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Is Pure $R$-Selection Really Selection?

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Lennox and Wilson (1994) critique dispositional accounts of selection on the grounds that such accounts will class evolutionary events as cases of selection whether or not the environment constrains population growth. Lennox and Wilson claim that pure $r$-selection involves no environmental checks on growth, and that accounts of natural selection ought to distinguish between the two sorts of cases. I argue that Lennox and Wilson are mistaken in claiming that pure $r$-selection involves no environmental checks, but suggest that two related cases support their substantive complaint, namely that dispositional accounts of selection have resources insufficient for making important distinctions in causal structure.

1. Introduction. Lennox and Wilson (1994) hold that cases of pure $r$-selection, i.e., changes in phenotypic or genotypic frequencies driven only by differences in intrinsic rates of increase, are not cases of natural selection, and further that dispositional accounts of selection are mistaken because they mis-classify cases of pure $r$-selection as cases of true, Darwinian, selection. They argue as follows. Changes in genotypic or phenotypic frequencies may be produced either by random causal processes or by non-random causal process. The former are nearly universally taken to be processes of drift, the latter processes of selection. Dispositional accounts of selection, among them Brandon’s important 1990 account, take this distinction to be the fundamental distinction a definition of selection must recognize, and define ‘selection’ so as to include virtually all non-random processes leading to reproductive success.

Lennox and Wilson point out, however, that Darwin was not alone in thinking that selection involves some sort of competition between

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individuals of differing types, a ‘struggle for existence’. ‘Competition’ here is to be broadly understood to include not only competition for one or more limiting resources, but also differing susceptibilities to predation, climate, and disease, differing ecological dependencies, and so on. Competitive processes, so understood, are a heterogeneous group, but they have one underlying common feature; indeed, for Darwin, it is this feature which makes a process competitive in the relevant sense: all such processes constitute, in some sense, an environmental ‘check’ on population growth. We may then differently pick out the set of selection processes as those non-random processes leading to reproductive success that also check population growth.

Lennox and Wilson assume that instances of non-Darwinian selection are at least possible, and that this possibility is exemplified by cases of pure $r$-selection. When two types differing in their intrinsic rate of increase, $r$, share a common environment that does not otherwise restrict the growth of either population (i.e., both types realize their respective maximal $r$ values), differential reproductive success occurs, and leads to changes in the relative frequencies of the two types in the population. The differential success is not, however, produced by any process that is an environmental check on population growth. Such cases of frequency change are therefore not cases of selection. The processes are, however, non-random in the relevant sense: for the two types to realize their different $r$ values in such an environment just is for them to differ in their dispositional fitnesses in that environment. Standard accounts of natural selection, which define selection in terms of differential dispositions to reproduce in specific environments, will therefore incorrectly classify cases of pure $r$-selection as cases of natural selection. Hence these accounts, Brandon’s among them, achieve a generally unified account of selection only at the price of failing to distinguish between two importantly different kinds of causal structures: those in which the causal processes driving reproductive success are in some sense an environmental check on population growth (so-called Darwinian selection), and those in which the causal process do not constitute such a check (changes in phenotypic or genotypic frequencies due merely to differences in reproductive fitness) (cf. 1994, 76).

Lennox and Wilson claim, then, that there is at least in principle a distinction between non-random processes that are and those that are not environmental checks on population growth, that standard accounts of selection do not recognize that distinction, and hence that such accounts illegitimately unify causally distinct evolutionary events. They suggest that a correct account of selection will have to recognize, in place of the standard distinction between drift and selection, a tripartite distinction between drift, Darwinian selection, and differences
in reproductive fitness, the first including all random processes leading
to reproductive success, the second including those non-random pro-
cesses that are environmental checks on population growth, and the
third all those non-random processes that are not environmental checks
on population growth (cf. 1994, 76).

The objection advanced by Lennox and Wilson depends for its force
on two claims: 1) our theory of natural selection ought to distinguish
between cases in which the environment constrains population growth
and cases in which it does not, and 2) in pure r-selection the environ-
ment does not constrain population growth. There is a clear sense of
‘environmental constraint’ on which claim 1 is correct. But I think, on
that sense of ‘environmental constraint’, claim 2 is suspect. To assess
the claims we require an account of causal processes and a view about
what makes such a process an environmental check on population
growth. Since Lennox and Wilson provide neither, I shall begin by
briefly delineating an account of causal processes and a view about
what it is for processes, so defined, to count as environmental checks
on population growth. I shall then show that cases of pure r-selection
do involve an environmental constraint on population growth.

