MODELS OF GROUP SELECTION*

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The key problem in the controversy over group selection is that of defining a criterion of group selection that identifies a distinct causal process that is *irreducible* to the causal process of individual selection. We aim to clarify this problem and to formulate an adequate model of irreducible group selection. We distinguish two types of group selection models, labeling them type I and type II models. Type I models are invoked to explain differences among groups in their respective rates of production of contained *individuals*. Type II models are invoked to explain differences among groups in their respective rates of production of distinct new *groups*. Taking Elliott Sober's model as an exemplar, we argue that although type I models have some biological importance—they force biologists to consider the role of group properties in influencing the fitness of organisms—they fail to identify a distinct group-level causal selection process. Type II models if properly framed, however, do identify a group-level causal selection process that is not reducible to individual selection. We propose such a type II model and apply it to some of the major candidates for group selection.

1. Introduction. The question of whether or not, and if so under what circumstances, group selection occurs has been at the center of considerable controversy for more than two decades, both among biologists (Vrba and Eldredge 1984; Gould 1982a, b; Lewontin 1970; Smith 1976; Williams 1966), and among philosophers of biology (Brandon and Burian 1984; Hull 1978; Sober 1981, 1984; Wimsatt 1980, 1981).

All discussants in the units of selection controversy seem to agree that group selection is to be invoked to explain heritable[1] differences in group

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[1]Heritability is not strictly required for selection and, accordingly, Sober's model of group selection does not require it (for example, Sober 1981, p. 99; 1984, p. 340). We drop the heritability requirement in our general characterization of type I group selection models so that none is ruled out prematurely. For sustained evolutionary change via selection, however, heritability is required. Since our particular model of group selection is used to explain sustained evolutionary change, we do require that the differential group fitness be heritable.

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fitness (differential survival and reproduction) only in those cases that appear not to be adequately explained by the more conventional process of individual selection, but seem to require recognition of special processes acting at the level of groups. According to Sober:

> The definition [of group selection] should allow one to distinguish changes in group properties due to group selection from changes in group properties that are due to processes operating at a lower level of organization. (1981, p. 107)
>
> Group selection should not be invoked, if individual selection suffices. (1984, p. 235)

and Wimsatt:

> If a context-independent component of fitness at a given level is shown to be exhaustively accounted for by an additive component at a lower level, then the putatively upper-level force is merely a summative redescription of lower level forces, not a new force. (1981, p. 151)

Thus, we can identify the task for proposed models of group selection as that of specifying when fitness differences among groups are *irreducible*, in some sense, to selection processes acting at the level of individuals contained within groups. The main source of the ongoing controversy is the lack of an adequate criterion of irreducibility. Without an acceptable criterion, empirical assessments of the importance of group selection in effecting evolutionary change cannot even begin.

Part of the controversy appears to stem from two very different uses of the concept of irreducibility (to individual selection). As Gould notes:

> Unfortunately, the terminology of this area is plagued with a central confusion (some I regret to say abetted by my own previous writings). Terms like 'interdemic selection' or 'species selection' may have been used in the purely descriptive sense, when the sorting out among higher-level individuals may arise solely from natural selection operating upon organisms. Such cases are explained by Darwinian selection, although they are *irreducible to organisms alone*. The same terms have been restricted to cases of higher-level individuals acting as units of selection. Such situations are non-Darwinian and *irreducible on this strong criterion*. (1982a, p. 384)

We agree with Gould that

> Since issues involving the locus of selection are so crucial in evolutionary theory, I suggest that these terms [reducible and irreducible] only be used in the strong and restricted sense. (1982a, p. 384)

Strong irreducibility, as Gould stresses, requires that groups, and not group members (that is, "individuals" as we use that term), be the units of selection. The challenge posed by Gould and other participants in the group selection controversy is to determine when a group is or is not acting as a unit of selection. Our aim in this paper is to meet this challenge by explicating the strong sense of irreducibility to individual selection unambiguously.

2. Models of Group-Selection.

2.1. Group-Individual Fitness versus Group-Group Fitness. To avoid a further ambiguity that often infects the group-selection controversy, we propose to distinguish at the outset two concepts to which the term "group fitness" may refer:

I) Group fitness may refer to the *average fitness*[2] *of organisms* contained within a group (averaging over all individuals in the group), or

II) Group fitness may refer to the *number of entirely new groups* that a given group produces on average.

