Heritability

and The Units of Selection

A Defense of Genic Selectionism

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I Introduction

For some years now there has been a dispute running among biologists and philosophers of biology concerning the unit(s) of natural selection. It is a complex issue that involves many intewoven and sometimes unclear lines of argument. Several authors have tried to confine the matter to a generalized discussion of the conditions that a unit of selection should meet, leaving open the empirical issue of which biological entities actually manage to fulfill the requirements. Such a general discussion would in be based on the idea that a unit of selection can be found at whatever level of organization one can sensibly ascribe heritable variation in fitness between competing entities; this includes such dissimilar entities as genes, genotypes, traits, organisms, groups, species, etc. There is, however, in concepts such as fitness a sea of confusion that allows the interpretational disagreements one finds. General (definitional) discussions of what the unit of selection is leave on the same footing disputes that are of dissimilar nature, they mistakenly assume that genes' causal role in evolutionary processes is to be judged by the same criteria as the roles played by traits and entities of other organizational levels.

A recent widespread position has been the so called "hierarchical" one, which proposes that causal responsibility of natural selection can in principle be attributed to any of the mentioned entities, leaving to empirical adequacy (or convention) the decision of which one is to be chosen as explanatory in a given selection process. It will be an aim of this essay to show that genic selectionism is not a proper participant in this dispute, as several authors take it to be. It is not the role of the exclusive bearer of fitness, or of the properties that are sources of fitness, that this position claims for genes. Genic selectionism is rather a claim about the causal (probabilistic) substratum that permit selective processes to have evolutionary significance. It refers basically to the fact that at whatever level of organization we might find the properties that natural selection pressures are picking out, evolution will require the simultaneous sorting of *units* with both causal influence over those properties and hereditary powers (i.e. genes or, more generally, replicators). Furthermore, the position holds that the latter will always be one fundamental component of the two part process. Richard Dawkins for instance was pointing to a real distinction, albeit in his strange wording, when he wrote that

> the conventional dispute between group selection and individual selection is different in category from the apparent dispute between individual selection and gene selection . It is wrong to think of the three as arranged on a single dimensional ladder, such that words as above and below have transitive meaning. I shall show that the well-aired dispute between group and individual is concerned with what I shall call "vehicle selection" and can be regarded as a factual biological dispute about units of natural selection. The attack "from below" on the other hand is really an argument about what we ought to *mean* when we talk about a unit of natural selection(Dawkins,1982,p.82).

Vehicle selection, or as Hull prefers, "interactor" selection is meant to refer to what intuitively one would choose as the entity that, in a selection process, bears the property that makes the difference for survival and/or reproduction. The acceleration capacity of an organism, the average temperature of a group, the extinction rate of a family. In Elliot Sober's terms, it would be related to the immediate property responsible for (the source of) the fitness of all correlated biological units under the given selection pressure. Genic selectionism need not dispute such immediate causal role of different level properties, which can be synthetically described by the versatility of a concept like fitness. Genic selectionism, as Dawkins has insisted, is about a parallel process of "replicator selection". And there is a sense in which this kind of selection has explanatory primacy for evolutionary transformations, as it is in the end the responsible for the persistence of favourable trait-varieties. The amazing power of accumulation that natural selection has is due to the existence of hereditary factors that are in various degrees responsible for biological traits or properties. That a given particularity of a trait can re-appear in the descendants of the bearer is due to the possession by these of similar genetic information. That differential survival and replication of genetic elements play a specially significant role in evolution is beyond doubt, the point genic selectionists try to establish is that such elements have a causal or active role in their destiny, and not the passive one that some authors see, and that some of the terms used to refer to it (like "genetic reaction") tend to suggest. Richard Dawkins has been particularly concerned with the defense of such causal claim.

To expose the specific sense in which one can affirm that genes (or genetic information) are responsible for their fate in the gene pools of evolving populations he devised the term "active germ-line replicator", with its clear

Weissmanian implications. And it is precisely such causal and explanatory claim that has recently been challenged by several authors, among which Elliot Sober is prominent. Under the banner of a hierarchical view of natural selection, these authors argue against the granting of a special status to genes in the causal workings of evolutionary processes. My intention in this essay is to defend genic selectionism mainly against some of Sober's arguments. My main line of defense is based on the necessity of what is referred to by biologists as *heritability* (of traits, of fitness) for evolutionary change. It is heritability that gives substance to genic selectionists' causal claims, and it is the overarching necessity of heritability for evolution (whatever the "level" of property selection) that justifies the different explanatory status of genic selectionism. It is the genes' capacity of behaving as units of populational causal influence in the long run that genic selectionism stresses. Such capacity is due to their unique position at the vertex of the interaction and the replication causal chains. If vehicle (trait) selection is understood as interdependent with the sorting of genes (replicators), the coordination of the two causal processes has to be properly understood. Heritability is the synthetic term biologists use to refer to such coordination. Evolution occurs when selected *for* vehicle traits are consistently (i.e. causally) correlated with specific alleles. This fact is in the end what makes the causal contribution of both processes possible. Genes'(replicators') causal role in enhancing its kind's probabilities of survival and reproduction is, I will conclude, a precondition of evolution. Given that they are, so to speak, the ultimate bearers (*units*) of heritability, they have a probabilistic causal role to play in all evolutionary processes. There is, I think, no denial of this open to Darwinists.

II Replicators and Vehicles

Most biologists would nowadays agree that a good shorthand formula that describes evolution is "the change in frequencies of alleles in a population's gene pool". Another formulation widely accepted is that of the conditions for evolution to be produced by natural selection: that there be "differential reproduction of heritable fitness variation". As it happens, the causal appeal that distinguishes driven "deterministic" selection processes from purely statistical error sampling (drift) relies on an appeal to such a troublesome concept as fitness differences. Some authors have tried to avoid the problem by making use of statistical concepts (as "nonrandom" or "consistent") to qualify natural selection processes, and recently Hodge(1987) emphatically defended the

use of "nonfortuitous" as the proper qualification for differential reproduction of heritable variation due to natural selection, because it stresses the causal character of the difference. The analysis of what the causal appeal in this case amounts to is central for the understanding of how explanations of evolution through natural selection are ultimately justified. Parallel to the so called "logic" of inferences based on natural selection, there is the task of giving a satisfactory general account of its causal workings. Much of the recent outburst of interest in the philosophical analysis of evolutionary theory has focused on this area, either on explications of concepts like fitness or adaptation, or on the question of at what levels and *for* what units natural selection exercises its sorting powers[1].(1)

The nonfortuitous differential reproduction of heritable variation determines frequencies of alleles in gene pools. This means that the survival and reproductive advantages that certain trait-varieties(or properties) provide to their bearers over their competitors, with alternative trait-varieties, are, in a sense, advantages of the genes (alleles) correlated with such trait-varieties against their allelic rivals. This correlation is what is meant by *heritability* of the trait-variety as a condition for its long term causal efficacy under natural selection. A given gene or group of genes should thus be able to re-produce more often if its associated trait(s) is/are favoured by selection. Many authors have found convenient for the analysis of evolutionary processes the separate consideration of the two correlated elements (genes and traits) and some have even insisted that *both* roles, the re-productive (associated with long term permanence and conservation of characters) and the selective (associated with events of differential survival and reproduction in every instance) can be played by different kinds of entities at different levels of biological organization and in different selection processes.

With this distinction, the causal question linked to natural selection becomes a dual one: What sorting interaction is able to discriminate between trait or property differences in such a way as to cause differential survival and/or reproduction of the bearers? and, as the second is sometimes worded, for whose benefit is the selection process working, or who is getting its kind through towards a bigger share in the next generations' biological lodgings? A derived question is, of course, which causal source is to be given priority for explanatory functions?

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[1] I emphasize *for* because crucially it can be taken to mean both the entity which benefits (evolutionarily) from the adaptations produced and the one that bears the traits or properties that are picked out by natural selection. This will have a close relation with Elliot Sober's use of the distinction between selection *for* properties and selection *of* objects.