2. Causal Processes and Environmental Checks. Let the set of causal
interactions be the set of causal connections which are basic in the sense
that such a connection, say between A and B, cannot be partitioned as
a composite of a more elementary causal connection between A and
some C, and yet another between C and B.¹ Since I mean here only to
be offering a scheme for individuating any given causal connection from
other causal connections, I take no stand on the nature of the items re-
lated by an interaction, or on the nature of the features of related items,
if any, in virtue of which such an interaction obtains. Nor am I here
advancing any view about what differentiates specifically causal inter-
actions from non-causal interactions, whatever such may be.

Any causal interaction may be completely individuated from other
causal interactions by a specification of the items between which the
interaction obtains, the features of those items in virtue of which the
interaction obtains (the licensing features for the interaction), the fea-
tures of the items produced by the interaction (the effect features of
the interaction), and the temporal interval over which the interaction
obtains. Two interactions are instances of the same kind of interaction,
or causal relation, if the licensing features for the two are instances of
the same feature-kinds, and similarly their effect features are instances

¹. A more thorough account of the explanatory individuation of causal processes, from
which this more brief discussion is drawn, can be found in Glymour 1998.
of the same feature-kinds. Causal relations may therefore be individuated from one another by a set of licensing feature-kinds and a set of effect feature-kinds.

Causal interactions can occur sequentially in causal chains, e.g., A and B causally interact, then B and C causally interact, and so on. A special kind of causal chain, or what I shall call a causal trace, occurs when one of the effect features of the A-B interaction is a licensing feature for the B-C interaction, and so on. Just as causal interactions belong to kinds, so to do causal traces. A trace-kind may be individuated from other trace-kinds as a sequence of causal relations. Any given causal trace is an instance of a given trace-kind just in case for all $i$, the $i$th interaction in the trace is an instance of the $i$th causal relation in the trace-kind. I shall call trace-kinds causal processes, and hence take an instance of a causal process to be a causal trace. I shall say that a causal relation which is in the sequence of relations used to individuate a causal process is included in the process, and if the relation holds between A-type items and B-type items, then A- and B-type items will be said to play a role in or to be on both the relation and the process.

We can now examine the notion of an environmental check on population growth. Consider a population $P$ of bacteria, all of a given species. The growth in the size of the population during the unit interval from $t$ to $t'$ is simply the increase in population size $G = N(P,t') - N(P,t)$. The rate of growth during the unit interval is simply $G$ divided by $N(P,t)$. Now consider a process which leads to reproductive success for our bacteria, i.e., a process instances of which terminate in interactions producing cellular division. Reproduction in our population may be produced by one such process or by many, but in either case the increase in population size between $t$ and $t'$ is determined by the frequency with which all such processes are instanced in $P$ between $t$ and $t'$. Each instance of each process takes one bacterium and produces, in its place, two new bacteria. Hence, where no bacterium dies, and there are $n$ processes leading to cellular division, $N(P,t') = N(P,t) + \sum f_i N(P,t)$, where $f_i$ is the frequency of the $i$th process in $P$ from $t$ to $t'$.

If the environment is to limit population growth, it must do so by limiting the frequencies of the processes leading to cellular division, i.e., $\sum f_i N(P,t)$.

2. If there are processes leading to deaths of individual bacteria, then $N(P,t') = N(P,t) + \sum f_i N(P,t) - \sum g_i N(P,t)$, where $g_i$ represents the frequency of the $i$th process leading to bacterial death, of which there are $m$. In this case the environment may check growth by limiting the value of the difference between the frequencies of the division producing processes and the death producing processes, i.e., by limiting $\sum f_i N(P,t) - \sum g_i N(P,t)$. 


the frequency with which a process is instanced is to limit the frequency of some causal relation constitutive of the process. If such a relation obtains between organisms and some kind of item in the environment, then the frequency of the relation will be limited by the number of items of the relevant kind. Suppose, for example, that in our environment the only source of energy for the bacteria are sucrose molecules. Each bacterium must absorb and metabolize a sufficient number of sucrose molecules in order to store enough energy to undergo mitosis. Suppose the relevant number of molecules is $M$. If the environment contains only $M'$ sucrose molecules during the interval $t-t'$, then the frequency in $P$ of the process leading from sucrose digestion to mitosis can be no greater than $(M'/M)/N(P,t)$. So I shall say that a causal process leading to reproduction constitutes an environmental check on population growth just in case that process includes a causal relation in which some kind of item in the environment plays a role.

