IS PURE *R*-SELECTION REALLY SELECTION?

Lennox and Wilson (1994) critique modern accounts of selection on the grounds that such accounts will class evolutionary events as cases of selection whether or not the environment checks population growth. Lennox and Wilson claim that pure *r*-selection involves no environmental checks, 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 modern 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 modern 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. Modern 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 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 do 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 an environmental 'check' on population growth, in some sense of what it is to be a check on such 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.

Moreover, Lennox and Wilson claim that there are instances of non-Darwinian selection in our world: 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 *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 which is an environmental check on population growth. Such cases of frequency change are therefore not cases of selection. The processes are, however, non-random processes 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) (c.f. 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 the history of evolving populations includes processes of both kinds, 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 processes that are environmental checks on population growth, and the third all those non-random processes that are not environmental checks on population growth (c.f. 1994, 76).

The objection advanced by Lennox and Wilson depends for its force on the

physical possibility of realizing, in our world, a case of frequency change driven only by non-random processes that are not an environmental check on population growth. If such cases are realizable in our world, then the free application of standard accounts of selection risks incorrectly unifying importantly different phenomena. If, however, such cases are not realized in our world, then the domain of application of standard theories is not restricted within our world and worlds relevantly like it. I shall argue that cases of pure *r*-selection do involve environmental checks on population growth. The argument requires an account of causal processes and of what it is for a process to constitute an environmental check on population growth. Since Lennox and Wilson provide neither, I shall begin by briefly delineating an account of causal processes, advanced by me elsewhere, and providing an account of what it is for a processes, so defined, to count as an environmental check on population growth. I shall then show that, on this view, at least one case of pure *r*-selection, the case Lennox and Wilson explicitly consider, does indeed involve an environmental check 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. Any such 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 features of the items produced by the interaction (the *effect features* of the interaction), and the temporal interval over which the interaction obtains. I am not here offering a metaphysics of causation, and so I take no stand on whether the items related by a causal connection are events, facts, or properties, etc., nor on whether the features in virtue of which such an interaction obtains are property instances, event features, or what have you. Nor am I here committing myself to any view about what differentiates specifically causal interactions from non-causal interactions, whatever such may be. I mean only to be offering a scheme for individuating any given causal interaction from other causal interactions.

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-kind, and similarly their effect features are instances of the same feature-kinds. If e.g., the connection between A and B is licensed only by A instancing property *P* and B instancing property *Q*, and the connection produces only an instance of property *R*, then the interaction between A and B is an instance of the kind of interaction licensed by instances of *P* and *Q*, and productive of instances of *R*. 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.e. a sequence of *n* causal interactions such that the *i*th interaction in the sequence produces a feature which is a licensing feature for the *i*+1th interaction in the sequence, for all $i<n$. I shall say that if an interaction between A and B is part of a sequence of causal interactions constituting a causal trace then the interaction is *in* the trace, and the items A and B are *on* the trace. Similarly, a causal relation which is in the sequence of relations used to individuate a causal process will be said to be *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 simply to *be in*, the process.

With this notion of causal process in mind, 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, $N(P,t')=N(P,t)+\sum_1^n f_i N(P,t)$, where f_i is the frequency of the *i*th process in P from *t* to *t*'. If there are processes leading to deaths of individual bacteria, then $N(P,t')=N(P,t)+\sum_1^n f_i N(P,t)-\sum_1^m g_i N(P,t)$, where g_i represents the frequency of the *i*th process leading to bacterial death, of which there are *m*.

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. $\sum_1^n f_i N(P,t)$, or by limiting the value of the difference between the frequencies of the division producing processes and the death producing processes, i.e. by limiting $\sum_1^n f_i N(P,t)-\sum_1^m g_i N(P,t)$. The obvious way in which an environment can limit the frequency with which a process is instanced is to include a limited number of items of a kind playing some role in the process. 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 $(M'/M)/N(P,t)$. It is possible for an environment to so limit the frequency of a causal process leading to reproduction, and hence to so limit population growth, only if the process includes a relation between some

kind of item in the environment and organisms of the kind comprising the population. So I shall say that a causal process leading to reproductive success, or to death, constitutes an environmental check on population growth just in case that process includes a causal relation between organisms and some kind of item in the environment.

