addendum adds nothing to his theory of the structure of explanation; a subjectivist with respect to explanation could perfectly well use (1)–(3) and add to them the subjectivist (4); the premises are believed to be true. In other words, Hempel’s theory concerning the structure of scientific explanation is open to both objectivist and subjectivist interpretations. Likewise, I think, for my account of experimentation.

2. Richard Lewontin (1985) has argued that, with respect to one of the central questions in population genetics, the neutralist–selectionist debate, gel electrophoretic data are incapable of testing the alternative hypotheses. But these data have been rapidly accumulating over the last twenty years and, as of the early 1980s, are very abundant. Lewontin goes on to suggest that DNA-sequence data, data which are only now becoming available, are the right sort of data to test the relevant hypotheses.

3. I am indebted to many people for comments on earlier versions of this work, especially Janis Antonovics, Richard Burian, and Bob Richardson. I also owe an intellectual debt to Ian Hacking (1983).

10

The coevolution of organism and environment

COAUTHORED WITH JANIS ANTONOVICS*

Can empty niches exist? One answer to this question is too simple, namely, it all depends on what you mean by ‘niche’. If, à la Grinnell and Elton, you think of a niche as an attribute of the environment, that is, a set of biotic and abiotic factors independent of the organisms of interest, then, of course, empty niches can exist. For instance, if a woodpecker’s niche consists of insects living under the bark of dead trees, then the woodpecker niche can exist without woodpeckers. On the other hand, if à la Hutchinson, you think of a niche as an attribute of the population, then, of course, empty niches are conceptual impossibilities.

This answer is only partly right, since there is an important sense in which even on the environmental conception of niche, niches are not independent of the organisms that occupy them. This, we take it, is the point made by Richard Lewontin’s constructor view of environment (Lewontin 1983). According to this view, evolution is not a process of organisms adapting to preexisting environments, but rather a complex dynamical process of reciprocal causal interaction where organisms construct their own environments which in turn selectively affect the genotypic and phenotypic composition of the population. Lewontin explicitly states that we need to model evolution by natural selection by means of a pair of coupled differential equations representing the coevolution (and reciprocal causality) of organism and environment.

\[ \frac{dO}{dt} = f(O, E), \]
\[ \frac{dE}{dt} = g(O, E). \]

(Here \( O \) represents the state, usually the genotypic composition, of the population of organisms of interest, and \( E \) the environment of those organisms.)

* Department of Botany, Duke University.
We think Lewontin has argued persuasively for his position, and much of what follows will support it. But without further development it is not clear how his position could serve as the basis of any research program. There is nothing particularly difficult in working with coupled differential equations provided one knows something about the functional form of $f$ and $g$. Although there is no single function $f$ that holds for all populations in all environments, it is the business of population genetics to construct $f$'s given information about population structure, genetics, and the environment. Thus the form of $f$ is not mysterious, the techniques of discovering various $f$'s are the techniques of population genetics. (None of this is meant to minimize the difficulties of finding an empirically adequate $f$ for any complex real-life situation.) But what about $g$? Without some idea about how to construct $g$'s Lewontin's position remains at best a truth having no consequences for the practice of evolutionary biology. In this paper we will present a method of considerable generality (though not a method that offers any guarantee of success) for constructing $g$'s and will illustrate the dynamics of organism–environment coevolution.

1. Three concepts of environment

To begin to come to grips with organism–environment coevolution we need to first clarify the notion of environment. At first glance this might seem unproblematic; the term ‘environment’ serves to demarcate the internal from the external and so the environment of an organism (or a population) is the totality of factors, both biotic and abiotic, external to the organism (or population). But as we have argued elsewhere, in population biology there are at least three quite disparate ways in which we can measure environmental factors, and corresponding to these different measures, three importantly different conceptions of environment.

