the number of offspring produced together with the interposition of the segregation mechanism renders this confounding effect undirect, if present at all.[5]

S. Wright (1969, but the work goes back to the 30's) used the population fitness as varying according to the gene frequencies in the population. Excluding competition among individuals, S. Wright states that every genotype is characterized by a fitness value, so that each individual belonging to that genotype has an expected number of progeny which is the fitness of that genotype. The population fitness $\overline{W}$ is the expected mean number of progeny of every individual of the parental population. $\overline{W}$ is a composite function: the

sum total of the products of all genotype frequencies by their specific fitnesses (or adaptive values).[6]

Adequacy criteria for a definition of fitness

We will first offer three criteria for a satisfactory definition of fitness. Then we will attempt to formulate a definition complying with these criteria, following the tradition of population genetics and regarding the simple case of non over-lapping generations. It will become obvious subsequently that satisfactory definition is impossible.

A satisfactory definition of fitness should comply to the three following criteria:

(a) It should be formulated in such a way that the results of chance events, affecting the progeny output, should be avoided in fitness estimations. Since the life history of an individual organism may be influenced by chance events, his reproductive output could greatly differ from that of another similar individual because of these chance events. Thus the fitness definition should be restricted to a life history devoid of chance events. Better it should be restricted to the mean of life histories of individuals that may, or may not, face all possible chance events with their natural frequency of occurrence. This drive us either to adopt as a measure of fitness the expected number of children or their mean number in an adequate sample of individuals of the same constitution. According to this criterion Brandon's (1990) suggestion to consider fitness of individual organisms has to be excluded from consideration.

(b) Fitness should stem from an internal property of the organism belonging to a certain class of individuals which have the same constitution (geno-type, theirs mother genotype, or both). This constitution should lead to a phenotypic trait, structural, functional or behavioural. Fitness should be related to and result from this trait and therefore explain why individuals belonging to this class leave such a number of offspring or why their genes are represented in such a number in the next generation. Thus it should provide an explanation for the outcome of the selective process.

(c) The number of offspring or genes these individuals leave should be counted during their entire life history, ideally starting at the beginning of it and ending at their death. However, in cases of maternal influence a different starting and end point may be selected. Inspite of this, in the case of a two gene genotype, we should not use different time periods for each to estimate their fitness, but the same time period for their combined effect. Ideally we should use the same time period for all possible kinds

of constitution. We will see that this criterion is difficult or impossible to meet.

An attempt to define fitness according to the tradition of population genetics. The case of non overlapping generations

Population geneticists regard "fitness" in its technical meaning as a relative or absolute measure of reproductive efficiency or reproductive success. Peter Medawar (1960: 160) expressed the following view regarding fitness, which is apparently adopted by many population geneticists:

> The genetical usage of "fitness" is an extreme attenuation of the ordinary usage: it is, in effect, a system of pricing the endowments of organisms in the currency of offspring; i.e., in terms of net reproductive performance. It is a genetic valuation of goods, *not a statement about their nature or quality* [my emphasis].

To comply with the first criterion we may at first consider as a measure of fitness of a certain genotype the mean number of offspring it produces. The only permissible way to perform this counting is to start counting the zygotes produced by individuals of this genotype from the beginning of its life as a zygote until its death. Counting from zygote to zygote may be a difficult or problematic operation, since in different animals internal fertilization may render these observations impossible. To bypass this difficulty, it was suggested to transport the counting period starting the counting at a convenient age after fertilization and ending it at the same age in the next generation. This is a mistaken suggestion since it excludes a part of life history of a genotype from consideration but instead it includes the corresponding period of possible different genotypes, since Mendelian segregation does not insure that offspring share the same genotype with their parent (Actually, this is also the difficulty in using Fisher's "fitnesses" or Malthusian parameters in sexually reproducing species).

Even so, the mean number of offspring, when estimated, may not be sufficient: the *distribution* of this number may be of importance. Thus Gillespie (1973, 1977) has shown that genotypes which have the same number of progeny but differ in variances have a different evolutionary fate. Everything else being equal, an increase in variance (temporarily, that is from generation to generation) is disadvantageous in the long run. From the actual mean of expected number of progeny (that is among generations), a quantity should be subtracted equal to $1/s^2$ for the case of temporal variation (where s^2 is the variance in offspring number). In the case of developmental variation (e.g., when one proportion of individuals of a genotype produces a different number

of offspring, while the arithmetic mean for all of them remains the same when compared to another genotype which has the same mean number of offspring but all individuals produce exactly the same number of offspring) Gillespie (1974, 1975, 1977) has shown that the genotype with the increased variance is disfavored. In this case again, a quantity equal to $1/Ns^2$ should be subtracted from the arithmetic mean in order to arrive at an estimate of fitness (where N is the population size). The same is true for spatial variation.