This division is the base of Richard Dawkins' distinction between replicator selection and vehicle selection (Dawkins,1976) and was modified by David Hull with the idea not to prejudge the question of causal primacy [as the term vehicle seems to do]. Hull prefers the descriptive connotations of replicators and *interactors*.(Hull,1981). According to Hull's distinction these two causal functions can be described as a) the reproduction or transmission of structural similarity (replicator) and b) the production of differential replication. Hull's worries about Dawkins attributions to replicators are symptomatic of the passive role concerning the causality of natural selection to which several philosophers have tried to confine the gene, urged mostly by biologists like R.Lewontin and S.J.Gould, who are, I believe, at least in part motivated by the social and political consequences of coarse genetical determinism. Hull wants, so to speak, to isolate the causal reach of genes. He writes:

> Dawkins has one vice...he tends to run two quite distinct functions together into one, replication and the interaction of entities with environments.
> (Hull, 1981)

Hull believes that a Dawkins paragraph that shows this "confusion" is the following:

> We may define a *replicator* as any entity in the universe which interacts with its world, including other replicators, in such a way that copies of itself are made. A corollary of the definition is that at least some of these copies, in their turn, serve as replicators, so that a replicator is, at least potentially, an ancestor of an indefinitely long line of identical descendant replicators. In practice no replication process is infallible, and defects in a replicator will tend to be passed on to descendants. If a replicator exerts some power over the world, such that its nature influences the survival of itself and its copies, natural selection, and hence progressive evolution, may occur through differential survival.(Dawkins,1978,p.67)

Hull is worried here about the claim that genes always interact causally with their environment, in an evolutionarily significant way. He wants to restrict this function to interactors, and genes are only interactors in limited intracellular events. Dawkins however has in mind a specific kind of causal

interaction in the above paragragh: the one exerted by the replicator on the interactor, and which is epitomized by the relation between genotype and phenotype; interactors depend causally on replicators, and that causal thrust is transitively communicated to the sortings in natural selection events. Besides, it seems clear that Dawkins has in mind only genes (or genetic materials) as replicators in "life as we know it", and that he chooses to use a general term in case of there being other types of replicators in other kinds of life. Hull however takes that generality in another direction, and gives a case for the possibility of other biological entities (as we know them) acting as replicators in evolutionary processes. The aim would be to have a general scheme in which optional units (genes, genotypes, organisms, groups, etc.) could be incorporated according to the peculiarities of the case. This kind of generality appeals to many philosophers but fails to do justice to the claims of genic selectionists (or for that matter, replicator selectionists) in the sense that the two types of processes should not be confused. Their causal workings *are* different and the hierarchical stance
is not equally viable in both. For Dawkins a central tenet is the point that replicators *have* a double causal function: they re-produce their structural traits *and* they affect the characteristics of those interactors that are causally linked with them. Such difficult duality is a major obstacle for any hierarchical pluralism in the replicator "side" of natural selection processes. And, in any case, the capacity of the replicator to re-produce its (type's) structure is just as important for the long run (permanence-linked) processes as the capacity to actively influence its (type's) chances of doing so through causal links with selective (nonfortuitous) events. Dawkins thus wants to make a distinction between active an passive replicators.

> An *active replicator* is any replicator whose nature has some influence over its probability of being copied. For example a DNA molecule, via protein synthesis, exerts phenotypic effects which influence whether it is copied: this is what natural selection is all about. (Dawkins,1982,p.83)

Hull however wants to loosen some of Dawkins' criteria for replicators. He claims that neither great fidelity (or identity) nor directness of replication should be asked for. He correctly points out that a criteria inspired by G.C. Williams would do a better job, this is that two genes are similar enough to count as the same replicator if they react similarly to similar selection pressures.(Hull,1981,p.150). But the wording here again fails to emphasize the

gene's causal responsibility and the verb *react* suggests for them a rather passive role. Nevertheless the criteria is clarifying because it favours the causal or functional over the structural similarity. On the other hand Hull wants to leave open the possibility of indirect replication of structure (and with less than perfect fidelity), so traits, organisms or groups could count as replicators. What Hull fails to consider is that besides the reappearance at some point in the species life cycle of similar structures (or properties) linked with the previous ones by descent (reappearance which can occur differentially with or without natural selection), there must exist a stable link (manifested through a correlation) between genetic information and whatever candidate one chooses for the role of replicator (trait, individual, or group) if there is going to be some sort of evolutionarily significant (selectable) similarity. Thus, if we want to consider an extra genetic entity as replicator we shall have the problem of justifying such correlation in terms that do not duplicate the description in a nonsense way: replicators type 1 (genes) that produce replicators type 2 (traits, organisms or groups).

For Hull it is enough for a replicator to be causally linked in some way with copies, however faithful, of itself. He leaves the significant causal role in relation to natural selection basically to interactors:

> Replication by itself is sufficient for evolution of sorts, but not evolution through natural selection. In addition, certain entities must interact causally with their environment in such a way as to bias their distribution in later generations... the characteristics of a good replicator are sufficiently different from those of a good interactor that eventually these functions began to be performed by different entities at different levels of organization. (Hull,1981,p.150)

What Hull forgets here is that it was only the interactor role that became specialized in that particular way, i.e. by climbing the ladder of complexity and finding new levels of organization. The replicator function or, in other words, the conservation of information role, specialized without trascending the molecular level. Therefore it is only the interactive function that can be *descriptively* stratified in evolutionary biology.

Active replicators must be causally linked with their own permanence in the evolutionary competition, and although they themselves do not have to bear the interactive properties or traits, they must somehow be linked to them. A

probabilistic and populational causal explication has been proposed for this relation, and this is, as we shall emphasize, basic for genic selectionism. Given genes are believed to be causally responsible for heritability of given traits (thus they are correlated with them) and heritability is a necessary condition for evolution through natural selection. Hull's analysis, as many others of the anti-gene selectionist wave, seems to be based on disconnecting this link, i.e. on obscuring the fact that the base for the correlation between genes and trait-varieties (or interactor's properties) is *causal* and not in any sense spurious and, furthermore, that the direction of such causality is from genetic information to trait variety, and not the contrary.

The use by Hull of Dawkins' term "replicator" (without the balancing effect of "active") is thus unfortunate because it fails to stress this point, which is crucial to the genic selectionist view. As we shall see later, the claim that such correlations bear no causal importance in evolution, absurd as it seems and is, has been defended emphatically by several authors[2](2).

Replicator selection and vehicle selection are then clearly two different but interdependent causal processes. The former, which is directly linked with permanence, is sometimes thought as the domain where one should look for \Ithe\i unit of natural selection, while the latter is said to be related to \Ithe\i "level" of selection (Brandon,1985). These processes can however be well understood without such definitional queries. Perhaps the clarification of the causal roles and relations between the different descriptive levels in evolutionary biology can be better attained by avoiding their confusions. There need not be \Ia\i unit or \Ia\i level of selection, but differing causal roles in the complex (two-fold) process of evolution by natural selection.

III Gene-trait Correlations

There is, I think, a misunderstanding of what the genic selectionist causal claims are. It stems from the tendency to fuse the discussions concerning replicator and interactor selection into a monolithic search for *the* unit of selection. Hull makes that evident when he writes about the poetic justice in that Dawkins... has taken the arguments which organism selectionists have used against group selection and turned them on the organism selectionists

[2] Lewontin, Sober, Wimsatt, and Brandon amongst them. Rosenberg has argued, that a mistake at the root of this position is that they confuse the use of fitness or selection coefficients for genes in models with causal claims:"Neither genic nor genotypic selection co-efficients are causes of fitness or selection, they are effects of fitness differences."(Rosenberg,1983,p.333)

themselves (Hull,1981,p.147). The reference here is to the arguments based on parsimony and simplicity of description that both G.C. Williams and Dawkins use to favour lower levels of selection against higher ones. The basic structure of this strategy is to devise an alternative, simpler, explanation based on properties or traits borne by a lower level entity, to undermine a given claim that some trait needs a selection process focused on a higher level trait. But such discussions affect only vehicle selection. As I understand these arguments they could be rephrased by saying that it is easier to imagine how a (caused) correlation between genes and, say, organismic traits can be strong enough to produce evolution, than it is to conceive a similarly effective (caused) correlation between genes and traits of a higher level of organization. In the end, the parsimony arguments are not about what we have been calling the replicator role. Unfortunately, Elliot Sober shows the same confusion as Hull when he takes his acute criticism on parsimony based arguments as damaging to genic selectionism in general (Sober,1984a,p.234). It must be said, that both G.C. Williams and R.Dawkins tend to mix their appeals to parsimony with their more basic claim (that genes are always causally active in evolution) in the same confusing manner. This was pointed out by David Papineau, who advanced the distinction I am focusing on. When reviewing Dawkins' book *The Extended Phenotype* he wrote:

> Much of the confusion surrounding the notion of "the selfish gene" is due to Dawkins originally having run two quite different ideas together. On the one hand there is the basic Weismannian principle that natural selection is at bottom always a matter of a certain gene producing some effect that brings it about that there is an increased proportion of like genes in the next generation. And on the other hand there is the modern ethological resistance to group selectionists accounts of animal behaviour. But these two ideas have very little to do with each other. There is nothing in Weismannianism as such to rule out group selection. The Weismannian principle that natural selection always involves a gene increasing[3](3) its representation in the next generation leaves open the question of *how* the gene manages this.(Papineau,1984.p.799)

[3] A small modification will make more precise Papineau's statement, before "natural selection" he should have written "evolution by".