It will not follow merely from the fact that in a given population in a given environment a process constituting an environmental check on population growth *can* be instanced that the environment *is in fact* checking the growth of the population. Prima facie, it might be, for example, that M' is so much greater than M, for our bacteria population in our experimental environment, that the frequency of the mitosis producing process is limited not by the number of sucrose molecules, but rather by the speed with which bacteria of this species can metabolize such molecules. Therefore, 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 those checks are actually operating to constrain population growth. We are now in a position to ask whether there are cases of pure *r*-selection in which such checks are operating.

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 *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 very 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 check on population growth. I disagree.

Consider the two distinct sets of processes by which bacteria of types A and B respectively find, absorb and metabolize nutrients, use some of the resulting energy to find yet more nutrients and store the remainder, and finally undergo cellular mitosis.[1] Since the relevant processes include environmental items, e.g. nutrients, each such processes is an environmental check on population growth. Hence the environment can be said to actually check population growth if the environment in one way or another constrains the frequency with which at least one process in each set is instanced per unit time in the A and B sub-populations respectively. If the environment does so constrain the frequencies of the processes, then if will be false that "... the rate of reproduction of both strains is not limited by environmental factors" (Lennox and Wilson 1994, 70).

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[1] The sets of processes must be distinct because the A set must include at least one process not in the B set. Were there no such process, no phenotypic property of A-type bacteria not also a property of B-type bacteria could play a role in any process in the A set, for any process including a relation licensed by such a feature could not be in the B set. Any phenotypic difference between types A and B would then be causally irrelevant to reproductive success. Consequently types A and B would not have different *r* values, since for any given process producing cellular division, the probability that a given bacterium is affected by an instance of that process would be the same whether the bacterium is of type A or of type B.

The actual frequency of any given A-type process can be no greater than the frequency with which A-type bacteria absorb sufficient nutrients to allow mitosis. That frequency is a function of several things: the speed and efficiency with which A-type bacteria absorb and metabolize nutrients and the time it takes such bacteria to move from a given particle of nutrient to another. This last is a function of the speed with which A-type bacteria navigate through the medium, the search pattern their movement follows, and the spatial distribution of nutrients in the medium. Hence, the expected value of the frequency of the A-type process, and therefore the expected value of the increase in the number of A bacteria per unit time, is a function of the spatial distribution of the nutrients in the medium. Similarly with respect to any arbitrary process which leads to cellular division in B-type bacteria. Whatever the actual spatial distribution of nutrients is, were it different in relevant respects the rate at which the A- and B-type reproductive processes are instanced would be different. Were, e.g., nutrients more closely packed, division times would be shorter; were they more sparsely distributed, the division times would be longer. The distribution of nutrients in the medium is of course a feature of the environment, and hence the environment can be said to actually constrain the frequency of the processes producing mitosis, and hence to actually constrain the rates of reproductive success for the two types of bacteria.

4. Objections Considered. Two objections to the above argument are likely to suggest themselves. An initial challenge to the soundness of the argument goes as follows. If the medium is such that both the A- and B-type bacteria really are reproducing at their

respective *r* values, then the time it takes bacteria to find new particles of nutrients is irrelevant to division times. Environments in which *r* values are realized are supposed to be optimal. Hence, we may presume that if the bacterial population really is subject to pure *r*-selection, the nutrients are distributed as closely as is nomically possible in the medium, e.g. the medium is saturated with sucrose molecules. Since it is not nomically possible for there to be a better distribution of nutrients in the environment, it is not the environment but rather the laws of nature that constrain the rate at which bacteria find new particles of nutrient, and hence there is in fact no environmental check on the rate of reproductive success for either type of bacteria.