The reason for all this, as Sober (2001) has insightfully explained, is that the expected value of a quotient is not identical to the quotient of expected values. This is the cause for the discrepancy observed in the next generation between those genotypes of which all individuals produce exactly the same number of offspring and those of another genotype of which individuals produce different numbers but overall display the same mean number.

In order to cover this complication we may restrict the definition of fitness to one only generation (and thus avoid temporal variation). Furthermore, we may restrict the fitness definition to a defined (homogeneous) environment [and thus avoid complications as those described by Brandon (1990: 90), where a genotype having two different fitnesses in two environments, both at absolute value, which are lower than the respective two fitnesses of a different genotype, may end up having a higher fitness due to an unequal distribution of individuals in these two environments]. Taking these into consideration we may try to formulate the following provisional definition of fitness:

> In diploid organisms the absolute or Darwinian fitness of a certain genetic constitution of individuals of the same species in a population of non-overlapping generations living in a defined [homogeneous] environment is equated to the mean number [or the expected number] of zygotes produced by a zygote of this constitution during its entire lifetime, whereas relative fitness will be the measure of the productive efficiency of a certain genotype, as defined above, compared to that of another from the same population.

As explained above, this definition does not cover the case of variance difference in the same generation at the same environment. This is not the only difficulty encountered.

Let us now see what is needed for compliance to the second criterion of adequacy.

Even if we make the correct counting from zygote to zygote, we face another difficulty which emerges from the delayed effects of the parent genotypes on the fitness of the offspring, effects independent of the offspring genotype, but dependent on the genotype of the parent. The delay may involve one or more generations to come. Although these effects seem to affect the fitness of the offspring, or of grandchildren, or even of more remote descend-

ants, in fact finally these traits refer to and affect the fitness value of the parent genotype, which is exclusively responsible for these delayed effects. In this category are included maternal genes expressed (via messenger RNA) in the early embryo [polar genes, genes determining dorsoventral axis, like b-catenin in frogs (O. Pourquie 2001), anterioposterior and lateral (left to right) axes, *macho-1* gene determining myoplasm in ascidia, responsible for the development of some muscles (Nishida and Sawada 2001)], as well as the maternal genes in strains of *Caenorhabditis elegans* mutants of *mortal germline*, that is deficient telomerase mutants.

Thus the *grandchildless* mutant of *Drosophila subobscura* (Spurway 1948; Suley 1953) as well as sililar polar mutants discovered later in *Drosophila melanogaster* (Niki and Okada 1981; Mariol 1981; Thierry-Mieg 1982) have such effects: female homozygotes for the mutant allele produce sterile offspring (regardless of the genotype of the male parent or that of the offspring). The reason for this is that in their fertilized eggs the posterior polar cells (from which gametes are derived) are not formed: there is no cell or nuclear migration for the production of these cells, and the maternal genotype (not that of the embryo) is exclusively responsible for it. Genes affecting the direction of cell coiling, dextral or sinistral, in gasteropod mollusks (of the genus *Albinaria* or of *Limnaea*, see Sturtevant and Beadle 1940: 330) belong to the same category. The phenotype of the individual is not determined by is own genotype but reflects the genotype of its mother (not necessarily from its mother's phenotype, which is determined by its grandmother genotype). In these cases offspring counting for fitness estimation should be delayed by one generation: grandchildren are to be counted. Actually, the entire cycle of observation and counting is transposed by one generation: the fitness of a homozygous for the *grandchildless* mother [the fitness of a female of the genotype for this gene] is estimated by counting the number of grandchildren produced by a fertilized egg of this mother.

Milk factor is another delayed character in horses. A foal may sometimes die because its blood cells react with antibodies produced by its mother and contained in its mother's milk. Antibodies are produced after repeated gestations when a foal inherits from the sire the ability to make a particular antigen absent in the dam. Then, counting from zygote to zygote, or from fertilized egg to fertilized egg to fertilized egg, does not provide an accurate estimation of fitness (Srb and Owen 1952: 262–263). This case is similar to that of Rhesus blood group types in men, the only difference being that in this case the delayed response is manifested at the birth of the offspring.

