Causal Explanation in Laboratory Ecology: The Case of Competitive Indeterminacy[1]

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1. Introduction

In this paper I contrast two causal explanations of the outcome of a set of laboratory experiments in population ecology conducted by Thomas Park in the 1940s and 1950s. These experiments shed light on the problem of adducing evidence for the operation of competition (see Lloyd 1987 for a recent philosophical discussion) and are central to the group selection controversy because they form the empirical base for experimental studies of group selection conducted over the last 12 years (see Griesemer and Wade 1988 for an analysis).

The experiments investigated competition in laboratory populations of two flour beetle species, *Tribolium confusum* and *Tribolium castaneum*, but the results were more complex and interesting than expected; in addition to demonstrating competition in a laboratory system, they exhibited what Park called "competitive indeterminacy." This phenomenon was of great interest to statisticians, who devised stochastic models to describe its demographic basis (e.g., M.S. Bartlett, P.H. Leslie, and J. Neyman; see Mertz 1972 for references). Another kind of stochastic model, based on the possibility that genetic founder effects occurred prior to the experiments, was developed by I. Michael Lerner to explain Park's results (Lerner and Ho 1961, Lerner and Dempster 1962).

These two kinds of models, demographic and genetic, suggest very different attitudes toward the adequacy of the *Tribolium* system for studies of competition. On the first, the experimental demonstration of competition showed that an operationalized competition theory was adequate for building laboratory models of competition. Competition is explained in such a system by appeal to the agency of the experimenter in bringing about conditions which satisfy requirements of an axiom of competitive exclusion that specifies the possible outcomes of competition. Moreover, the fact that a new phenomenon, competitive indeterminacy, was discovered indicated the fruitfulness of the model for further elaboration and study. The model was also judged fruitful because it stimulated the development of new theory: a stochastic version of competition theory.

The second attitude, however, says that because an alternative cause of the indeterminism can be invoked to explain the results, the laboratory system is a model for

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two different theories (a genetic theory and a competition theory). Therefore, not only is the appeal to the behavior of the system itself an inadequate basis for explaining the novel phenomenon, but the basic claim that the model demonstrates competition is not warranted by appeal to the experimenter's effectiveness as a causal agent. Appeals to agency can only ground causal explanations in cases where the agent's causal efficacy is unproblematic, and the ambiguity of the model in question brings the status of such appeals into doubt. The success of a causal explanation of the outcomes of experiments is thus seen to depend on the adequacy of a theoretical assertion, that a given system is a model for a particular theory, and tacitly, not a model for another theory which identifies different causes (see Giere 1979).

Thus, a conflict over the adequacy of the model for causal explanation *at all* underlies the surface debate about which model (and which invoked cause) *better* explains the phenomenon of competitive indeterminacy. Lerner's genetic founder effect view locates descriptions of the relevant causes in an account of genetic sampling events in the foundation stock from which the experimental materials are drawn, prior to the operation of the experiments. On this view, the stochasticity of outcomes results from a lack of genetic control of these prior events by the experimenter. Additionally, Park's causal explanation is seen to fail because the theory of competition which justifies Park's laboratory system as a model of competition could not exclude the alternative possibility that it was also a model of genetic founder effects. Park's demographic view locates the descriptions of the relevant causes in the workings of the laboratory system, including the control operations of the designer and the biological interactions of the beetles. The stochasticity results from demographic effects of interactions within the model system.

2. Niches and Competitive Exclusion

The notion that each species occupies a characteristic place in the economy of nature, a "niche," was developed in the early part of this century (Grinnell 1904, 1917, 1924, Elton 1927; for historical reviews see Hutchinson 1965, Kingsland 1985, McIntosh 1985). Niche concepts have been variously used to express the distributional, resource utilization, and ethological properties of species. These properties in turn can be investigated in relation to the physical features of the environments in which organisms occur and to the evolutionary processes presumed to produce them.

The niche concept became linked to the theory of competition through the principle of competitive exclusion (Hardin 1960, Kingsland 1985). A number of lines of inquiry suggest that in communities at equilibrium with respect to population numbers, and for which resources are limiting, no two species occupy the same niche (Hutchinson 1965). Categories of evidence for competitive exclusion include: (1) observations of the distributions of related species by field biologists, (2) formal consequences of deterministic mathematical models (e.g., Lotka-Volterra models), and (3) experimental results. The principle is usually taken to imply that if two sufficiently similar species are brought together in a local geographic area, they will compete, either directly by interfering with one another or indirectly by exploiting resources the other species requires, until one of the species populations emigrates, goes extinct, or diverges evolutionarily from the other. With respect to the experiments considered here, this principle serves to *specify* the possible outcomes of systems which satisfy its conditions.