One approach is to measure some factor, or set of factors, entirely external to the organisms of interest. If we were interested in a population of some species of grass in a field, for instance, we could measure the concentrations of molybdenum in various parts of the field. Such measurements need not involve the organisms themselves. They measure the external environment. By means of such methods we could discover a pattern of external environmental heterogeneity like that depicted in Figure 1a. More generally, the external environment is the sum total of factors, both biotic and abiotic, external to the organisms of interest. (Later in the paper we will explore just how such factors are external – for now note that they are external in that they can be measured independently of the organisms.) This is, more or less, the environmental conception of niche operative in ecology.

Unfortunately, the external environment is only indirectly related to organismic evolutionary dynamics. There are complicated reasons for this that will be illustrated later, but for now it should suffice to point out that the factor or factors we pick out to measure in the external environment may or may not

Figure 1. Schematic representation of external (1a), ecological (1b), and selective (1c) environments. The horizontal axes represent distance along the same spatial transect. The vertical axes represent: (1a) the amount of some arbitrarily chosen factor, here molybdenum; (1b) the reproductive output of a single genotype, as it contributes to population growth; and (1c) the fitnesses of two or more genotypes. The vertical broken lines in each graph represent where the environments change. Notice how they change at different places, thus showing different scales of heterogeneity.
influence the organisms' survival and reproduction. In our example, the grasses we are studying may or may not respond to different concentrations of molybdenum (the factor we have arbitrarily chosen to measure). If our concern is the environment as experienced by the target organisms, then a more direct way of measuring the environment is to use the organisms themselves as measuring instruments. This has been called the "phytometer" method. (The name "phytometer" was introduced by Clements and Goldsmith in 1924, but the method as we are describing it is quite a recent innovation in ecological genetics pioneered by Antonovics, Turkington, Stratton and a few others. These studies all involve plants, as the name phytometer implies, and it is easiest to envision this sort of study applying to plants, because, as John Harper has put it, "plants stand still and wait to be counted"; however the method is by no means restricted to plants. For heuristic reasons we will use plant examples.)

Suppose we could clone multiple copies of a seed of some genotype. We could then plant these seeds out along a spatial transect in our field and over a generation we could measure each seed's reproductive output. (See Figure 1b.) By this method we measure the ecological environment. (Note that we would want to use multiple copies of a single genotype, rather than seeds of various genotypes, so as not to confound environmental differences with genotypic differences. Alternatively, we could sample the genotypes extant in the field, clone them and plant them out along the transect in numbers proportional to their extant frequencies. We could then average over all genotypes.) The ecological environment reflects those features of the external environment that affect the organisms' contributions to population growth. Because different organisms (or different genotypes) may respond to the external environment differently, it follows that the scale of heterogeneity of the ecological environment depends on the organisms (or genotypes) used as measuring instruments.

If our concern is with the concept of environment as it functions in the theory of natural selection, we must go one step further. Natural selection is essentially comparative. The selective environment is measured in terms of the relative actualized fitnesses of different genotypes across time or space. For example, suppose we clone multiple copies of two genotypes, \( G_1 \) and \( G_2 \), and plant them out along our transect. The result is Figure 1c. The scale of heterogeneity of the selective environment reflects the differential performance of genotypes in different regions or at different times, that is, genotype-environment interactions, and, like the ecological environment, it depends on the organisms (or genotypes) used as measuring instruments.

What is the selective environment? It is just the pattern of relative expected fitnesses (or adaptedness values, see Brandon 1990, Chap. 1) of the relevant types over space or time. Because we cannot directly observe expected fitness values, we measure it by means of recording the patterns of actualized fitnesses of the various types across space or time. But the selective environment is just this pattern of expected fitnesses across our environmental gradient. Similarly, the ecological environment is just the pattern of the reproductive output of a single type (or the weighted average of extant types) across space or time.

It will be useful to compare the relations among these three types of environments. The external environment consists of all factors external to the population of interest. The ecological environment reflects those aspects of the external environment that affect the target organisms' reproductive output. As Figures 1a and 1b illustrate, not all variation in the external environment is reflected in ecological environmental variation. That is, some differences in the external environment make no difference to the organisms of interest.