An earlier appearance of the lethal effect in mice is observed when the mother lacks a heat shock protein (Hsfl: heat protein factor 1). The Embryos die after fertilization, at the stage of one or two cells (Christians et al. 2000).

There are several instances of delayed action one should take into account in this respect, including the silencing of parental genes in the embryo in mammals, either paternal or maternal, by methylation: the genomic imprinting (Reik and Walter 2000). Another case of maternal effect combined with the paternal phenotype is encountered in zebra finches: females deposit a varying amount of testosterone in eggs according to the attractiveness of the males they mate with, the greater the attractiveness the greater the amount deposited. Testosterone plays an important role on the fitness of the offspring (Birkhead et al. 2000). An egg to egg count misses this effect which is independent of the offspring's genotype, but depends on the parental male phenotype (and eventually genotype). However, this case may be relegated to the section discussing the fecundity component of fitness (see below) with the addition of the presence of a delayed effect.

The problem is further complicated when the generation delay involves not just one but many generations. Complete sterility may be produced by *mortal germline* mutants in some strains of the round worm *Caenorhabditis elegans* after four or even after sixteen generations, varying with the mutant (Ahmed and Hodgkin 2000). These mutants exhibit progressive chromosome telomere shortening and accumulate end-to-end chromosome fusions in later generations leading to complete sterility. Here the counting of progeny should be delayed by several generations, varying according to the strain until complete sterility is achieved.

These delayed effects present non trivial difficulties when the third criterion of adequacy has to be met. The fitness of most of the genes may be computed by counting from zygote to zygote, while those with delayed effect may be computed in a different time schedule including, eventually, one or more generations. Thus, for a composite genotype, two different fitness evaluations should be considered in contradiction to the third criterion.

Using a quantitative genetic model Wolf and Wade (2001) have investigated the effects of assigning fitness components of offspring to parent fitnesses. A maternal behavioural character that contributes to children survival could be assigned either to offspring fitness or to the fitness of the mother. Provided that it is not assigned to both of them (thus being counted twice, an obviously incorrect practice), it seems that the assignment to either of them is not free of potential shortcomings. Assigning these components of offspring fitness to the offspring avoids potential problems but may miss a component of kin selection provided by the mother, not detected in selection analyses. Thus it goes against the second criterion. On the contrary, the assignment to parent's fitness (e.g., the effect on early survival due to maternal care) of components of fitness of the offspring (when there is a genetic correlation between the parental effect increasing offspring's fitness and the direct effect on offspring fitness) may lead to incorrect dynamic equations and

eventually to incorrect conclusions regarding the direction of evolution. But this assignment is in accordance to the second criterion: it justifies the aim of population geneticists to provide an explanatory version of the selective process.

It is obvious that in defining fitness we do not want (following the third criterion) to consider a longer or even an extremely long evolutionary fate, as did J.M. Thoday (1953, 1958) and W.S. Cooper (1984), but a short one, of one generation. *The afore mentioned cases of delayed gene action affecting the next or even more remote generations, mostly or rather completely neglected in theoretical discussions until now, present a non trivial complication rendering impossible a satisfactory definition of fitness.*

Gametic selection and meiotic drive

Thus far we have only considered the fitness of diploid individuals. Selection processes may apply as well to the haploid phase of the life cycle, the gametic one. Although not differing in their mathematical treatment, two different forms are distinguished: the meiotic drive, that is the production of gametes in non-Mendelian proportions by a heterozygote, and simple gametic selection, that is gametic differential viability or differential capacity to fertilize or be fertilized (for a review see Birkhead and Moller 1998). Cases of meiotic drive have been described in mice (the *t* alleles, Lewontin and Dunn 1960; Young 1967; Silver 1985) and in *Drosophila* species (SD system in *D. melanogaster*, Hartl and Hiraizumi 1976; Hartl 1980; Brittnacher and Ganetsky 1984; Temin and Marthas 1984; Sex-ratio system in *D. affinis, D. subobscura, D. pseudoobscura*, for a review see Krimbas and Powell 1992, and in *D. simulans* Merçot et al. 1995). Several of these systems are complex, composed of a set of genes, enhancers, inhibitors, etc.