From the 1930s to the 1970s Thomas Park conducted ecological studies of *Tribolium*. His work on competition is summarized in a series of papers called "Experimental Studies of Interspecies Competition" (see, e.g., Park 1948, 1954). In addition to these experimental reports, Park and his collaborators published numerous reviews and theoretical statements regarding the laboratory approach to ecology (see especially Neyman, Park and Scott 1956, Park 1962).

There have been four chronological stages of this laboratory experimental competition research moving from intergenus to interspecies studies and from results in which a determinate outcome was always achieved (a given species always wins) to indeterminism (see Park, Leslie and Mertz 1964 for review). A number of factors were investigated, including climate, initial numbers, food, and presence of parasites. In this paper I consider only two of Park's goals: proof that competition occurs and demonstration of the end-result(s) of that competition (see Park 1954).

The results of Park's work considered here were summarized by Neyman, Park and Scott (1956). This remarkable paper presents a philosophy of laboratory and mathematical model-building, a summary of the experimental work to that point, and a series of stochastic models which explain the experimental results with varying degrees of success. The paper merits careful attention by philosophers.

3. Competitive Indeterminacy

The experiments are based on the following reasoning. If two species are ecologically similar (i.e., can be shown to have the same or similar niche), then the principle of competitive exclusion requires that they cannot coexist. If populations of two such species are put together into a common environment, there are only three possible outcomes: one may emigrate, one may diverge from the other evolutionarily with respect to resources (i.e., come to have a different niche), or one may go extinct. In a laboratory environment where emigration is prevented and the experiment is not conducted long enough for substantial evolution to occur, the assumption that the two species are ecologically similar leads to the expectation that one of the two species will go extinct. Hence, elimination of one species *in an appropriate laboratory context* can be used as an operational criterion for interspecies competition (Park 1954).

The experiments are conducted by placing a number of pairs (one male and one female) of beetles of one, the other, or both species in vials of flour and yeast (which serves as both food supply and habitat), with controlled temperature and humidity, and censused every 30 days for up to 5 years. The census is carried out by filtering the different life stages in different mesh bolting cloth, counting individuals by sex and life stage, and returning them to vials with fresh medium.

The population sizes at each census are recorded and the experiment is continued long enough to witness the extinction of one species (in the mixed- species cultures) or persistence at stable population size (in the single- species cultures). The results of part of one study (Park 1954), in which initial densities are all set at 8 pairs (1 beetle per gram of medium), and a number of climate treatments are arranged, are summarized in the table, next page (after Table I, Neyman et al. 1956).

The single-species cultures show that in all but one climatic treatment (VI), each single-species population persisted long enough (26-62 censuses) to permit the judgment that in the mixed-species populations eliminations are due "not to the direct action of temperature and humidity, but rather to the *new* impact incorporated in the model when the two forms are forced to live together" (Neyman, Park and Scott 1956, pp. 54-55). Differently put, causal explanations of elimination results in mixed-species populations appeal to the experimenter's act of creating conditions realizing a new impact in comparison with controls. It is important that persistence in single-species culture shows not only that controlled variables have no direct effect, but also that there is no systematic effect of known, but uncontrolled factors. Column two of the table shows that climatic conditions (or correlated, uncontrolled factors) are relevant causal factors in the single-species cultures because there is a treatment effect on population numbers; only in the first treatment are the mean densities not significantly different between species (column three).