However, one might think that external environmental heterogeneity was a necessary condition for ecological environmental heterogeneity. But whether this is so depends on whether or not we include conspecific organisms as part of the external environment. This makes a difference in cases of density-dependent and frequency-dependent selection. For instance, one spot on our transect may be a poor spot for a \( G_1 \) seedling because that spot is surrounded by a large number of \( G_1 \) individuals and there is negative frequency-dependent selection. A meter away another \( G_1 \) seedling may do much better because it is surrounded primarily by \( G_2 \)s. This heterogeneity is important in understanding what is going on in our field, but we do not think it should necessarily be considered heterogeneity of the external environment. From a population point of view the external environment contains only factors external to the population of interest. Thus, the number of \( G_1 \) plants in a given area is not part of the external environment. The crucial issue is whether or not the frequency- (or density-) dependent effect is mediated by some external factor or factors. In the case of plants, this is almost certain to be the case. For instance, negative frequency-dependence might be mediated by genotype-specific pathogens in the soil community (see Bever, 1994). In that case, the soil community, and so the external environment, would be different in the area where \( G_1 \)'s are frequent as compared to an area where \( G_2 \)'s are frequent. On the other hand, in cases of behaviorally mediated frequency-dependence, for example, minority mating advantage, we see no necessity in external environmental mediation. Our point is that, conceptually, external environmental heterogeneity is not necessary for ecological environmental
heterogeneity; even though empirically it may, or may not, be true that most ecological environmental heterogeneity reflects external heterogeneity.

Heterogeneity of the selective environment reflects differing relative fitnesses in time or space (genotype–environment interaction in fitness). For the relative fitnesses of multiple types to vary in time or space, the absolute fitness of at least one type must vary. Thus ecological environmental heterogeneity (when measured by a single genotype) is a necessary condition of selective environmental heterogeneity. It is not, however, a sufficient condition. Variation in the ecological environment will not be reflected as variation in the selective environment if all of the competing types respond to the ecological heterogeneity in the same way, that is, if their relative fitnesses do not change.

From the point of view of the theory of natural selection the relevant environment is the selective environment. Selection occurs when differential adaptedness to a common selective environment leads to differential reproductive success. When these differences in adaptedness are inheritable adaptive evolution can occur. But the process of differential reproduction across a selectively heterogeneous region is quite different. For instance, suppose we plant one seed in good soil and another in toxic soil. The first is likely to grow better and produce more seed. But this is not natural selection (we are assuming the plants are not “choosing” their soil); here, rather, the differences in reproductive success are due to environmental differences. It follows from the considerations already discussed that external environmental heterogeneity is neither necessary nor sufficient for selective environmental heterogeneity. It is not necessary because in cases of density- or frequency-dependent selection (not mediated by external environmental factors) changes in the population structure alone can result in a change in selective environments. It is not sufficient because many changes in the external environment either do not affect the organisms at all or do not affect them differentially.

Given that it is the selective environment that is directly relevant to evolution by natural selection, and given that the selective environment is defined in terms of the relative performance of different types (genotypes or phenotypes) of organisms, it follows that from an evolutionary point of view environments have no existence independent of the organisms that occupy them. This is a purely conceptual point. It corresponds exactly to the point alluded to at the very beginning of this paper. If a niche is an attribute of a population (à la Hutchinson), then, and this is purely a conceptual point, niches have no existence independent of the organisms that occupy them. Lewontin’s constructionist view of environments goes well beyond this purely conceptual point. Using the distinctions just made, in the next section we will argue that external environments, although conceptually independent of the organisms that occupy them, are causally linked to, and so not independent of, the organisms that occupy (or better, construct) them.

2. A model of organism–environment coevolution

To illustrate how organism and environment can coevolve, and how, given that they are coevolving, one can empirically get into the system and understand the dynamics of this process, we use a computer model which, although fairly simple, has, in some important respects, a high level of biological realism and generality. We use this model to illustrate and explore how the functions \( g \) and \( f \) might operate to generate an interactive dynamic between genotypes and their environments.