To model these systems one may use coefficients similar to those used when dealing with the diploid phase of organisms, that is coefficients similar to fitness. When selection operates both at the haploid and the diploid phase, a sequential combination of algorithms permits to deal with them. The same could be applied in case on wishes to deal separately with selection operating at different components of fitness (as we shall see below).

Components of fitness of non overlapping generations

It is often stated that selection acts on survival and reproduction (Brandon 1990: 15). This is not an exact formulation. Fitness is the mean number of progeny left; therefore viability components (survival, longevity) are important as far as they affect the net reproductive effect. Longevity may be

important in those cases where it may affect the net reproductive effect. Selection is blind to longevity at a post-reproductive age (including the component of parental care to offspring). This is the reason why inherited pathological syndrome of Huntington's chorea appearing after the reproductive age seems not to be selected against. On the other hand, there are cases of selection against longevity, e.g., the adoption of a life strategy that promotes early sexual maturation. As Reznick, Bryga and Endler (1990) have shown, when a predator is present, some fishes are selected for this regime, contrary to what happens in absence of predators (establishment of a life strategy for a later sexual maturation).

According to D.L. Hartl (1989), starting from the stage of the zygote, the components of fitness are the following: *Viability*; subsequently *Sexual selection* operates favoring or prohibiting a genotype to find mates (for a review see Andersson 1994); in the general case every combination of genotypes of the mating pair may correspond to a specific *Fecundity*. Thus, *Fecundity* depends on the genetic constitution of both parents. For a simple one gene two alleles case nine different Fecundity values are defined. It seems more difficult to reconcile the factual data of this case with the presence of an individual's dispositional capacity or property postulated by the propensity theory of fitness. It seems more plausible to consider interaction as a better explanation, fecundity not being attached to the genotype but to the combination of the genotypes of the two mating partners.

Before the formation of the zygote, gametic selection (one aspect of which is meiotic drive) may take place, and sometimes counteract the direction of selection exercised at the diploid phase. Gametic selection was discussed above in a separate section.

There are cases of fecundity selection that may be improperly considered as gametic selection, because selection operates at the gametic stage. According to the "male function hypothesis" in hermaphrodite angiosperms, once a certain number of female seeds is secured by hermaphrodite flowers, it pays more to invest exclusively in male flowers. The production of male flowers requires less energetic expenses than hermaphrodite, while it permits the dissemination of the genetic constitution of the plant to a wider range by fertilizing stigmata of receptive carpels of flowers of different individuals (Burd and Callahan 2000). The capacity to produce male flowers is partly genetical and characterizes the diploid individual.

Sperm displacement in Diptera is another relevant example. In a second mating, the sperm of the last male displaces to a certain extent the sperm of the previous mate. There are several devices to prevent this as well as counter measures to these devices. The capacity of displacement is a phenotypic character of the male, not of the sperm.

Components of fitness-overlapping generations

Developmental time is an important, but generally neglected or ignored, component of fitness in populations of overlapping generations at the phase of increase of their size (e.g., at the beginning of colonization of a new umoccupied territory; r-selection, see Mac Arthur and Wilson 1967).

R.C. Lewontin (1965) examined the case of insects that follow a triangular schedule of oviposition. A triangular egg productivity function is characterized by three points, apexes of the triangle, i.e., the age of first production, the age of peak production, and the age of last production, all these being stated in a time coordinate while the number of eggs produced per time unit is stated at the other coordinate. In his specific model, a reduction of developmental time may be equivalent to a doubling of total net production: The reduction is equal to an 1.55-day decrease of the entire egg production schedule (what Lewontin calls a transposition of the triangle to an earlier age), or to a 2.20-day decrease only of the age of sexual maturity (the age of the first egg produced), leaving the other ages as well as the total number of eggs deposited unchanged. It is also equivalent to a 5.55-day decrease of only the age of the highest egg production (the peak of the triangle) other things remaining unchanged or, finally, to a 21.00-day decrease of the age at which the last egg is deposited, other variables remaining the same.

However, when the population does not increase but remains stable (K-selection) or shrinks, the genotype that produces earlier in its life-cycle the same number of offspring is the one that is selected against, in comparison to a genotype producing the same number of offspring later in its life-cycle. The situation is inversed when the population increases. This is another case of fitness not being a property of a genotype, but better understood as a result of an interaction under the prevailing conditions. In this respect the work of Brian Charlesworth and J.T. Giesel (1972) should be consulted.