Weismannianism is the real source of genic selectionism and makes a quite different causal claim than that which some arguments around the levels of selection are centered upon. By discovering the causal split between germplasm and soma, Weismann made possible a major clarification of the workings of evolutionary processes: The privileged situation of the genetic elements in what could be described as a causal vertex*, where on the one hand they act and are acted upon by their environment to produce somatic elements (properties or traits), and on the other hand the information they carry is differentially copied and passed on to future generations. Both are statistical processes where causation can only be understood in a probabilistic fashion, and where genes (or *bits* of causal information) function as causal factors.

But genetic elements play different causal (and thus explanatory) roles in the somatic and in the germ-line sequences. On the one hand, that the germ-line (long run) sequence depends for its causal efficacy on the statistical outcomes of individual (short run) somatic sequences seems undeniable. On the other hand, somatic (physical) interactions (possibly at different levels) are causally responsible for differential survival and reproduction of biological entities, and they are in a sense the source of fitness differences referred to by abstract models. But the latter does not provide the other basic causal element of evolutionary processes, heritability, which links the germ-line sequence with somatic events. The fact that certain genetic elements tend in given sets of environments to favour the occurrence of certain trait-varieties gives them a major causal position in any evolutionary process. This was of course clearly perceived by the theoreticians that developed population genetics. The misunderstanding of what this stance of genic selectionists amounts to, has made possible, I believe, several recent mis-aimed critiques that tend to talk about genically based descriptions of evolution as acausal "bookeeping"[4](4) based in some sort of spurious correlations between genes and traits. The most thorough and enlightening analysis, which nevertheless succumbs to this error, is Elliot Sober's recent book *The Nature of Selection*. In a considerable measure the rest of this essay will be an attempt to undermine Sober's arguments and conclusions. I begin then by quoting two revealing assertions:

> the correlation of genetic characteristics with
> organismic or group phenotypes in these [selective]
> processes is what is represented in the different

[4] This strongly suggestive expression is due to William Wimsatt(1980):"remarks suggesting genetic reductionism are better seen as having more import as a kind of genetic bookkeeping than as a promising reductionistic theory of evolutionary change in terms of genes frequencies" (p.154)

> fitness values assigned to single genes. The existence of such correlation is an absolute criterion for evolution by natural selection, no matter what the unit of selection is. But correlation is no more sure sign of causation, any more than *selection of* is no sure sign of *selection for* (Sober,1984a,p.284)

> Selection for or against the phenotype may cause the frequency of the gene to change but this will be due to the *correlation* between the gene and the actual phenotype. There will be selection *of* the gene, but not selection *for* it.(Sober,1984a,p.313)

Selection *for* and selection *of* stand in Sober's works stand for a distinction between causal and acausal processes. Based on the common dictum that correlation does not imply causation, he stresses the point that while a selective process can be based on sortings between kinds of objects at different organizational levels (selection of), it usually is the case that the efficient cause (selection for) of the sorting is a property (or properties) that might or might not be at the same level. This means that many other properties "carried" by the objects or entities are selected just by the sheer fact of being "correlated" with them, without having any causal connection with the selective process, and thus no explanatory thrust[5].(5)

Sober elaborates this distinction in the realm of statistical events where he equates population-level (or probabilistic) causation with what he calls property causation. Such view of causation is at the bottom of Sober's argument against genic selection as a separate and pervasive element of evolutionary processes. One of my aims will be to show its inapplicability for actual (historical) natural selection processes[6](6).

Both of the above quotes point towards the undeniable possibility of there being selection *of* genes without selection *for* them, but Sober is too quick in his generalization. There are two evident weaknesses in his assertions. First there is a clear and dangerous similarity between what he calls property selection (which he loads with causal powers) and what above was referred to as vehicle or interactor selection; and second the underlying assumption that the correlation

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[5] An illustration used by Sober to clarify his distinction is a children's toy with small and big marbles that are sorted by holes through which only the small ones can pass. If all the small ones happen to be also all the green ones, then there would have been selection *of* green marbles and simultaneously selection *for* small marbles.

[6] In contrast with what would be ideal or generalized population causation in which infinite populations or unlimited trials would be relevant.

between genes and selected *for* properties or traits has no causal base[7](7), thus implying either that heritability is some kind of accident or that evolution can proceed based on a magically consistent (i.e. acausal) association between genes and traits[8](8). The following quotes exemplify, I believe, both weaknesses:

> In all cases of genic selection, a genic property is a positive causal factor in survival and reproduction. Cases of genic selectionism may differ, however, with respect to what the *objects* are that these genic properties attach to. Genic selection may impinge upon organisms, upon chromosomes, upon gametes, and upon genes themselves.(Sober,1984a, p.310)

Evolutionary theory now deploys a striking hierarchy of possible selection mechanisms. Indeed it is a double hierarchy -of both objects and properties- that contemporary theory has had to consider. Objects at different levels may be selected; and there may be selection for and against properties at different levels as well. The adequacy of the various hypotheses made available by this hierarchy is subject of continuing empirical controversy, which philosophical reflection cannot resolve. (Sober,1984a,p.368)

Leaving aside the conditions that Sober imposes on "positive causal factors", which will be dealt with later, I want to suggest a simple terminological shift which I consider revealing with respect to Sober's abuse of the versatility of "property" as a concept. If in the first quote one reads "genic effect" where it says "genic property" (and what else can a genic property that is attached to an organism or a gamete be?) the matter would, I believe, suddenly become transparent. And then one would be back to Dawkins' distinction between active replicators (genes) and vehicles (that carry "genic effects”), and furthermore it would seem unavoidable that, for all evolutionarily significant events of natural selection, at least some "genic effects" would have to be "positive causal factor(s) in survival and reproduction". Organisms, chromosomes, gametes and genes themselves appear thus as the vehicles (interactors) whose traits or properties are, at least partially, caused by "correlated" genes. With the elimination of the obscure property talk, the whole mystery of the above pluralistic second paragraph is thus dissipated; the mentioned double hierarchy becomes one (a vehicle hierarchy) which is connected to genic selection. Genes

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[7] As there is no causal link between the greenness of the marbles and their having been sorted out, in the toy described in note 4.

[8] Of course, naturally selective interactions can happen without there being a consistent genetic base (i.e. without heritability), and thus without evolutionary consequences in the population. Random drift on the other hand can build on heritability to produce fortuitous changes.

causal role seems thus clear enough due to the awareness that although other objects might indeed be spuriously (acausally) correlated with any given selected *for* trait or property, genes are usually causally linked with them, at least in the evolutionarily significant cases. Genic selectionism mantains thus its special status as *the* explication of active replicator selection in our kind of life. Furthermore, it will by now seem evident that the search for *the* explication of what a unit of selection is in evolutionarily significant natural selection events is futile. Two complementary processes (replicator and vehicle selection) admit at least two kinds of "unitary" elements. And the former of those will always be of genic nature.