The model is of a population of haploid organisms with two genotypes A and a. They live along a line, and we assume there are eighty sites for them (this number is arbitrary). (Sites number 1 and 80 touch one another so the line comes round into a circle.) Thus the population is limited to eighty individuals at any time. Each site on the line is occupied by an individual, and the external environment at these sites is classified into four categories: 0, 1, 2, and 3. These values reflect the number of A individuals living in the triplet centered on that site in the last generation. In other words, we are assuming that the environment at a site is determined by the genotypes there in the last generation. Thus put in discreet form the function \( g \) is: 

\[
E_{n+1} = (\text{number of As in the triplet neighborhood centered on } n \text{ in generation } t).
\]

(Where \( E_{n+1} \) denotes the external environment at site \( n \) in generation \( t + 1 \).) For example the following distribution of genotypes in generation \( t \) would result in the following external environments for generation \( t + 1 \):

- \( aA A A a A a a a \)  
- \( 2 3 2 2 1 1 0 \)  

We assume that at each site the fitness of an individual is determined by the external environment in the following way:

- Ext. env.: 0 1 2 3
- Fitness a: 1.0 2.0 2.0 4.0
- Fitness A: 4.0 3.0 3.0 1.0

In effect, we have negative frequency-dependent selection with a one-generation time lag. (The reasons for this time lag will be discussed later.)
We assume all individuals survive and that the fitness of an individual determines the number of offspring it produces. These offspring are dispersed according to a dispersal function we control. Dispersal can be either local or global. Global dispersal means an offspring can land anywhere along the line, while local dispersal means the offspring lands either at the parental site or at one of the two nearest neighboring sites. The number of offspring individuals landing at a site for the next generation depends on the fitnesses of the individuals in the previous generation at sites that, according to the dispersal functions, might send offspring to the target site. More than one individual may land at a given site, but since each site supports at most one individual, the offspring that make it to the next generation at a site is chosen with a probability proportional to its frequency among those that have landed at the site. Thus if three As and two As land at a site the probability of A being there for the next generation is 0.60. (This process corresponds to a nonselective density-dependent population regulation.) Then the process starts all over again.

Figure 2 shows the time course of changes in the external, ecological, and selective environments over ten generations. As explained above, in this model the external environment at a site is determined by the number of A individuals in the triplet neighborhood of the site during the last generation. In contrast to Figure 1, in our model the ecological environment at a site is determined by the weighted average of the fitnesses of the two genotypes at that site. In other words, if the external environment at a site is a 2, then the fitness of a at that site would be 2.0 and the fitness of A 3.0. We then weight these fitnesses by the relative frequencies of the genotypes in the whole population. This represents how the “average” genotype would experience that external environment. One consequence of this conception of the ecological environment is that, given our fitness functions, when the gene frequencies are around 0.50, the curve of the ecological environment will be very flat. We will return to this point below. Finally, the selective environment compares the relative fitnesses of A and a. In Figure 2 this is presented as $\log_{10}(\text{fitness A/fitness a})$.

The initial conditions of run presented in Figure 2 were: fitness functions as specified above; local dispersal of both genotypes; uniform initial external environment of 0; and initial frequency of $A = 0.10$. All of the runs of this model generically illustrate how the external environment can evolve as a function of the evolution of the organisms inhabiting it, that is, illustrate the coevolution of organism and environment. More specifically, the following points can be gleaned from Figure 2: (1) How a spatially structured external environment can evolve from one with no spatial structure (recall that the initial external environment was uniformly zero). (2) How external environmental heterogeneity can exist and not be reflected in either the ecological or selective environments (right side of the graph of Generation 2). (3) The ecological environment, which can be thought of as measuring the spatial scale of a species’ or population’s fundamental niche, can be quite uniform while both the selective and external environments are quite variable (Generations 3, 5, and 6). (Notice that the frequency of A in each of these generations is less than 0.40, thus the flat curves are not the artifact of our model mentioned above, viz., flat ecological environment curve regardless of the external environment at gene frequencies near 0.50.) This means that one cannot take ecological homogeneity as a sign of lack of evolutionary action, in particular it may be hiding selective and external heterogeneity which will ultimately feed forward to destroy the ecological homogeneity (as it does by Generation 10). We take this to exemplify an important point for community ecology; namely that if one wants to understand the fate of a given population within a community one cannot treat the population as a genetic black box. (4) An interesting phenomenon occurs in Generations 7, 8, and 9. The spatial structure of the ecological environment stays fairly constant except for the sign of deviations from average fitness. That is, a spot that is slightly worse than average in Generation 7 is better than average in Generation 8 and again worse than average in Generation 9. This should make us wary of evolutionary predictions made without an attempt to understand the coevolutionary dynamics of the relevant organisms and environment.