The above response, I think, simply misses the point. It is true that, the laws of nature being what they are, no more optimal environment is physically possible, i.e. any environment which would allow higher rates of reproductive success would be super-optimal in the sense of being both better than the actual optimal environment, and, perforce, physically unrealizable in our world. But it does not follow from this that there is no logically possible world in which a distribution of nutrients allowing higher rates of increase is physically possible. Given such a super-optimal distribution, reproductive rates would be higher since the time bacteria spend moving from one nutrient particle to another would be shorter. Therefore, counter-factually, were the actual environment to include a super-optimal rather than merely optimal distribution, the rates of reproductive success would be higher. That the actual environment does not include a super-optimal distribution is a feature of that environment, notwithstanding the fact that

it is a feature that environment is nomically guaranteed to have. Hence, the environment does constrain the rates of reproductive success. The environment allows pure *r*-selection because it has the optimal physically realizable distribution of nutrients in our world. But the environment nonetheless constrains rates of growth because the distribution of nutrients it realizes constrains the frequency with which the processes leading to cellular division are instanced: had, albeit per physical impossibility, the distribution been different in various ways, the relevant processes would have been instanced with a higher frequency.

It might differently be replied that although the environment does constrain rates of reproductive success, it does not constrain either the absolute size of the population as a whole, nor of the relevant sub-populations, nor need it constrain the difference between the respective rates at which those populations increase in size. The first supposition, at least, is correct. More, the difference between environments which constrain population size, those which constrain the rate of population growth, and those which constrain the difference between rates of growth for distinct sub-populations is both theoretically interesting and commonly held to be of explanatory importance.

But in fact, environmental constraints on the rates of sub-population growth are sufficient for the existence of environmental constraints on differences in the rates of sub-population growth. If the features of a common environment can be deployed to explain why the rate of increase for one phenotype is not more than r_1, while rate of

increase for an alternative phenotype is not more than r_2, then those features of the environment explain, in part, why the difference between the rates of reproductive success for the two phenotypes is what it is when they are reproducing, respectively, at r_1 and r_2. So if an environment actually constrains rates of reproductive success for distinct phenotypes, as it does in our bacteria population, it also constrains the differences between the rates of the respective phenotypes. Hence, even if selection requires not merely the former, but the latter as well, the envisaged example will involve selection.

Nor will it do to insist that selection occurs only when environments actually constrain the absolute size of the total population. Should we so insist, there could be no selection on a population below *K* in its environment. So although the envisaged example does not involve constraints on population size, it is not, at least for that reason, disbarred from being a case of selection.

It therefore appears that at least one case of pure *r*-selection will involve an environmental check on population growth, and hence must count as selection. Perhaps there are other cases of pure *r*-selection which do not involve environmental constraints on population growth, though it is hard to imagine such a case. One possibility is for there to be no causal process running from environment to reproduction, i.e. environmental items play no causal role in the production of offspring. But no case of *r*-selection, of which I am aware, is like this: in our world organisms produce offspring

only by exploiting environmental resources. It might otherwise be that though there are such processes, the environmental items which play a causal role in those process are super-optimally distributed in the environment. But there is no reason to think our world instantiates any super-optimal environments; it is therefore unlikely that cases of pure *r*-selection are, in our world, very frequently like this.

5. Environmental Checks and Unified Accounts of Selection. If the above arguments are right, then cases of pure *r*-selection can, after all, involve environmental checks on population growth, and hence can after all count as cases of natural selection. While modern dispositional accounts of selection, Brandon's among them, do fail to differentiate between cases in which the frequencies of the processes leading to reproductive success are constrained by the environment and cases in which such processes are not so constrained, there is no reason to think they are for this reason inadequate. They are inadequate, in our world, if and only if they incorrectly unify cases of the distinct sorts, and that is possible only if cases of both sorts actually occur in our world. The point of the arguments in sections 3 and 4 is precisely that Lennox and Wilson do not give us reason to think that, in our world, cases of frequency change without environmental constraints on population growth ever occur. If the correct domain of application for standard accounts is restricted, our world appears to lie inside rather than outside the bounds of those restrictions.

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. But there are 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 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.

References

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