IV Genes and Probabilistic Causation

Genic selectionism, Sober (1984a) argues, has been faced by two standard and related criticisms: context-dependence and indirectness of genes' effects. The former builds on the instability of such effects from context to context. As Ernst Mayr famously wrote:

> No gene has a fixed selective value; the same gene may confer high fitness on one genetic background and be virtually lethal on another (Mayr,1963,p.296)

Some time later the indirectness criticism was aptly worded by S.J. Gould:

> Selection simply cannot see genes and pick among them directly. It must use bodies as intermediary. A gene is a bit of DNA hidden within a cell. Selection views bodies.(Gould,1980a,p.90)

In their simple forms these objections can be said to be a product of traditional uncritical views, amongst biologists, both of causation, and of the relation between genotypes, phenotypes and selective events. To both these objections the facts of epistasis and pleiotropy are central, i.e. that genes can have many-one, one-many and many-many, causal relations with phenotypic traits. The ghost they are aiming at is that of "reductionistic bean-bag genetics" where only one-one relations are considered. But on the whole such worries have more to do with the problems of theory reduction in functional (synchronic) biological disciplines but not with evolutionary (diachronic) ones[9] (9). David Hull (1974) has brilliantly analyzed the

[9] Ernst Mayr makes this distinction clear in his characterization of "functional" vs "evolutionary" disciplines.(Mayr,1976)

conceptual problems that arise from taking at face value the oversimplification of gene-phenotype links in classical mendelian models, and its consequence to the idea of theory reduction in genetics. But this discussion has no bearing on the position of genic selectionism[10] (10) whose base is the probabilistic and statistical nature of causal links within evolutionary theorizing, and which is thus in no way tied to oversimple mendelian models.

Kitcher and Sterelny (1987) agree with Sober that neither context-dependence nor indirectness objections, in their simple formulations, seem to damage the standard, statistical formulations of gene selectionism, which is not committed to "beanbag-genetics"[11](11). Perhaps the clearest statement of the latter is due to G.C. Williams:

> Obviously it is unrealistic to believe that a gene actually exists in its own world with no complications other than abstract selection coefficients and mutation rates. The unity of the genotype and the functional subordination of the individual genes to each other and to their surroundings would seem, at first sight, to invalidate the one-locus model of natural selection. Actually these considerations do not bear on the basic postulates of the theory. No matter how functionally dependent a gene may be, and no matter how complicated its interactions with other genes and environmental factors, it must always be true that a given gene substitution will have an arithmetic mean effect on fitness in any population. One allele can always be regarded as having a certain selection coefficient relative to another at the same time. (Williams,1966,pp.56-57)

Sober has, perhaps involuntarily, wasted too much paper to leave it clear that the main claim of genic selectionism has little to do with the "parsimony" or "representability" arguments he deals with extensively (Sober,1984a,pp.234-249)[12] (12). It is the causal role of genes (as probabilistic causal factors)

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[10] Although it might have damaging consequences for those who defend non-genetical entities as possible replicators, because of the difficulty of the re-appearance, in a similar enough fashion, of traits (or structures) in the next (linked) generation.

[11] "advocates of genic selectionism have a very different picture of the causal relation of genotype, phenotype, and natural selection, one which, if true refutes both these objections" writes Sober (1984a,p.228).

[12] But Sober cannot resist the temptation to write fifteen pages demolishing a shadow. Parsimony as used by Williams and Dawkins to undermine group selectionists' claims is a pragmatical strategy that cannot be conclusive. Furthermore, it does not bear on what we have been calling active-replicator selection but on vehicle or property selection. That is

and thus the explanatory value of their presence or absence in given populations and environments, that is the central issue. And that is why it becomes crucial to understand Williams' "arithmetic mean effect on fitness in any population" not as meaning that the gene will possess a fixed selection coefficient, nor that the affected "fitness" should be attributable to it or to any other entities (organism, group). It simply means that according to its effects on the different physical surroundings in which it is likely to appear, the gene (as type and as active-replicator) will have a certain general survival probability that can affect its future frequencies in the species' gene-pool. The particular kind of effects on which different genes' chances are based can be as varied as different selective biological interactions which can exist[13](13). Such a picture of genes' causal influence on their survival and reproduction makes them appear not as causes of individual selective events[14] (14) but, again, as probabilistic causal factors in populations of sometimes quite intricate selection processes. This immediately raises the problems of how to explicate probabilistic causation (in general) and of how such explication can help us (or not) in an analysis of genic selectionism. These were Elliot Sober's basic concerns in the positive part of *The Nature of Selection*. Basically, he takes both the context-dependence and the directness objections and transforms them, through his account of probabilistic causation, into much stronger and damaging arguments against genic selectionism.

Under Sober's account, as sometimes genes can have opposing effects on their bearer's fitnesses in changed genetical and physical environments they cannot (but seldom) count as positive causal factors in evolving populations. On the other hand, as causation in its probabilistic or populational mode, is not neccesarily transitive, the biologist's intuitive "directness" objection gains strength and respectability.

V Context-dependence

For a gene to be a positive causal factor (p.c.f.) in a given selective process it would have to favor(increase the probability of) a positive outcome in at least one context and should not disfavor it (reduce its probability) in any. This is what Sober calls the Pareto-style requirement and what according to his

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because it centrally deals with the issue of which entity bears the property that serves as base for the sorting action. The issue of representability is a lateral one in causal discussions.

[13] Of course, this is precisely the rationale for the replicator-vehicle distinction.

[14] Something like Sober's property selection, or for that matter Dawkins' vehicle selection must occupy this role.

analysis genes often fail to fulfill. Each context, following standard statistical relevance analysis[15](15), is a cell from a partition produced by considering every causally relevant background factor (except the purported p.c.f.). Williams' quotation (above) can be understood as an appeal to consider that the average effect of the gene's presence in all the pertinent cells is the way to adjudicate a fitness value to such gene. Sober's objection is to consider that this procedure discovers anything causally significant. In reference to the archetypical example of the effect of smoking in a population he writes

> there is no way to describe the causal role of smoking in the population as a whole if the direction of the probabilistic inequality is reversed by switching from context to context (Sober,1984a,p.294)

Averaging over contexts, as genic selectionists like to do, will only obscure the authentic causal structure. Although genic coefficients are sometimes readily obtained by such procedures they are not causally relevant:

> As we have seen, it is always open to the advocate of genic selection to reconstrue such processes in terms of selection coefficients that attach to single genes. The strategy of averaging over contexts is the magic wand of genic selectionism. It is a universal tool allowing *all* selection processes, regardless of their causal structure, to be represented at the level of the single gene.(Sober,1984a,p312)

A simple example of what is meant here is given by heterozygote superiority where, as Sober and Lewontin (1982) argue, the selection coefficients at the genic level are secondary to those at the genotypic (allelic pair) level. Averaging, they claim, always might foster the illusion of selection taking place at lower level. But it is at the level where one can assign context independent (in this case: frequency-independent[16](16)) fitness values where the unit of selection should be ascribed. Genotypes are Lewontin and Sober's candidates for playing that role in most selective events. An extreme case of

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[15] See for example Salmon,1984 p.158-182

[16] Frequency dependence is a special case of context dependence that is of special interest in evolutionary processes. The interesting aspect it provides is the changes in the causal workings of a causal factor when other factors of its type are part of the causally relevant background in different proportions. This is particularly important for selection processes where what is at stake is precisely the proportions of the causal factor in future populations (or contexts). Frequency dependence has been, I believe, rather confusingly used by Elliot Sober because he tends to overlook the fact that changing the frequencies of the causal factor is effectively changing the causally relevant background. We shall discuss this later.

heterozygote superiority (in which both homozygotes are lethal) helps them make their point. The genotypic fitnesses would in such case be constant: AA=0, aa=0, Aa=1; whilst the allelic fitnesses would depend on the frequencies of each allele in the population. A alleles would flourish when a alleles are abundant (as they will tend to end in a heterozygote organism) and diminish its numbers in the reverse situation, where a alleles will flourish. Selection would always lead to equilibrium where allelic frequencies would be 0.5 for both a and A. Fitness values for each allele would be 1, and thus selection coefficients would be 0. So focusing on individual alleles would give the wrong impression that no selection is taking place. Genic selection coefficients...