Figure 3 comes from a run of 120 generations. The initial conditions for this run were: fitness functions as specified above; local dispersal of A, global dispersal of a; uniform initial external environment of 0; and initial frequency of $A = 0.10$. Figure 3 shows the selective, ecological and external environments for Generations 1 and 120, and the external environment for the intermediate Generations 40, 80, and 100. Thus it illustrates how a (locally dispersing) invading genotype, A, can increase the external environmental heterogeneity over time. The external environmental heterogeneity of Generation 1 represents where the initial As happened to land. Notice that the heterogeneity of the external environment actually decreases from Generation 1 to 40. This is because, except for the patch on the left side of the graph, the other As failed to persist. (Recall that the line representing the 80 sites is actually a circle; thus the high points on the right side of the graph in Generations 40 and 80 are just the leftmost edge of the patch of As growing on the left side of the graph.) Also notice that Generation 120 again illustrates a case where the ecological environment is almost completely homogeneous while the selective and external environments are very heterogeneous.
Figure 2. Diagrams showing the time course of changes in the external, ecological, and selective environments over successive generations. The external environment is the plain line, the ecological environment is the line with triangles, and the selective environment is the line with squares. The horizontal axis represents 80 habitat positions where individuals establish. The vertical axis is scaled such that the maximum possible range of values for each of the three types of environments is the same. In contrast to Figure 1b, here the ecological environment is the weighted average of the fitnesses of the two genotypes. The selective environment is presented as \( \log_{10} \) (fitness A/fitness a). \( p \), the relative frequency of A, is shown for each generation.
ecological models (Comins et al. 1992), and it would be interesting to extend the kind of model presented here to examine the effect of spatial substructuring on equilibria under frequency-dependent selection.

Before commenting on the basic implications we want to draw from this model, let us briefly discuss its biological basis. Negative frequency-dependent selection seems to be common in nature. The biological mechanisms of such selection can, presumably, be quite varied. Two mechanisms seem particularly relevant to our model. One is differential resource utilization (see Wilson and Turelli 1986). A possible example of this will be discussed shortly. Another plausibly very common cause of negative frequency dependence is parasitism. In the coevolution of parasite and host, the parasite would tend to adapt to the commonest host genotypes, thus creating negative frequency-dependent selection for host and parasite (May and Anderson 1983; Seger and Hamilton 1988). Two aspects of our model are realistic in the case of parasitism and, we suspect, in most other plausible cases of frequency-dependent selection.

First, our model explicitly represents the spatial structure of frequency-dependent selection. In nature, if selection is frequency-dependent, it is so because of interactions among individuals (trying to mate, garnering resources, fighting off parasites, whatever). These causal interactions are unlikely to occur randomly with respect to space and time. For instance, if the fitness of a grass in a field is frequency-dependent, its fitness will presumably be dependent on the frequencies of types among its surrounding neighbors, not on the frequencies of types in the entire field or on the frequencies in some clump of grasses 20 meters distant.

Second, the one generation time lag in the frequency dependence in our model is realistic for one case that may be very general. That is the case of parasitism where parasites have a much shorter generation time than their hosts. In such a case parasites can effectively adapt to the local distribution of host genotypes during a single host generation. So if host genotype A is locally common in a certain site during host generation t, that site is likely to be filled with parasites particularly adept at exploiting A at the start of generation t + 1. Some investigators now think that this scenario plausibly explains the evolutionary advantage of sexual reproduction—because in such an evolutionary arms race, the host is always better off producing offspring different from itself (Seger and Hamilton 1988).