Other capacities may play an important role when the population saturates the environment. In K-selection it is not fecundity but a better exploitation of environmental resources that is favoured by selection (Mac Arthur and Wilson 1967).

Indirect selection schemes – inclusive fitness

W. Hamilton's (1964) concept of *inclusive fitness* was formulated to provide a Darwinian explanation for altruistic actions that endanger the life of the individual performing such acts. An individual may multiply its genes in two different ways, directly by its progeny, and indirectly by protecting the life of other individuals of a similar genetic constitution. If the danger encountered is

overbalanced by the gain (as this is calculated in genes), then the performance of such acts may be fixed by natural selection. Not only do inclusive fitness estimations take into account the individual's fitness, but also they take into account the fitness of its relatives (of similar genetic constitution). Inclusive fitness is the sum total of two selective processes, individual selection and kin selection. In fact, in this case the counting tends to change from the number of individuals in the progeny to the number of genes preserved by altruistic acts in addition to those transmitted directly throughout the individual's progeny. Of course this way the traditional definition of fitness, already provided, is rendered inadequate. To save it one should incorporate into the fitnesses of genotypes all favorable results they are recipients, which originate from altruistic behavior of their relatives, and subtract all costs deriving from their own altruistic behavior. In this way, one may reconvert inclusive fitness estimations to usual fitness ones. It is an apparently feasible operation [and may be recommended in the spirit of Wolf and Wade's (2001) results, although they do not address directly to this case]. However, acting this way, a significant part of the natural history, regarding altruistic behavior, is obscured, lost or, at least, rendered less obvious.

A digression on the disputed concept of adaptation

Natural selection acts on phenotypes: certain traits of these phenotypes are the targets of selection. The individuals bearing some traits are said to be adapted. However, no common and general property may characterize adaptation. A search in the writings of all important neodarwinists reveals that, inspite of the suggestion that "adaptation" has an independent meaning, it is in fact used as an alternative to "selection". L. Van Valen (1976) seems to differ from all other authors, because he equates adaptation with the maximization of energy appropriation, both for multiplying and for increasing the biomass, solving thus the problem of lianas and other clone organisms. This is a recurring problem, when organisms combine a mixture of vegetative and sexual reproduction. Counting offspring may then be a difficult task. Are we to consider as separate individuals those of a colony produced by vegetative reproduction? And how can we distinguish between an enormous individual and that of several independent, freshly separated by departing this enormous individual? Should we count unseparated individuals as one and separated as many? Isn't this arbitrary? Although the solution Van Valen provided is consistent, it has not been generally adopted. Anyway, it significantly departs from the classical notion of fitness.

The concept of adaptation was shown to be completely dependent on the concept of selection (Krimbas 1984). Brandon (1978) provided an argument

proving the impossibility to establish a criterion or trait for adaptation, which is independent of selection. He argued that we may be able to select in the laboratory against any character but one: fitness. There is no reason to exclude from natural selection the selection experiments performed in the laboratory, since the lab is also part of nature. Thus there is no character or trait in the diploid organism that could be taken in advance as an indication of adaptation independently of selection. Despite this demonstration, Brandon was reluctant to abandon the concept of adaptation; however, Krimbas (1984) has proposed to do so. According to him the "description of evolutionary phenomena based entirely on terms of kinematics would be more adequate", where by "kinematics" he means the kinematics of reproductive agents during selective processes. Fitness is a variable substantiating and quantifying the selective process.

While one would expect that "adaptation" would disappear from the evolutionary vocabulary, it is still used to describe the selective process changing or establishing a phenotypic trait as well as the established by selection trait itself. Sometimes the engineering approach is used: adaptation, it is argued, is in every case, the optimal solution to an environmental problem. The difficulty with such an approach is twofold (Gould and Lewontin 1979). First, we are often unable to define precisely the problem the organism faces (e.g., it might be a composite problem) in order to determine in advance the optimal solution and, as a result, we tend to adapt the "solution" encountered in nature to the problem the organism faces. That is we "construct" the problem the organism encounters. Second, it is evident that several selection outcomes are not necessarily the optimal solutions, the evolutionary change resembling more a process of tinkering rather than an application of an engineering design (sensu Jacob 1977).