> are gerrymandered hodgepodges, conceptually and dynamically quite unlike the genotypic selection coefficients that go into their construction. For genic selection coefficients are defined in terms of genotypic selection coefficients *and* gene frequencies...they vary as the population changes in gene frequency, whereas the genotypic coefficients remain constant. And if their uniform zero value at equilibrium is interpreted as meaning that no selection is going on, one obtains a series of false assertions about the character of the population. (Sober & Lewontin,1982,p.230)

Another example, particularly dear to Lewontin, of similar genotypic selection processes involves genes from different loci. As different genes' effects are sometimes not independent, Lewontin writes,

> It is *only* when the fitnesses at the two loci have a multiplicative relationship that they can be treated independently (Lewontin,1974,p.279)

Here again there will be situations where genotypic fitness will depend on considering which particular alleles are occupying a place in the relevant loci, and thus any fitness value assigned to genetic entities below that level (allelic pairs, individual alleles) will be context sensitive, i.e. they will change according to the allelic frequencies in relevant loci[17].(17)

Even though we may be interested in following only one segregating entity...an understanding of evolution along that one dimension requires *first* a synthetic

[17] An example of this is given by Lewontin and White (Reported in Lewontin, 1974), it deals with the interaction of two chromosome inversions in the grasshoper *Moraba scura*. It is also described in Sober & Lewontin,1982

treatment of the genotype and *then* an abstraction of the a single system of interest from the complex mass.

We cannot reverse the process, in general, building a theory of a complex by the addition or aggregation of simple ones (Lewontin,1974,p.281) the technique of averaging may still be pressed into service. But the selection values thereby assigned to ... genotypes at a single locus will be artifacts of the fitnesses of the...genotype complexes(Sober & Lewontin,1982,p.220)

To describe the dynamics of genetic change in a population undergoing natural selection in the most causally informative(i.e. explanatory) way only single alleles' fitness value at a given time.(Lewontin,1974,ch.6) From similar considerations William Wimsatt produced his definition (ready-made for the genotype) of what a unit of selection is:

> any entity for which there is heritable *context-independent* variance in fitness among entities at that level which does not appear as heritable context-independent variance in fitness (and thus, for which the variance in fitness is *context-dependent* at any lower level of organization.(Wimsatt,1981,p.144)

In the end, it is the consistency or stability of independent genes' causal influences that is questioned by such criteria. As replicators (to use Dawkinspeak) genes fail, according to this view, to affect in a stable way the fate of the vehicles they appear in. Only collections (ensembles) of genes can do this, it is argued, and, to bring the statement close to this essay's line, heritabilty will hence be a property of genotypes, not of genes. I will show later that these arguments, and others, based on the frequency-dependence of genic coefficients do not really undermine the genic selectionist causal claim. I would now just like to point out two evident sources for my criticism. One is the conflation between the mathematical procedures that produce fitness values and the (independent) physical processes and interactions that will justify (or not) such attributions[18].(18)

Many authors, including Sober, recognize that the mere ascription of fitness or selection coefficients cannot be taken as causal explanation of the processes described. The "implications of the mathematics of selection" writes Jonathan Hodge "must be supplemented with physicalist and causalist notions" and the same will be required in "considerations of adaptation and fitness" (Hodge, 1987, p.256). The other one is the biased distinction that is repeatedly used by

[18] Rosenberg (1983) wrote against Sober and Lewontin (1982):"The assimilation of this theory [genic selectionism] to one of its potentially convenient mathematical models [population genetics] is a persistent error of this paper". p.333

Lewontin and Sober between genetic contexts (frequencies) and extragenetic ones. Genetic context-dependence should not be considered as different in a general analysis of how natural selection works. Kitcher and Sterelny have recently used an argument initially forwarded by R. Giere[19] (19) in order to show how the apparent damage made to genic selectionism by frequency dependence is dissolved when partner genes and loci are analysed as forming part of the causally relevant background factors that need to be taken into account in the statistical search of causal factors. To quote again from Jonathan Hodge:

> In specifying the environmental conditions wherein a physical property difference has causal relevance to survival and reproduction, we may have to specify what the populational proportion of variants are. But to do this is not to substitute mathematical for physical factors, for the frequency differences only make a causal difference because they have different physical consequences. (Hodge,1987,p.258)

Once again we are left with the central issue of causation. Context-dependence could not count as a criticism if genic selectionism were a claim about individual level causation. As it concerns the causal role of a *kind* (type) of gene in a population it is only when we consider replicator selection as a probabilistic and populational process that the gene, in opposition to the genotype, is clearly seen as the best candidate for the role of causal factor. Gene types must be taken to be independent causal factors. As Dawkins has pointed out, their size and limits should be understood as a compromise between their ability to copy themselves into the future (avoiding crossing overs, etc.) and their ability to causally improve their chances of doing so. The latter can be described as the ability of genes to be linked with favourable traits (in the Extended Phenotype view, where the trait need not be beared by its own vehicle), correlation which, I have insisted, is the basis of heritability. Sober is well aware that it is probabilistic causation we must deal with.

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[19] Ronald Giere (1984):"Sober is correct in pointing out that concentrating on the extreme values of r (r=0 and r=1) masks the frequency dependence of the causal factors. As before, however, we can unmask the dependence by making r an environmental parameter and applying the standard model to a negligibly small subpopulation of the original population. Thus the frequency dependence can be represented by a set of frequency-independent models indexed by the frequency r." p.391; Kitcher & Sterelny (1987) transform the above in a causal analysis:"[We shouldn't] be alarmed by the fact that the distribution of environments in which alleles are selected is itself a function of the frequency of the alleles whose selection we are following... the phenomenom is thoroughly familiar from studies of behavioral interactions"p.12 "the effect of each allele varies with context, and the contexts across which variation occurs are causally relevant" p.6

The distinction between individual-level and population-level causality makes all the difference in the world when we consider how "context dependence" affects the truth and falsity of causal claims.

> It is a truism that [individual] causes bring about their effects ...Population level causal claims are a different matter...if a factor augments some individual's chances of an effect but diminishes the chances of others the factor will not play a determinate causal role in the population as a whole. Here context dependence of a certain sort is enough to defeat the causal claim (Sober,1984a,p.297)

To refute genic selectionism under this construal of causation, as Sober writes, "it is enough to refute that all selection is selection for or against single genes". And this apparently can be done, as Lewontin has always believed, focusing on polygenic effects.

> If a gene raises the probabilty of a given phenotype in one context and lowers it in another, there is no such thing as the causal role that the gene has in general...The point is that ensembles may have determinate causal roles in selection processes even when single genes do not. (Sober,1984a,pp.313-314)

This dismissal is a consequence of the Pareto-style requirements for positive causal factors. Before going into the analysis of such view of probabilistic causation, I again want just to point out a feature that can be appreciated in the previous quotations and that will help me to weaken such a case. The fact is that any causal factor that one can think of in evolutionary biology (replicator or interactor, trait or property) will fail to have an homogeneous unidirectional causal influence in the survival and reproduction of its type and of any "correlated" biological objects[20] (20). Changing the environmental (background) factors (genetic or not) can always be a way to change the influence a causal factor has and it is hardly surprising that sometimes even the direction of causation is altered (from positive to negative, or vice versa). Biological populations are of course susceptible of being described and *partitioned* for statistical causal analysis using whatever causal background

[20] "Sober's principle [that there is selection for a property P only if in all causally relevant background consitions P has a positive effect on survival and reproduction] seems to hanker after something like the uniform association of effects with causes that deterministic accounts of causality provide...[it] cannot be satisfied without doing violence to ordinary ways of thinking about natural selection"(Kitcher & Sterelny, 1987).p.12 One of their conclusions is that no causal factor whatsoever has unidirectional effects under natural selection.

factors are viewed as relevant, but it so happens that sometimes part of the effects of causal factors that drive selective processes is to alter the causal background under which they are acting thus changing their own role. This is not exclusive of genetic elements nor, of course, is it the only way through which contexts can be altered. A specific way to deal with this situation has been devised by biologists but philosophers have only recently paid attention. It is to restrict the causal contexts under consideration to those more feasible and, crucially, to weight the causal influence such context (or environment) is to be given in the overall picture by its probability of occurrence[21](21). The Pareto-style requirement for positive causal factors (that they never lower the effect's probability) is in this manner blocked, and the overall causal influence of a given factor is reasonably ascribed explanatory thrust for the selection of the effect in some subsets of contexts.

If context-dependence is a general feature affecting any probabilistic causal factor in evolutionary biology, and if selective processes are generally affected by frequency-dependence, the mentioned arguments favouring the genotype against the gene for the replicator causal role in natural selection collapse. Context-dependence must be incorporated rather than banned from the definition of probabilistic causation in evolution.