In the parasitism case, the external environment is the group of parasites at a given place at a given time. We can certainly imagine other scenarios, scenarios involving differential resource utilization, that our model would describe. For instance, perhaps genotypes A and a differ in their physiology such that
A requires nitrogen for growth and maintenance, but a not only does not require nitrogen, it actually produces excess nitrogen which is leached into the surrounding soil. a performs better in nitrogen-poor soils than in nitrogen-rich soils. In this case the external environment is the amount of nitrogen in the soil at a given place and time. Clearly, in both of these cases and others, the external environment, whether the relevant factor is biotic or abiotic, is conceptually independent of the organisms whose evolution we are tracking.

The selective (and ecological) environment is defined in terms of organisms' performance and has no existence apart from those organisms. In contrast, the amount and type of parasites in the soil, or the amount of nitrogen in the soil, is not only conceptually independent of the organisms of interest, it can be measured independently of them. And so from an atemporal, or synchronic point of view, the external environment is independent of the population. But from a diachronic, evolutionary point of view, in our model the external environment is causally linked as both cause and effect of the evolving population. That is, the external environment is the cause of fitness differences in a given generation, but is also the effect of fitness differences in past generations.

This, we think, is the best way to interpret Lewontin's constructionist view of environments. Synchronically speaking, constructed environments have a reality independent of the organisms that inhabit them. Diachronically speaking, they are not independent, rather they are linked as both cause and effect of organic evolution. This point, if true, is not a conceptual truth, but a contingent truth about the way evolution works.

3. How to construct G

The simple model presented above is a model of organism environment coevolution. It is not the only such model in the evolutionary literature. For instance, Kauffman's models of evolving landscapes are of this type (Kauffman 1993). More closely related to our simple model are host parasite coevolutionary models (e.g., those of Hamilton 1980, 1982, 1986), especially those more recent models that take spatial structuring into account and show that such structuring can result in spatial heterogeneity (Comins et al. 1992). A model is, of course, a human invention. Given that we make the organism environment coevolutionary model we can, to return to Lewontin's suggestion, specify the functional form of g. That is not the problem. For example, in our case the environment in subsequent generations is a simple sum of the number of A genotypes in the triplet neighborhood in the previous generation. The problem is, given that the process is occurring in nature, how can we discover the dynamics of environmental evolution? How can we discover the g for empirical systems?

We can think of methodologies as coming in three grades. The best methods are algorithmic and guarantee a correct answer. For instance, the method of long division is algorithmic and, if properly applied, always yields the correct answer. Unfortunately the empirical sciences contain no such methods. At the second grade are methods that are algorithmic, but do not guarantee the correct answer. Such methods are not common, but are not unknown, in empirical science. The best example in biology is phylogeny reconstruction. There are a number of computerized algorithms that will take a given data set and generate a (usually many) tree(s) relating the taxa of the data set. If the tree is thought of as representing the phylogeny of the taxa then it is a hypothesis, which may or may not be true. The method guarantees a tree, an answer, but does not guarantee the truth. At the lowest grade are methods that are neither algorithmic, nor guarantee truth. That is the best sort of method we can offer for empirically constructing g. (Who would have thought we could have done more?) The method of investigation we suggest will generate the data that are seemingly necessary, though not sufficient, for constructing g. The method (the phytometer method) has been discussed in Section 1 (p. 164).

If g is the functional representation of environmental change, why not try to measure it directly? For instance, why not measure the rate at which nitrogen concentrations in the soil change as certain plants grow there? This would not be a useful way to estimate g since, as discussed above, without looking at nitrogen from the organisms' point of view – via phytometer experiments – we have no knowledge of how, if at all, changing nitrogen concentrations affect the evolving population. In other words, to determine the relevance of an external environmental factor we need to see how that factor feeds back onto the evolving population. Then, and only then, can we be confident that the organism's effect on the external environment is of evolutionary significance.