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Comparison of Single-Species Densities with Species-Elimination Patterns in Mixed Populations

Climate	Single-Species Population		Mixed-Species Population
Treatment	Higher Density / Lower Density	Prob. of equal N's	Usual Winner (% wins) / Occasional Winner (% wins)
I (HW)	con (41) / cast (38)	>5%	cast (100) / con (none)
II (HD)	con (24) / cast (10)	<1%	con (90) / cast (10)
III (TW)	cast (50) / con (33)	<1%	cast (86) / con (14)
IV (TD)	con (30) / cast (19)	<1%	con (87) / cast (13)
V (CW)	cast (45) / con (28)	<1%	con (71) / cast (29)
VI (CD)	con (26) / cast (3)	<1%	con (100) / cast (none)

HW = "hot, wet" = 34 degrees C, 70 % relative humidity
HD = "hot, dry" = 34 degrees C, 30 % relative humidity
TW = "temperate, wet" = 29 degrees C, 70 % relative humidity
TD = "temperate, dry" = 29 degrees C, 30 % relative humidity
CW = "cold, wet" = 24 degrees C, 70 % relative humidity
CD = "cold, dry" = 24 degrees C, 30 % relative humidity

Numbers in parentheses in column two are mean population numbers of the indicated species in single-species culture over the period of the experiment. Numbers in column four are percentage of "wins" in competition in mixed-species culture.

In the mixed-species cultures (column four) the major qualitative conclusion is that one species always persists and the other is always eliminated (total % wins per treatment = 100% of replicates), and Park concluded that this proves competition is operating because: (1) in single-species culture each species can persist under all climatic treatments (except *castaneum* in VI), and (2) the fact of elimination in mixed-species culture holds regardless of treatment. However, there are two patterns of outcome. Either one species is eliminated

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in 100% of replicates (I and VI) or one species is eliminated in a fraction of replicates and the other is eliminated in the rest (II-V). Moreover, within the latter pattern, the species which wins the larger fraction of the time (on top in column four) can either be the same as the species which does better in that climatic treatment in single-species culture (II-IV) or different (V). Park argued that *a priori* one would expect the species favored in single-species control culture would be favored in mixed-species competition, since its performance seems to be affected by treatment, but this expectation is not born out in treatments I and V. Hence, "...competition adds an impact over and above the control performance..." (ibid., p. 56). Park expressed the interest of these results as follows:

> It is evident that there is an element of "empirical indeterminism" among the replicates comprising the competition cultures of II, III, IV, and V. This is to say, the dependence of the survival of a species on the characteristics of its environment appears to have the nature of a chance event, though not a 50-50 event. From this point of view some generality might be gained by considering the survival of a given species always as a chance event with variable probability, P, of survival where, on occasion, P may be unity or zero. The intent of this statement is not to deny or discard the utility of the "analytical approach" in study of causation but, rather, to set the stage for the probabilistic considerations about to follow. (Neyman, Park and Scott 1956, p. 56).

4. Interpretations of Competitive Indeterminacy

Two interpretations of the indeterminacy described above have been elaborated in the biological literature following Park's empirical work. Stochastic competition theory (the analog of deterministic Lotka-Volterra equations) gives conditions under which there is an unstable stationary state and specifies the probability with which one of the species goes extinct in terms of initial population sizes. The alternative outcomes are explained as due to random variation in population sizes with time (Mertz, Cawthon and Park 1976). Since the probabilistic outcomes are consequences of the varying behavior of apparently identical individuals it has been called "demographic stochasticity" (May 1973). This approach also gives approximations for the time to extinction for the losing species and projects zones of determinacy and indeterminacy in a phase space of population sizes as a function of founding population sizes.

An alternative interpretation of the outcome events is the genetic founder effect hypothesis ("genetic stochasticity") formulated by Lerner and his colleagues (Lerner and Ho 1961, Lerner and Dempster 1962, Dawson and Lerner 1962, Dawson and Lerner 1966). This view claims that the identity of the winning species is determined by the genetic characteristics of the founding populations, such that whichever species wins *in a given replicate* is due to a genetic sampling process prior to the competition experiment which results in a genetically superior population of that species.

Mertz, Cawthon and Park (1976) showed that the two views make different predictions about zones of indeterminacy in population phase space for some sets of initial conditions. They showed that the demographic stochasticity hypothesis is upheld in a number of elegant experiments in which both large and small founding populations produced indeterminate outcomes. However, my concern in this paper is not to decide which interpretation is correct, but to examine the conditions under which biologists holding different interpretations will accept an account of the indeterminacy.

Park cautioned that "the functional existence of interspecies competition may be inferred from a body of data when, indeed, the inference either is not warranted or even not true" (1954, p. 177). Competition experiments must compare what species do in isolation with what they do in coassociation in order to support the *ceteris paribus* claim that they are ecologically similar, i.e., homogeneous in their responses to causal factors *other than* those

directly controlled by design of the experimenter. Unobserved ecological differences would, for example, violate competitive exclusion conditions required to justify the claim that "competition between the populations can be suspected as a causal agent" (ibid., p. 178). In this respect, laboratory competition studies differ from laboratory group selection studies where the point is to use the *knowledge* that group selection is operating to examine the relation between known causal and experimental outcome. In the competition studies a *theoretical postulate* is required to structure the experiments and to give a particular expectation about their outcome.