VI Transitivity

The directness objection raised against genic selectionism received a sophisticated exposition in Robert Brandon's paper "The levels of selection". (1982) Brandon first distinguished between the search for a unit of selection, which is what matters in the context-dependence discussion, and the search for the level of selection, where the directness objection applies. According to him, the former confronts genic selectionism with an alternative (replication) unit, the gene ensemble or genotype. The latter with an alternative (interaction) level, the phenotype[22](22). Brandon uses the Reichenbach-Salmon

[21] "A fully detailed general approach to population genetics from the Dawkinsian point of view will involve equations that represent the functional dependence of the distribution of environments on the frequency of alleles, and equations that represent the fitnesses of individual alleles in different environments"... "[in population genetics] the frequencies p.q are not only the frequencies of the alleles, but also the frequencies with which certain environments occur. The standard definitions of the overall (net) fitnesses of the alleles are obtained by weighting the fitnesses in the different environments by the frequencies with which the environments occur" (Kitcher & Sterelny, p.13)

[22] One must note of course the similarity of this distinction and that of the replicator-interactor (or vehicle) distinction. Once more, it is not the precision of the criteria for the dichotomy that seems most weak but the obscurity in which it leaves the causal links between both elements,i.e. the sources of heritability.

notion of *screening-off* (Salmon,1971) to argue that differential reproduction of organism can be better explained by reference to the occurrence of certain favourable phenotypes. Given the information of their occurrence, the information about the underlying genetic elements becomes irrelevant for the causal explanation of the outcome.

If A renders B statistically irrelevant with respect to outcome E but not vice versa, then A is a better causal explainer of E than is B.

Screening-off typically is invoked with respect to correlations due to common causation, but of course it also applies for intermediate causes: more immediate causes screen-off more remote ones. So much is clear. What seems unsustainable is Brandon's claim that this undermines genic selectionism. As Sober has emphatically argued, as long as causation is transitive, arguments like Dawkins' [that it is in the end at the genic level where the causal chains start that end with differential survival and reproduction of biological entities] are unaffected by in-directness objections. Obviously transitivity implies screening-off of further causes by intermediate ones but that brings nothing new to the discussion. Either genes' differences produce different outcomes (effects) under the range of occurring environments or they don't. In the latter case they are undistinguishable by natural selection events and their relative frequencies will not depend on them. In the former they exert a transitive action and indirectly cause their (kind's) frequency dynamics. It seems to me that Brandon's argument is a strangely tortuous way to redescribe vehicle (or even property) selection, with which no genic selectionist would disagree. Evidently, whatever the source of the phenotypic trait it will have the same effects on the particular destiny of its bearer, but again it is the further consideration of a nonspurious correlation between traits and genes that allows an assessment of the global evolutionary significance of traits.

But what if causal transitivity sometimes fails to apply? then the directness objection would be justified. Again, Elliot Sober uses the distinction between individual level causation and population causation to base his case against genic selectionism. Individual causes are transitive but population causes can fail to be so. He writes

> The distinction between individual-level and population-level causal claims also is important to the question of transitivity of causal chains...the analysis of population level causal claims implies that *that* sort of causal relation is not in general transitive.(Sober, 1984a,p.297)

Perhaps genes cause phenotypes and phenotypes cause reproductive success. It might nevertheless fail to be true that genes cause reproductive success. If we interpret *selection for X* as meaning that possessing characteristic *X* causes reproductive success, we would have to say that natural selection is not selection for and against single genes. An *effect* of selection would be change in gene frequencies, of course. But the gene would nevertheless fail to be the unit of selection. (Eells & Sober,1983,p.53)

Genes of a certain kind can produce different effects from context to context. They can both participate in polygenic (non-multiplicative) assortments or divide their causal workings in a pleiotropic mode. Within the same evolving population their effects can be spoilt, enhanced or counteracted. It seems to me however that again it can be argued that only when, within a range of contexts, a gene can manage to produce a certain positive (phenotypic) effect and this in turn in most cases does make a difference for survival and reproduction, the situation is evolutionarily significant. Transitivity of probability enhancement can also be context dependent and thus, as before, the possibility that contextual (genetic or external) factors might change a gene's causal transitivity (for instance *neutralize* it) is acceptable for genic selectionism. In their above statement, Eells and Sober take for granted that gene frequencies will be directionally changed in selective events, without realizing that for that to happen a causal link (heritability of the selected trait) must exist, and that is a restriction over the domain of events they are actually referring to, to those where transitivity applies! So the mere possibility that sometimes causation in populations will not be transitive [i.e. that the raising of the probability of an intermediate causal factor B by a previous causal factor A, does not necessarily imply that if B raises the probability of an effect C, so will A] does not make it irrational to develop a scientific hypothesis where one is interested in the cases where transitivity does follow, because that produces outstanding long term consequences in the behavior of the systems (evolution, for instance). Genic selectionism is that kind of a interested hypothesis. It depends on two beliefs:
a) that only genetically determined fitness differences will produce evolution under natural selection, and b) that no matter how complex the pleiotropic or epistatic effects of individual genes may be, the competition structure of differential replication will permit that strong or persistent enough selection pressures will sort out better genes from lesser ones. This sorting between different genes at the same place and "for" the same function is what genic selectionism focuses on. That not all replicators are active (in Dawkins sense),

that not all bits of DNA have adaptive causal interactions in/with their environments cannot be denied. But as Dawkins has insisted, the appropriate way to look at actual (active) genes is as the survivors in the subset of the actual sequence of environments. Evolutionary explanations of adaptations are not about neutral or indifferent genes or traits subject only to the fortuitous sortings, they refer to the subsets of both genes and traits that have had, and can have causal consistency in the long run. Traits owe their long run causal consistency to the genes that raise their probabilities of occurrence, and it is only those genes that have managed in the past to transitively (via traits) influence their own kind's probabilities of survival through the generations that can be said to have been active replicators. If differences at any *vehicular* (trait) level have been decisive in the past for evolutionary outcomes, they must in the end have been differences between the "correlated" genes that favored one or the other at different loci and in the prevailing contexts[23] (23).

Eells and Sober, just a few paragraphs after the above quotation, manage to produce a surprising assertion that goes against their stance:

> Transitivity will be blocked only if the average effect of the gene is deleterious [we can read causally negative], and in that case we wouldn't be tempted to say that there is selection for the gene in any event. This means that it is polygenic effects and not pleiotropy that poses a problem for genic selectionism (Eells & Sober,1983)

But polygenic traits are properly understood as causally intransitive only when their effects can have no consequence on genic frequencies; i.e. when heritability is null. There is then no variation at the genic level that corresponds to the variation at the vehicular level. Allelic fitnesses are then all equal and there is no consistent correlation between certain genes an certain traits or effects, so selective events will leave on average gene frequencies unaltered. No evolution occurs. Vehicle selection alone, disconnected from replicator (genic) selection, enters in a kind of causal neutrality. Equilibrium states under selection are somehow always due to such lack of correlation that make genes temporarily "invisible" to selection. The fact remains however that sometimes allelic differences are really causal, and

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[23] Dawkins writes: It is a fundamental truth, though not always realized, that *whenever* a geneticist studies a gene "for" any phenotypic character he is always referring to a *difference* between two alleles. (Dawkins,1982,p.92)
And: a neutral mutation *isn't* a mutation at all, when we are thinking about legs and arms and wings and eyes and behaviour! (Dawkins,1986,p.304)

not only structural (like neutral amino acid substitutions), and then overall neutrality between two or more competing alleles can only happen if their negative contexts (where their effects are adverse) and their positive contexts are perfectly balanced. That is, when they occur in exactly the proportions needed to counteract each other's long run consequences. This is exactly the proposal for some polygenic traits, such as birth size in humans. Granting the (difficult) possibility of such context induced neutrality does not however limit the genic selectionists' claim that all *evolution* needs genic caused selection. And, as Rosenberg pointed out for the case of heterozygote superiority[24] (24), there is a sense in which there is really no selection going on in such processes. To state it properly: vehicle selection does not imply replicator selection, and only when it is causally coupled with it, does adaptive evolution proceed. Even if the contexts in which genes tend to appear will sometimes give an actual overall neutral effect that leaves its frequencies unaltered, a rather
special (highly improbable) situation is required for this to happen continuously. It is reasonable however to suppose that even in the most complex and non-mendelian polygenic effects (traits) there can be a permanent although slow non-fortuitous statistical change in allelic frequencies, and that however minimal differences in different alleles selective values exist; i.e. the selection coefficients are not all exactly equal. This of course is the basic pervasive belief of neo-darwinism. So, once again, I have to emphasize the central role in evolution played by the probabilities that possible contexts have of occurring.
Even if for an abstract theory of probabilistic causation all possible contexts must be equally taken into account, for the analysis and explanation of *historical* selection processes such generality can confuse the actual role of causal factors for given outcomes. A differential weighting of contexts is needed, emphasizing those sequences with higher probabilities of occurring and diminishing or ignoring those with lower probabilities. The counterfactual consideration that if other contexts had occurred the outcome would have been the opposite should not be an obstacle to explanation.