The phytometer method uses organisms as measuring instruments. When used to measure the ecological and/or selective environment, they are as direct a measure as is conceivable. (In contrast, using a plant to measure the amount of nitrogen in the soil would be an indirect measure, like using a canary to measure the amount of oxygen in a mine shaft.) Suppose there is a system in nature where there is negative frequency-dependent selection with a one-generation time lag (Don Stratton's (1992, and unpublished manuscript) phytometer studies of the Erigeron). How would we know it? We could know it plant Erigeron suggest such a system.) How would we know it? We could know it through the appropriate sort of phytometer study. That is, through an empirical uncovering of the patterns of selective heterogeneity and homogeneity through
space and time. These patterns should suffice, if we are at all perceptive, to show negative frequency-dependence with a one-generation time lag.

We cannot claim that phytometer generated data concerning the spatial and temporal patterns of the selective environment will always suffice to lead us to the correct hypothesis about the dynamics of environmental evolution. We can easily invent crazy models which, if true, would generate data that would easily befuddle us. (For example, suppose we modify our model so that the habitat quality is determined by the average number of As in the triplet neighborhood in the last generation, the third from last, and the eleventh from last generation.) Nor can we claim that phytometer data are strictly necessary for empirically uncovering the dynamics of environmental evolution. One can imagine knowing enough about, say, a parasite-host system that one could confidently predict the dynamics of its coevolution without having first observed the phenomenology of its evolution. One can imagine this, but the history of population biology does not lead us to think it very likely.

Our claim is that the phytometer method of investigating patterns of selective environmental heterogeneity and homogeneity, though neither strictly necessary nor sufficient, is the method which promises success, where success is obtainable, in empirically uncovering the dynamics of organism-environment coevolution.

4. Conclusion

We would be hard pressed to claim any originality in making the point that organisms affect their environments which in turn affect the organisms. This point, in some ways, has long been recognized; for instance, it is the basis for the agricultural practice of crop rotation. However, it has been almost completely ignored in evolutionary biology. Lewontin’s (1983, 1978) arguments that we ought to think of evolution by natural selection in terms of organism-environment coevolution have been, unfortunately, of limited use to empirically oriented evolutionists since he offered no hint of any method for the empirical investigation of this dynamic interaction.

In this paper we hope to have accomplished three things. First, we have clarified Lewontin’s constructionist view of environments. We have argued that the dependence of selective environments on the organisms that inhabit them is a conceptual truth, and that the dependence of external environments on their organisms is a contingent fact about the way evolution often works. Second, we have reinforced the arguments of Lewontin (and others) that understanding the dynamics of organism-environment coevolution is of the utmost impor-

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Notes

1. On the fundamental distinction between the environmental conception of ‘niche’ associated with Grinnell and Elton and the populational conception of Hutchinson see Colwell (1992) which contains references to the original literature. For some important differences between Grinnell and Elton see Griesemer (1992).

2. See Antonovics, Ellstrand and Brandon (1988) and Brandon (1990, Chap. 2; 1992).

3. There are two relevantly different sorts of cases here. The first involves environmental factors, e.g., temperature, which are in many ways, e.g., physiologically or behaviorally, relevant to the lives of the organisms of interest, but which do not affect the organisms differentially. The second involves factors, such as the relative positions of the planets, which do not affect the organisms at all. The first sort of factor is the kind of thing often studied by physiological ecologists. Notice that our definition of the external environment does not attempt to differentiate factors that are interesting in some biological context from those utterly devoid of biological relevance. Our interest is in the evolutionary context and our position is that the selective environment is the relevant notion in that context. This does not, of course, imply that the selective environment is the relevant notion of environment for every sort of biological question.


5. The actual quote comes in the context of Harper’s defense of plants as better systems of study for many population biological questions: “plants stand still to be counted and do not have to be trapped, shot, chased or estimated” (Harper 1977, p.9).

6. However, as the model we present shows, when the ecological environment is measured by means of a weighted average of extant genotypes, ecological environmental heterogeneity is not necessary for heterogeneity of the selective environment.