Adaptedness and the propensity interpretation of fitness

Recently, several authors (Brandon 1978; Mills and Beatty 1979; Brandon and Beatty 1983; Sober 1984; Brandon 1990) have supported the *propensity interpretation of fitness* (which they equate to *adaptedness*). In doing so they first try to disentangle *individual fitness* (something we are not considering here; for as mentioned earlier, we take into consideration only the fitnesses of a certain category or group of individuals), from the fitness that is expected from the individual's genetic constitution. Indeed, accidents of all sorts may nullify its contribution to the next generation. But selection is a systematic process in the sense that in similar situations similar outcomes are expected. Thus, in order for these authors to pass from the individual or actual fitness to the expected one, they are obliged to consider two different interpretations of

"probability". The first, *the limiting-frequency interpretation*, is to consider probability as the limit of a relative frequency of an event in a finite series of trials; but sine this series is never achieved, they might instead use the observed frequency in a finite series of trials. The second interpretation is *the propensity interpretation*. According to this, the very constitution, the physical properties of the individual, underlie the propensity for performing in a given way. This performance may rely on a dispositional property, a property displayed in certain way in some situations, and differently in others.

The dispositional properties, according to a well known view, derive from the physical structure of the reproducing individual, at that or even at a lower (e.g., molecular) level, depending on more basic (categorical) but non dispositional properties. Thus, according to the propensity interpretation of fitness, the reproductive capacity of a genotype derives from its inherent (categorical), non dispositional physical properties. This interpretation attributes fitness to physical causes, linked to the very structure of the individual, and thus attribute to them the tendency for the individual to produce a specific number of offspring in a particular selective environment. This is another way of reifying fitness, and, via fitness, relative adaptedness, and finally adaptation. It reminds us of the Aristotelian *potentia et actu* [dynamei kai energeia, $\delta \upsilon \nu \acute{\alpha} \mu \epsilon \iota \ \kappa \alpha \acute{\iota} \ \epsilon \nu \epsilon \rho \gamma \epsilon \acute{\iota} \alpha$], where the propensity is "potentia" and the actual mean number of offspring corresponds to the "actu".

In some situations of viability selection this interpretation seems quite satisfactory (e.g., in mice resistant to warfarin). No-one would deny that selection depends most of the time on the properties of a genotype which performs in a certain environment. But this may not be as general as one may think. There is an array of cases from situations like the aforementioned resistance to warfarin, which agree with the propensity interpretation of fitness, to those that gradually depart from it to the point of being alien to it. In these situations the contribution to fitness from the part of the organism is not clear or does not seem preponderant.

Consider, first, frequency dependent selection: a genotype being in advantage when rare but in disadvantage when common. Should we attribute its fitness to an inherent property? Is it not natural to describe the situation in terms of interaction between the individuals of this genotype and individuals of the remaining genotypes in the population (especially if a causal interpretation is looked for)? In this interaction, since the two interacting parts contribute equally to the outcome, they are of equal importance and status. Consider a key and a lock. They may fit and from their interaction the lock may be locked or unlocked. Describing the locking or unlocking as dependant exclusively (or mainly) on the key, we are betraying reality. Using so-called dispositional properties is an elegant way to neglect on half of the story,

focusing on one of the two partners. In a similar way it is more difficult and much less satisfactory to attribute to a certain genetic constitution the mating advantage of the males when they are rare and their mating disadvantage when common.

A more extreme case regards the fecundity which characterizes the two members of a mating pair. These fecundities depend generally on the male partner as well as on the female one. In a simple case they characterize the combination of the two mating genotypes. An extreme example is that of the change of selective advantage of a genotype highly productive in offspring when the population is expanding and when it is shrinking.

To ascribe dispositional properties to entities is to downgrade interactions and to render natural history a preformed, preconditioned tale narrated in advance. Its explanatory power is severely restricted. It reminds us of the explanation provided in *Le malade imaginaire* of Molière for the dormitive effect of a plant extract: it is attributed to the dormitive principle it contains!