To show that there are cases of pure $r$-selection in which the environment actually checks population growth it will not do simply to show that there are environmental checks on population growth, one must show as well that the actual frequency of the relevant processes depends, in some sense, on the environment. I will say that the environment $E$ occupied by a population $P$ constitutes constraint on population growth just in case there is no physically possible alternative environment in which the rate of growth for $P$ is different. I will say $E$ is a constraint on differences in rates of growth for two sub-populations $P_1$ and $P_2$ just in case there exist at least two possible environments $E$ and $E^*$ such that the ordinal relation between the rates of growth of $P_1$ and $P_2$ true of $E$ are not true of $E^*$, i.e., if $r_1 > r_2$ in $E$, then $r_1 \leq r_2$ in $E^*$. The idea is this: if the rates in $E^*$ are different from those in $E$ (as they must be if ordinal relations between rates are reversed), then at least one process leading to reproduction for members of at least one sub-population has a different frequency in $E$ than in $E^*$. Since the phenotypes remain unchanged, the reason this process has a different frequency in $E$ than in $E^*$ must be found in the difference between $E$ and $E^*$. In this sense, then, the frequencies of the processes leading to reproduction causally depend on the environment.

It is now straightforward to show that any environment $E$ will count as a constraint on the growth of any population $P$ if $P$ has a non-zero growth rate in $E$ and the processes that lead to reproduction in that population are all environmental checks on growth. Since each process is a check on growth, each involves a relation on which lies some kind of environmental item. If that kind of item never occurs in some $E^*$, the relation, and hence the process, must never be instanced in $E^*$. Let $E^*$ be an environment lacking some environmental item on each pro-
cess leading to reproduction in P. The frequency of the relevant processes in E* will be zero, and hence $r_p$ in E* is different from $r_p$ in E. E therefore constrains the growth of P. Similarly, any E will count as a constraint on differences in population growth for P1 and P2 if $r_{p1} \neq r_{p2}$ in E and all processes leading to success in P1 or P2 are checks on population growth. Simply let E* be an environment in which for each process leading to success in either P1 or P2 some environmental item kind in the process is absent. Then no process leading to reproduction in either sub-population is ever instanced, and in E* $r_{p1} = r_{p2} = 0$. E is therefore a constraint on differences in rates of growth.

3. Pure R-Selection. Lennox and Wilson repeatedly recur to a particular case of pure r-selection, henceforth referred to as experimental setup 1. Consider two bacterial types, A and B, with different division times (i.e., different maximal r values), growing in a medium containing excess nutrients and not containing predators, parasites, etc. If we begin our experiment by inoculating the medium with equal numbers of types A and B, we will shortly find that the type with the higher r value, say A, has increased in frequency, and that it will continue to do so as long as the experiment is run. Lennox and Wilson claim that in this case there is no ‘struggle for existence’ and hence no environmental constraint on population growth. I disagree.

Denote the environment in setup 1 by E. In that environment $r_A > r_B$. E will count as a strong environmental constraint on differences in population growth, and as a strong constraint on population growth for both type A and B bacteria, provided all processes leading to division in A- and B-types are checks on growth. But that they must be, for no process will lead to division unless it involves the ingestion of nutrients by bacteria. Thus, I claim, pure r-selection does involve environmental constraints on population growth. It does so because there will be some alternative environment, i.e., one that lacks all nutrients, in which neither type reproduces, and hence the actual environment E will count as a physical constraint on both rates of growth and differences in rates of growth.

4. An Objection Considered. This result depends on the notions of ‘environmental constraint’ deployed above, and to those notions one might object as follows. Let $E_{MA}$ be any environment in which the value of $r_A$, call it $r_{A'}$, is as large or larger than its value in any other physically possible environment. Then it is reasonable to think that the reason $r_A$ is not larger than $r_{A'}$ in $E_{MA}$ has nothing to do with features of $E_{MA}$ and everything to do with the phenotypic limitations characteristic of A-type bacteria. Since E in setup 1 is both an $E_{MA}$ and an $E_{MB}$ environ-
ment, an explanation of why \( r_A \) and \( r_B \) are not higher than \( r_A \) and \( r_B \) in setup 1 must appeal to phenotypic rather than environmental features. Differently, complete explanatory responses to this question must, in cases of Darwinian selection, appeal to environmental features, for in those cases the actual values of \( r_A \) and \( r_B \) will be less than \( r_A \) and \( r_B \) respectively. Since, here, the phenotypes permit higher \( r \) values, part of the reason the actual \( r \) values are not higher must lie in the environment. Thus, we have differing explanatory possibilities marking differences in the causal structures generating differential success in the cases of pure \( r \)-selection and Darwinian selection. In the second, but not the first, case the rates of growth, and hence the ordinal relation between them, can be explained by appeal to environmental features. But this difference in causal structure is concealed by the notions of environmental constraint introduced above. Hence, it may be argued, they are the wrong notions.