7. Notice that the fitness functions for A and a are not exactly symmetrical. There is no reason to assume exact symmetry, and deviations from symmetry yield slightly more interesting results. Thus we have not assumed symmetry.
8. Hutchinson (1957) introduced this concept. It represents the set of environmental factors that would enable the population to persist indefinitely, i.e., it is the set of factors within which mean fitness is equal to or greater than unity. Thus our measure of the ecological environment can be used as a measure of the spatial and/or temporal location of a population's fundamental niche. For an interesting discussion of how spatial and/or temporal heterogeneity of a species' fundamental niche might affect its evolution see Holt and Gaines (1992).

9. Robert Colwell (1984) makes this point. According to him, "a rose is not a rose is not a rose", i.e., in our terminology, genetically different roses will respond differently to the same external environment.

10. See Mettler and Gregg (1969), pp. 165–71. The point is an obvious one, if both genotypes are fittest when rare, then, everything else being equal, they should settle into intermediate equilibrium frequencies. Interestingly, one of us (RB) checked five standard population genetic textbooks and only Mettler and Gregg mention the point at all. This is surprising given that many would consider the problem of explaining extant variation in natural populations to be the central problem of population genetics.

11. The clearest case of this in nature comes from the work of Edmunds and Alstad (1978, 1981). They have shown that infestation by the black pine leaf scale of Douglas fir and other pine species in western North America increases with the age of the tree, and that is mainly due not to any weakening of the tree with age but to local adaptation of the scale to the individual tree's genotype-specific defenses. For a review of this and other relevant empirical studies see Seger and Hamilton (1988), pp. 187–9. Also relevant here are the studies of Curtis Lively. In one study (Lively 1989) he showed by means of reciprocal cross-infection experiments that a local trematode parasite was more infective to local snails than to snails from a different habitat (different lake). That is, local parasites were adapted to local snail genotypes and did worse on different genotypes. In another study (Lively, Craddock and Vrijenhoek 1990) of minnows it was shown that parasites disproportionally attack the most common genotype.

12. Although hypothetical, this case is not at all biologically unrealistic; many features of it are found in the experiments of Wedin and Tilman (1990). John Beatty and Christopher Horvath independently pointed out this reference.

13. For a good example of just such a study see Bever (1994). This study shows not only the plants' effect on the soil, but also, reciprocally, the effect of the soil on the plants.

14. Laland, Odling-Smee and Feldman (in prep.), starting from a conceptual standpoint similar to ours, develop a two-locus population genetic model of 'niche-construction' where one locus controls the capacity for niche construction which affects the pattern and strength of selection acting at a second locus. This, as far as we know, is the only other modeling activity directed towards the dynamics of organism–environment coevolution. Odling-Smee (1988) gives a more general treatment of Lewontin's arguments.

**Reductionism versus holism versus mechanism**

Go down the hallways of most of any biological laboratory at a research university and ask the local biologists whether they are reductionists or holists. The response, I predict, will vary somewhat among different groups of biologists. Perhaps the vast majority in the cell and molecular wing of the building will happily label themselves reductionists, while in the ecology and evolution wing many more will be willing to be called holists (or some stripe or other). Despite these differences among groups I would be hesitant to bet much money on the outcome for any individual even if I knew his or her group membership. What I would be willing to bet on is that the vast majority of biologists will give some response to the question with considerable definitiveness, perhaps verging on passion. As Philip Kitcher (1984c) puts it:

> The history of biology is marked by continuing opposition between reductionists and anti-reductionists. Reductionism thrives on exploiting the charge that it provides the only alternative to the mushy incoherence of vitalism. Anti-reductionists reply that their opponents have ignored the organismic complexity of nature. (p. 369)

How can this be, how can there be such a disagreement about such a seemingly fundamental issue in so mature and progressive a science as biology?

In this essay I will argue that this longstanding disagreement is based on a confusion—a confusion between reductionism and a doctrine I will call mechanism. Thus, I will argue that the opposition between reductionism and holism is based on a false choice, and that neither should be endorsed. On the

*Two earlier papers of mine (Brandon 1985b, 1985c) dealt with the topics of reductionism, holism and mechanism. The second and third sections of this essay borrow from those two earlier papers.
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