Park's experimental design was such that the only *determinate* outcome, given the competitive exclusion principle and his operationalization, was competition. Indeed, not finding competition to result from Park's experiments would seem to show that the experimenter, not the guiding principle of competitive exclusion, had failed. However, an additional unanticipated property of the outcome events, the identity of the species eliminated in a given replicate, seemed unpredictable on the basis of the experimental set-up. The genetic stochasticity hypothesis would seem to suggest that Park's claim of effective agency in causing competition (by putting similar species together) could not be grounded by appeal to the axiom of competitive exclusion and his agency in structuring his experimental set-up because he was insufficiently aware that his sampling process may have *generated* another causal factor, genetic constitution, not controlled by the experimental design.

In short, Park could not rule out the possibility that his laboratory system was a model for two alternative theories: (stochastic) competition theory and genetic sampling theory. Since Park's objective was to give a causal explanation of the outcome of these experiments, and since causal explanation here depends on appeal to the experimenter's agency in an experimental context, the unanticipated indeterminacy raises the possibility that *unexamined* causes resulting from, rather than those controlled by, the experimenter's agency were responsible for both the unanticipated property (stochastic pattern of eliminations) and the anticipated one (occurrence of elimination in each replicate).

Thus, the problem is to determine which of two reasons explains the indeterminacy: (1) the experimenter failed to understand the causal impact of his agency such that the *relevant* factors were not controlled, or (2) the experimenter failed to fully understand the causal impact of the beetles themselves under experimental conditions. The second sort of failure is the kind biologists hope for: the expectations of the experiment are supported, but new phenomena arise which warrant further investigation. Hence, the model's properties are richer than first anticipated. The first sort of failure is the kind biologists fear: the experiment "succeeds," but for reasons sufficiently unclear that the experimental design provides no grounds for a causal explanation. The genetic stochasticity hypothesis claims a failure of the first type in that the experimenter failed, despite his best intentions, to sample from laboratory stocks at random with respect to genetic properties relevant to competitive ability.

The results of a laboratory experiment in biology are partly under the causal control of the experimenter and partly under the control of the experimental organisms. It is hoped that the outcomes of experiments will be such that a report of experimenter agency in light of an experimental design can serve as an adequate causal explanation (see Griesemer and Wade 1988 for further discussion). In the case of Park's experiments, the design seemed to permit only one outcome event, competitive exclusion, contingent only on Park's agency being causally efficacious in coassociating the two species. (Note that this is not a trivial matter: merely putting members of two species together in a vial may or may not force coassociation.) The additional feature of stochasticity could not be immediately explained as a consequence of putting populations of two species together because: (1) no observations of "demographic" interactions were made and (2) relevant genetic causal factors may not have been controlled. Accounting for indeterminacy became an important explanatory task

for Park because otherwise he could not justify the claim that he had *knowingly* caused, and could therefore explain, competition.

The success of Park's explanation depends on the operation of a well-structured laboratory system in which a set of outcomes can be specified in theoretical terms to support the claim that the model system satisfies a particular theory. The central role of experimentation is to link phenomena producible in the laboratory to causes we at least potentially understand unproblematically, namely those which result from our own agency (Griesemer and Wade 1988).

A causal explanation, on this view, is one which grounds statements of the conditional probabilities of outcome events in terms of claims about the *actual* causal consequences of agency. The outcomes are stated, in Park's case, as probabilities of extinction of one of a pair of competing species, relative to a set of relevant background contexts (controls). It is thus crucial that the actual causal consequences be adequately described with respect to the theory which the system models. The set of actual causal consequences depends on the experimental set-up relevant to the production of the outcomes to be explained. The problem is not in fixing the context so that the laboratory system exhibits causal consequences, but rather in giving an adequate causal justification in terms of the agency necessary to bring about an experiment which serves as a model process. The object of a carefully designed experiment is to put the experimenter in a position to allocate explanatory import to various causes, and if the experimenter is clever, the allocation will be to the only candidate agent, the experimenter.

Notes

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