VII Genic Causal Efficacy

[24] "But since [in lethal heterozygote superiority] the gene ratios and the genotypic ratios in fact remain the same from generation to generation, it seems equally reasonable to deny that any selection is taking place in this case. There is a change in the proportions of surviving genotypes over time *within* each generation, but this is arguably no selection at all" (Rosenberg, 1983, p.335) It seems more adequate however to say there is no evolution.

The time has come to pick up the loose threads I have been dropping in the previous sections and to join them in an attempt to give an interwoven answer to the criticisms raised against genic selectionism. I have up to now mentioned or suggested the following statements:

-Genes (as kinds) are situated at the Weismannian causal vertex; they transmit directly their structure (replicators) and they interact causally with their surroundings (directly or through vehicles).

-Both these causal roles make genes *active replicators*: they influence their own (kind's) survival and reproduction. Such influence is probabilistic and depends on the connection of two causal processes: trait or property production (epigenesis) and selection or sorting. All heritable fitness values depend ultimately on this interaction.

-A kind of genic effects is the raising of the probabilities for the occurrence of certain traits or properties within a range of environments.

-Trait or property differences account for fitness differences within a range of environments. Fitness differences are caused by internal *and* external physical differences, by their interactions linked to survival and\or reproduction. It would be better, in a sense, to say they *are* those physical differences. (See Hodge,1987)

-Heritable fitness differences are those directly linked to differences at genetic level, when genes and traits are non spuriously "correlated".

-Fitness attributions are expectancies of causal behavior (within a range of environments). They are not predictions nor explanations.(See again Hodge, 1987)[25](25)

-Fitness attributed to any entity or trait at any level of complexity will be context-sensitive. As context changes brought about by the selective process (like frequency changes), or not, are in the end physical and causally relevant changes. The range of environments where a given fitness value will remain stable can be big or small, that will only make a pragmatic, not a qualitative, difference.(See Kitcher and Sterelny,1987)

-Genetic environments must be treated statistically and probabilistically in similar way as external environments. Any "linkage" among genes would thus

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[25] "That fitness... is an expected quantity makes it appropiate to see it as a reproductive expectancy analogous to a life expectancy. Now, expectancies are in themselves not causal and so without explanatory content. For these reasons, it is misleading to conceive of fitness differences as causally mediating between the causes of a difference in reproductive performance and the difference in the performance itself. We should not suppose that physical property differences somehow make organisms differ in fitness and that those fitness differences then somehow make them reproduce differentially".(Hodge,1987,p.257)

appear as coadaptation.
-Genes as evolutionary units are best defined by causal rather than by structural criteria (although similarity of structure is of course required). It can be said to summarize their causal role in evolution that they are the unit of heritability.
-However complex the vehicle or interactor, that is, at whatever level is the gene's effect finally being assessed, for adaptive evolution to proceed there should be allelic differences that are being picked out. Adaptation, i.e. the tuning of evolutionary change, can be attributed to selection acting independently, in a sense, at each locus. Of course, selection can fail to produce evolution, or evolutionary significant changes can be fortuituous and non adaptive, but genic selectionism can harmlessly accept both possibilities.
-Genes, as probabilistic causal factors, can be said to be responsible, within given ranges of environments, for their (kind's) frequency in subsequent stages of selective processes. Overall effects, or averages, can certainly be indicative of their statistical causal performance. The possibility that in some contexts they will cease to have the positive causal effect (becoming neutral), or even have a negative one, is not a reason for withdrawing their relative explanatory thrust.

Elliot Sober's mistrust of averages and overall analysis of causal factors in populations is founded on the open possibility that the correlations found by statistical procedures are not really indications of direct causal links, and on the symmetric possibility of there being causal links overlooked by statistical handlings. There are several conceivable situations in which overall probabilities provide a misguided impression of the individual level causal events that give rise to them, and on the other hand there are individual causes that cannot be considered causal factors for the general class or population of events to which they belong. If smoking increases the probabilities of heart diseases in the members of a population, and if exercising decreases them, and both factors (smoking and exercising) are somehow correlated, then the overall analysis will not reflect the strength of the probabilistic causal link between smoking and heart failure.[Unless of course exercising is considered as a causally relevant background factor and a further partition is produced]. What Sober argues is that, supposing correlation of smoking and exercising is produced by a certain common cause k, then there will not be a given stable causal role attributable to k in the population as a whole with respect to heart condition, even though in different subpopulations

it can play positive or negative roles. Any purported positive causal factor C for an effect E must be assessed in relation to how it affects each individual's probabilities of attaining E. That is to say, how it affects the chances of the members of each cell of the partition generated by considering all the causally relevant background conditions. Average performance of C in the whole population can produce correlations between C and E but

> Correlation looks at *overall* probabilities. It compares the probability of heart attacks among smokers with the probability of heart attacks among non-smokers. The causal concept we are considering looks at probabilities on a case-by-case basis; we see whether each individual would run a higher risk of coronary if he smoked. The overall probabilities need not reflect the individual probabilities; this fact is sometimes called Simpson's paradox. Even though the overall frequency of coronaries among smokers may be *lower* than the frequency among non-smokers, it may nevertheless be true that each individual runs a *greater* risk of a coronary if he or she smokes
>
> (Sober, 1984a,pp.285-286)

I believe that the above obscuring of the causal influence is better understood as a case of frequency dependence. It is only the frequency with which "smoking" will find itself (as casual factor) in the company of "exercising", due to the correlation postulated, that the overall analysis is, so to speak, misleading. Although it may be wrong to conclude that smoking is not raising each individual's chances (i.e. each exhaustive partition cell's[26] (26)) of heart failure, the population's relative health is due to a contextual bias. To put it another way, it is only because in the population as a whole there will be few members of the cell that considers smoking without exercise, that this condition has little weight in the averages. If we imagine that heart disease will have a negative evolutionary consequence, (i.e. if we add a selective process to the example) and furthermore if we postulate a gene k that pleiotropically favours both smoking and exercising (thus accounting for the correlation), it is possible to see that the evolutionary destiny of such gene

[26] It should be noted that Sober doesn't make clear in his analysis that, although correlated, smoking and exercising have to be considered as background factors, one for the other, when the partitions to search for their causal roles with respect to heart disease. So any individual will be representative of a cell, and his chances of having the disease will be equal to all the other members with and without the causal factor under consideration. Disconnecting correlated antagonic causal factors will then open the possibility to discern, statistically such antagonism.

will depend at least partially on the frequency with which the negative effect(smoking) will find itself neutralized by the positive one (exercising). If for any reason the frequency of the individuals where only the negative effect is exerted rose, the gene k would be selected against. To come back to the non-genetical example: if somehow one could manage to disconnect the correlation between smoking and exercising (to have for instance all partitions of the *same* size) then the positive effect of smoking for heart disease would be made clear by overall statistics. And, similarly, if gene k is selected "for" it would mean that its positive effects are favoured by the context shuffling. This of course does not count as a general criticism of the view of probabilistic causation that Sober advocates, but it does undermine the plausibility of his view, as contextual causal workings acquire a major probabilistic explanatory role that Sober underestimates. I have tried show how the application he intends to make of his view in evolution is not valid, mainly because of the peculiarities of context occurrence for the behavior of evolving populations, which dramatically oppose his conditions for being a probabilistic causal factor. The so called Pareto-style requirement is somehow invalidated in selection processes by the causal importance of contexts' probabilities of occurrence. One should now be able to see the gap in the following assertions:

> To say that there is selection for a given gene at a particular locus is to say that possessing that gene is a positive causal factor in survival and reproduction. This in turn requires that the allele must not decrease fitness in any context and must raise it at least in one(Sober, 1984a,p.302)

> [In heterozygote superiority] the *a* allele does not have a unique causal role. Whether the gene *a* will be a positive or a negative causal factor in the survival and reproductive success of an organism depends on the context (Sober,1984a,p.302)

There is no evaluation of the actual causal importance of each context. They are all given the same weight. Taken to the absurd, in this position there would be no positive causal factors whatsoever. The heterozygote superiority case is illustrative of such mistake. When not at equilibrium the minority allele will enjoy a selective advantage because the context in which it will more often be found will have the "rival" allele as partner. As a consequence, its frequency will increase in the population. It would seem ludicrous not to acknowledge that

such allele is a positive causal factor in that process (that presumably will drive the population to equilibrium) just because once the favourable contexts become scarcer its "causal powers" diminish. At equilibrium, the allele will have 50% chances of finding itself (after fecundation) in a good site (heterozygote) and 50% of being in a bad one (homozygote); what has been lost is the correlation, due to the allele plus the genetic environment, between survival and the possession of the gene; that is, in a sense, the heritability of the selected for trait. Evolution, as we have already stressed, comes to a halt. The causal activity of the evolving context, which in this case is the change in allelic frequencies, is as important, and should be assessed as such, as that of the causal factors.