The objection is, I think, confused. If there is some question about \( r \) values that can be answered by citing environmental causes in the case of Darwinian selection, but not in the case of pure \( r \)-selection, that will count as reason for thinking there are differences in the causal structures driving success in each case. If it is further important for some explanatory or predictive purpose that our theory of selection recognize that difference in causal structure, we will have here a cogent objection. But I think that the noted difference in causal structure is less significant than it appears, and that it serves no explanatory or predictive purpose essential to an account of selection proper.

We may ask two questions about actual reproductive rates in a given environment: Q1: 'Why are the rates not different than they are', and Q2: 'Why are the rates not higher than they are?' Environmental features are part of the complete explanatory response to Q1 in both Darwinian and pure \( r \)-selection. The environment is relevant in both cases because in both cases there is some alternative environment in which the rates would be different. The same is not true of Q2. Because the relevant contrast class in Q2 is restricted (to pairs of reals \( r_1 \) and \( r_2 \) in which \( r_1 > r_A \) and \( r_2 > r_B \)), the environment is not explanatorily relevant when, as in pure \( r \)-selection, the rates take their maximal values, \( r_A = r_A \) and \( r_B = r_B \). The environment will be relevant when these equalities do not hold, i.e., in Darwinian selection, because in these cases there is some alternative environment in which the rates would be higher than they are in the actual environments. So it is the difference in ex-

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3. Henceforth I attend only to the causes and explanations of \( r \) values rather than of differences in \( r \) values, on the assumption that whatever causes the \( r \) values explains the differences between them, since the former determine the latter.
planatory responses to Q2 rather than Q1 which must mark the difference in causal structure that it is somehow essential for our account of natural selection to recognize.

The question is, why should we insist that our theory of selection, as opposed to some other sort of biological theory, in ecology or morphology for example, provide the resources for making this distinction in causal structure? The reason cannot be one of predictive power, for there is no question that the machinery of population genetics, which does not differentiate between pure r-selection and Darwinian selection, is predictively adequate. So, I take it, the worry must be explanatory.

It is clearly essential that we be able to explain why the growth rates are not different than they are, and do so causally, since the rate at which the frequencies of types change in a population over generational time depends on the growth rates. One aim of such explanations is to identify which of several contrasting causes generates the growth rates. Hence, if there are cases in which the environment plays no role in causally generating growth rates, our theory ought not unify these cases with more standard cases in which both environment and phenotype causally contribute to growth rates. Unifying Darwinian and pure r-selection does not, however, violate this constraint, since in both cases the growth rates causally depend, in a sense identified by the above deployed notions of constraint, on the environment.

One might further demand that our theory of selection enable the rather more intricate distinction between cases in which the environment causes growth rates not to be higher than they are (Darwinian selection) and cases in which the environment does not play this causal role (pure r-selection). The demand is appropriately made of some theories. The distinction is required by, for example, functional morphologists or physiologists who wish to answer questions of the form: ‘Why does phenotype A not reproduce faster than $r_A$ in $E$?’ where $r_A$ and $E$ are replaced by a real number and the name of an environment respectively. The distinction is required because in some cases the question is completely answered by appeal to features of the phenotype A and nomic relations between those features described by bio-chemical, physiological or developmental laws. In other cases environmental features must be mentioned as well. But for all that there is no principled reason the distinction need be recognized by the general theory of selection proper. The distinction is required for a very specialized explanatory aim which, while broadly evolutionary, it is not an aim for which the theory of selection must, on pain of inadequacy, be competent.

5. Environmental Checks and Unified Accounts of Selection. Lennox and Wilson are correct that a theory of natural selection need do more than
distinguish between drift and selection. In particular the theory must not unify cases in which growth rates causally depend on both the environment and the phenotypes with other cases in which the growth rates depend only on phenotypes. One way insure that our theory does not illicitly unify such cases is to count as natural selection only cases in which the environment acts as a constraint on growth. On this understanding of the role of such a notion, the above definitions of ‘environmental constraint’ are appropriate. And on those notions of constraint, pure r-selection involves such constraints, and hence does count as natural selection. Dispositional accounts of selection, Brandon’s among them, do fail to differentiate between cases in which the environment plays a role in causing rates of growth not to be higher than they are (Darwinian selection), and cases in which the environment plays no such role (pure r-selection). But there is no reason to think they are for this reason inadequate. That distinction is not essential for any explanatory or predictive project that a theory of selection must, on pain of inadequacy, make possible. Hence, if dispositional accounts do illicitly unify cases of differential growth produced by relevantly different causal structures, that does not result from some failure to distinguish between cases in which the environment constrains growth rates and cases in which it does not.