The best solution is to consider genotypic fitness[7] as a usefull device to perform some kinematic studies regarding changes in gene frequencies or searching for an equilibrium point to which is the population attracted to. It seems useless to attribute other qualities, properties, or a substantial role to this device. It is useless to reify it. Its function is to permit the quantitative description of changes or the establishment of an equilibrium point in any one of the multifarious instances of selection. Modern evolutionary theory is basically of historical nature (although some processes may be repeated). A complete and satisfactory explanation of a specific case should comprise a historical narrative including information on the phenotypic trait, which is the target of selection, the (ecological, natural history or other) reason driving the selective process (why this trait is being selected), the genetics of the trait, the subsequent to selection change of the genetic structure of the population, the corresponding to it change in the phenotypes. In natural history, generality and search for hidden and non-existing entities and properties may only contribute to an increase of the metaphysical baggage of evolutionary theory, an unwelcome baggage inherited from Natural Theology.

Conclusion

Fitness is a device to figure out the kinematics of gene frequency changes due to selection in mendelian populations. Otherwise it is devoid of any meaning or substance. A recent attempt to reify it, in the propensity interpretation of fitness, is found in several cases inadequate (such as the cases of frequency dependent selection and of fertility selection). This attempt of reification is related also to the effort to provide for adaptation an independent meaning

from that of selection; I have argued already that adaptation is a concept without independent meaning from that of the selective process.

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Notes

[1] "Fitness" first appears in "The Origin of Species" first edition 1859, on page 472: "Nor ought we to marvel if all the contrivances in nature be not, as far as we can judge, absolutely perfect; and if some of them be abhorrent to our idea of fitness".

[2] Herbert Spencer used this expression in (1864) page 311. There is an interesting exchange of views on this subject among Darwin, A.R. Wallace and Spencer referred to in Spencer 1898 note in page 530.

[3] See for this R. Michod 1999, page 59. Also regarding the fundamental theorem see the discussion at Michod 1999 pages 57–62, which includes Price's analysis.

[4] Pollak (1978) has shown that if fitness is based only on fecundity (depending on the genotypes of the two mating partners) then there is no equivalent to the fundamental theorem. Fitness, e.g., mean fecundity, may decrease under selection.

J.F. Crow and M. Kimura (1970: 209) have also noted that "One interpretation of the theorem is to say that it measure the rate of increase in fitness that would occur if the gene frequency changes took place, but nothing else changed". Thus environmental deterioration that would affect fitness values, and thus decrease mean population fitness, is not considered by Fisher.

[5] Michod 1999 follows Fisher's line regarding fitness. As he himself admits "he does object to view fitness as a cause of anything, thus he cannot be charged of confusing causes and effects. For him the kinematics of gene frequency change involves both causes and effects of organism traits in the context of genetic system and environment". Michod also (p. 50) defines fitness as the expected number of gametes produced by individuals of given genotype. He apparently means the number of gametes that contribute to the formation of zygotes.

[6] Unlike Fisher, Sewall Wright, in his shifting balance theory, envisages most of the species to consist of many small, more or less isolated populations, each having its specific gene frequencies. Populations occupy the peaks of an Adaptive Surface, formed by the values of W (population fitnesses), for every point corresponding to certain gene frequencies. These peaks are positions of stable equilibria. Due to drift, gene frequencies may change and, hence, populations may cross a valley of the adaptive surface and be attracted by another peak. Equilibrium points are local highest points of population fitness values.

[7] It is much more difficult to deal with population fitness. Population geneticists calculate the mean individual fitness in a population. But this exercise is quite futile when we compare two different populations. A group of adapted organisms is not necessarily an adapted group of organisms. Demographers, earlier, equated size (or increase in size) with population fitness. However, as Lewontin once remarked, it is not sure that a greater or denser population is better

adapted, since it may call for parasites and epidemics; on the other hand a population deplete of individuals may suffer collapse and extinction. I have argued (Krimbas 1984) that according to the *Red Queen Hypothesis* of L. Van Valen (1973), all populations (at least of the same species) seem to have, a priori, the same probability of extinction, and thus posses, a priori, the same long term population fitness. In addition it is not clear how we should consider a group: a group is not an organism, which survives and reproduces. Although individuals of the group interact in complex ways and thus provide some image of cohesion, the "individuality" of the groups seems most of the time to be quite a loose matter. Should we consider group extinction per unit of time to determine group fitness? What about group multiplication? In order to achieve a modelization in various attempts to model group selection cases, one may resort to different population selective coefficients, or population adaptive coefficients (something related to the population fitness). In these cases the search for the nature of population fitness becomes even more elusive. As a result population fitness is a parameter useful exclusively for its expediency; no search for its hidden nature is justified.

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