Richard Dawkins has made that point over and over:

> genes are selected, not for their intrinsic qualities, but by virtue of their interactions with their environments. An especially important component of a gene's environment is other genes. The general reason why this is such an important component is that other genes also change as generations go by in evolution (Dawkins, 1986,p.192)

Part of Dawkins' emphasis since *The Selfish Gene* is on the tendency to find or generate stable and restricted environments that selection processes produce. That is precisely the rationale of the mediating activity of vehicle selection. And that can be extended, of course, to the selection of genetic company. Phenomena like linkage disequilibrium, and co-evolution of teams of genes are readily picturable as a kind of contextual selection from individual gene's perspectives. Of course, if the correlation becomes too strong between several (physically and causally linked) genes it almost certainly means that there is a very strong selection pressure that in the end is making every bit of the team a neccessary element for survival and reproduction[27](27). In a sense, the phenomena might be better viewed by focusing on the ensemble as the causal factor (or replicator)

> so the possibility of strong linkage disequilibrium does not weaken the case[of genic selectionism]. It simply increases the size of the chunk of genome that we can usefully treat as a replicator (Dawkins,1982,p.89)

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[27] "It is clear that when permanent linkage disequilibrium is mantained in a population, the higher order interactions are important and the chromosome tends to act as a unit"(Slatkin,1972, quoted in Dawkins,1982,p.89)
"...the unit of selection is a function in part of the intensity of selection: the more intense the selection, the more the whole genome tends to hold together as a unit"(Templeton et al,1976, quoted in Dawkins,1982, p.89)"

Finally, to close this issue of context dependence, I want to insist on Rosenberg's point (1983, see note 18) that one must not confuse a causal and explanatory claim, as the one made by genic selectionism, with hypothetical (acausal) attributions of fitness coefficients to genes or alleles. If, in a typical situation, a given statistical procedure correlates an allele with survival under certain conditions (as often happens in geneticists' experiments) it is of course a healthy position not to attribute uncritically the cause of survival to the allele; at most that would be a possible working hypothesis that would have to be explored. Here Sober's cautions are more than relevant. But a very different matter is the case when we know or have reasons to suppose that a given allele's effects (within a highly probable range of environments) are included among the causes of differential survival. We have then every reason to consider the allele a causal factor, or as Sober would perhaps say, as a ultimate *source* of fitness values (See Sober,1984a,p.255).

As has been suggested before, the so-called transitivity objection is closely linked to the context-dependence one. Simpson's paradoxes, Sober insists, force us to focus on the movement of individual chances with the presence or absence of the proposed causal factor, instead of doing it on overall chances.

I have suggested that for selective processes, where outcomes affect the context in which the next trial will take place, the sensible thing to do is to restrict one's attention to the probable contexts and the action of the causal factor in (sequences of) them. A similar suggestion applies for the intransitivity objection. An allele G can raise the probability of occurrence of an advantageous phenotypic property P, and P in turn can raise its bearer's chance of survival and reproduction(E), without there being a similar rise in the probabilty of E given G. In algebra:

$$p(P/G) > p(P) \;\&\; p(E/P) > p(E)$$

$$\text{but } p(E/G) \text{ not}> p(E)$$

This situation can come about in different ways and it means among other things that genic effects will fail to be correlated with survival and reproduction, and it will also mean that selective events, even if they would somehow change the frequencies of genes present, will fail to bring about the reappearance in further generations of the selected properties (unless of course the population is in equilibrium, and so the frequencies were not affected). This is again what is known as lack of heritability, and is a major obstacle for evolution to

proceed (as breeders well know). So once again, a restriction in focus to those cases where transitivity applies would seem a sensible move for the causal analysis of evolutionary processes.
Transitivity of causes, even if not a general feature of probabilistic causation, is a requirement for the effectiveness of natural selection in causing evolution.

VIII Conclusions

I have argued against the sophisticated form of two basic objections against genic selectionism. Context-dependence and in-transitivity of causal influence. The former objection favours the genotype as the unitary genetic element (replicator) that is selected *for* during evolution by natural selection.
One criteria used (Lewontin and Sober,1982)is the stability of fitness coefficients (mistakenly taken to mean stability of causal influence) with the changes of gene frequencies brought about by the selection process. The fact is that no context independent causal stability whatsoever can be warranted in selection processes (Kitcher and Sterelny,1987). Evolutionarily significant causal influence can only be statistically and probabilistically assessed considering the actual environments' probabilities of occurrence. Genic (replicator) selection will always depend on the causal differences between alternative alleles within the range of occurring (genic and external) environments. Such causal differences, obviously due to physical differences, will manifest themselves in different genes being correlated, in a range of environments, with different traits or property varieties, and thus with different selective values. Such correlation is the basis of those traits or properties heritability. Natural selection's efficacity as adaptation producer depends on that genic characteristic: being the unit of heritability. The second objection, intransitivity of causal influence, challenges genic selectionism by favouring other levels of selection, besides the genetic, as causally fundamental in evolution. The idea is that properties from several levels of organization can be the cause of selective events. Genes it is said are invisible to selective events, as their interactive properties are restricted in scope to molecular intracelular actions. The phenotype, it is argued for instance, exerts the same influence on survival and reproduction, no matter what the genetic and environmental combination produced it. The answer to such criticism I have favoured is to say that replicator selection, which

includes gene selection, is quite different in its causal structure from vehicle selection. The emphasis I have tried to make is in the idea that the link between both is captured by the causal correlations we call heritability. Transitivity of causation between replicators and vehicles is secured by it, specially in evolutionarily significant processes, in which natural selection drives the changes in gene frequencies in a population. Elliot Sober's central tenet in *The Nature of Selection* is that for every process of evolution under natural selection there is one and only one causal description that depicts the real workings of natural selection. If for instance the property that is being used as sorting criteria (that is being selected *for*) is a group property, then only group selection will explain appropriately the process. Richard Dawkins(1982) and, recently, Kitcher and Sterelny (1987) defend the view that several adequate representations of selection processes are always possible, and that it is the maximalization of such adequacy what makes genic selection the better option.

> For *any* selection process there is a maximally adequate representation that attributes causal efficacy to genic properties. (Kitcher & Sterelny, 1987, p.33)

The discussion of course runs in parallel with the general dispute within Philosophy of Science between realists (Sober) and empirists (Kitcher). I have been trying to show why I believe both positions are mistaken with respect to genic selectionism. Their basic mistake is to suppose there is a competition for being *the* unit of selection, and that the gene is one of the participants. My view is that the gene has in evolutionary processes a separate causal role which has no alternative. It is not the same causal role that Sober calls property selection (and that Kitcher and Sterelny follow in the above reference), and which is best understood as an explication of Dawkins' vehicle selection. The latter can be said to be the territory where these disputes between the alternative units and/or levels of selection, and between the philosophical stances are really taking place. But they only cover one aspect of a dual process; an aspect that seems still rather confused. Conceptually, the talk of property selection, or of trait selection or, generally of vehicle or interactor selection, seems to perpetuate the difficulties that previous concepts, like fitness, presented.

Replicator selection is much better understood. It means the selection of genes and, however passive a role some authors want to establish for them as selected objects, it is the active rising of the survival and reproduction

probabilities of their kind in the given range of environments what will be evolutionarily significant. And that cannot be but causal and explanatory.

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