But perhaps what is really at issue, for Lennox and Wilson, is not the distinction between non-random processes which are and those which are not environmental checks on population growth per se, but rather the more general fact that Brandon’s theory, and dispositional accounts of selection generally, unify the phenomenon of selection by abstracting virtually all the causal detail from any given case of selection. On dispositional accounts of selection, to explain a given case of frequency change as the result of natural selection one must show that the processes driving reproductive success are non-random. But one need describe neither those processes nor their actual frequencies in any detail, nor, importantly, need one say much about how those frequencies are nomically constrained or about the environmental features in virtue of which the frequencies are constrained. If Lennox and Wilson desire that kind detail in selection explanations, they are surely right that neither Brandon’s theory, nor any other generally unified theory of selection currently on offer, require it. But this inadequacy is surely not rectifiable merely by insisting on the tripartite distinction that Lennox and Wilson suggest. Moreover, it is not at all clear that we should want it rectified. There is at least a long tradition in philosophy of biology of abstracting from precisely this sort of detail. There are, however, reasons we might wish to depart from the tradition.

Consider the contrast between setup 1, examined above, and the
following two alternative setups, each beginning the same way as setup 1. In setup 2, however, we allow the two bacterial strains to grow only until the total population size is 100,000, after which we hourly remove, in random fashion, bacteria so that the population size is reduced to 100,000. Setup 3 is identical to setup 2, except that instead of sampling randomly when we remove excess bacteria, we select our sample so that the sample frequencies of A and B types exactly reflect the population frequencies of the two types at the time the sample is taken.

In setups 2 and 3, but not in 1, the population eventually becomes fixed for either type-A or type-B, and it would be hard to maintain that selection is not operating in either setups 2 or 3. But the sampling process by which bacteria are removed is in neither case legitimately regarded as a selection process, on either standard accounts or on that of Lennox and Wilson. By hypothesis the sampling process in setup 2 is a process of drift, since it is random. And if the sampling process in setup 3 is not a process of drift, since it is non-random, neither is it a selection process, since the probability that any given A-type bacteria is included in the sample removed is exactly the same as the probability that any given B-type bacteria is included: the sampling process is not discriminate, in Beatty’s (1984) sense.

If selection is, by hypothesis, operating in setups 2 and 3, but no process operating in either that is not also operating in setup 1 is a selection process, then it seems hard to deny that selection is, after all, operating in setup 1, contra Lennox and Wilson. On the other hand, the differences in causal structure exhibited by the setups generate important differences in the behavior of populations subject to the different setups. For example, trivially, populations in setup 1 never become fixed for one or the other type, while populations in setup 2 and 3 do become fixed. An ensemble of populations in setup 2 will, however, exhibit greater variance in time to fixation than will an ensemble in setup 3. Moreover, populations subject to setup 2 may, on occasion, become fixed for type-B bacteria, while populations subject to setup 3 never do. The differences in causal structure between these various cases is entirely concealed by Brandon’s account of natural selection, and hence that account can provide no explanation for the different behaviors exhibited by populations subject to the differing regimes. Such explanations are to be had, if at all, only by piecemeal appeal to a wealth of ecological detail and substantive ecological theory.

If it is true that all three setups involve selection, it is also true that populations subject to the differing setups behave in systematically different ways. Arguably, since the differences in behavior are systematic, an explanation of them ought also be systematic. Such explanations ought not require appeal to this or that special ecological theory, but
rather to some fundamental fact about differences in the general causal structure exhibited by populations subject to the different selection regimes. If so, then a general theory of natural selection ought not only count all three sorts of cases as selection, it ought also provide the resources for recognizing the fundamental differences in causal structure, and predicting their consequences. If one is puzzled about how exactly the sampling process in the third setup ought to be described (since it is prima facie neither a selection process nor a process of drift), or about why such sampling processes generate such different consequences than random and merely non-random sampling processes, then to that extent one must regard now standard accounts of selection based on dispositional theories of fitness, Brandon’s among them, as inadequate. Remedies for this inadequacy, however, will require more than ever more subtle dispositional definitions of selection, built to satisfy ever more intricate distinctions between differing causal structures. I suggest a remedy requires a systematic method for individuating and describing the causal structures driving evolution alternative to the now standard machinery in which very different structures are all lumped together as cases in which dispositionally defined fitnesses probabilistically cause reproductive success. To the extent that Lennox and Wilson can be fairly understood as defending the thesis that the current machinery is simply insufficiently subtle for the task to which it is applied, it seems to me they are not very wrong.

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