To distinguish these two, we refer to them as *group-individual* fitness and *group-group* fitness, respectively. (Our use of the terms "group" and "individual" is not to be interpreted as precluding groups behaving as "individuals" in the sense of Hull (1978).[3]) While group-individual fitness refers to a group's ability to produce more individuals of the kind it contains, group-group fitness refers to a group's ability to produce distinct new groups.[4]

Consider for example two groups of beetles; individuals belonging to the first are flightless, but individuals belonging to the second fly. Suppose that the average fitness of flightless beetle individuals is k, and the average fitness of flying beetle individuals is m, where m exceeds k. Then the group-individual fitnesses of the group of flightless and the group of

[2]The fitness of an organism is not to be regarded as its actual reproductive success (for example, number of offspring, or one of many such measures of actual reproductive success). It is to be regarded as the mean number of offspring that would be produced by an organism with a particular genotype over a large number of hypothetical trials. While we do not address the question of the most appropriate measures of fitness here, our arguments and the group selection model we develop are compatible with those generally in use by population biologists.

[3]For Hull (1978) a group *must* play the role of an "individual" if it is to be selected. Our model of group selection is consistent with that requirement.

[4]Group multiplication is to be viewed as the production of one or more discrete new groups. As in the case of organismal fitness, group-group fitness is to be regarded as the mean number of discrete new groups that would be produced by a given group over a large number of hypothetical trials. See note 2.

flying beetles are k and m, respectively, and the two groups differ in group-individual fitness. Suppose, however, that the group of flightless beetles produces 100 distinct new groups of flightless beetles on average, and the group of flying beetles produces only 10 distinct groups of flying beetles, on average. Then the group-group fitnesses of the group of flightless and the group of flying beetles are 100 and 10, respectively, and the two groups differ in group-group fitness. While the group of flightless beetles is *less fit* than the group of flying beetles from the perspective of group-individual fitness, the group of flightless beetles is *more fit* than the group of flying beetles from the perspective of group-group fitness.

2.2. Type I Models versus Type II Models of Group Selection. By distinguishing the two capacities to which "group fitness" can refer, it is possible to identify two approaches that have been taken in developing models of group selection. We refer to these as *type I* and *type II* models of group selection. Type I models of group selection focus upon differences among groups in their respective rates of production of contained individuals—for example, why members of flying beetle groups have a higher reproductive rate than members of flightless beetle groups. Such differential rates are due to type I group selection when they are due to a special (irreducible) group property or process.

2.2.a. Type I Model: Type I group selection occurs when an irreducible group property or process results in group-individual fitness differences among groups.

Accordingly, models of type I group selection seek to specify what is required for an irreducible group property or process to be responsible for differences in group-individual fitness among groups.

Type II models of group selection focus upon differences among groups in their respective rates of production of distinct new groups—for example, why groups of flightless beetles produce distinct new groups at a higher rate than do groups of flying beetles. Such differential rates of group reproduction are due to type II group selection when they are due to a special (irreducible) group property or process.

2.2.b. Type II Model: Type II group selection occurs when an irreducible group property or process results in group-group fitness differences among groups.

Accordingly, models of type II group selection seek to specify what is required for an irreducible group property or process to be responsible for differences in group-group fitness (that is, in average rate of group multiplication) among groups.

Introducing these two new concepts allows us to distinguish explicitly

between the two types of group fitness and thereby distinguish their corresponding models of group selection. This enables us to analyze the two often confused group selection hypotheses separately.[5] We shall first argue, taking Elliott Sober's model as an exemplar, that type I models of group selection describe at most what Gould has termed a *descriptive* concept of irreducibility. That is, while type I group selection may succeed in identifying group properties that cause differential group-individual fitness, we question whether it is a process objectively distinct from individual selection. We shall then develop a type II group selection model that we argue embodies what Gould regards as a strong concept of irreducibility—one that genuinely requires recognition of groups as units of selection.

TYPE I MODELS OF GROUP SELECTION

3. Candidates for a Type I Model of Group Selection. A minimal requirement for type I group selection is a set of groups that differ in group-individual fitness. Thus a minimal requirement for irreducibility in specifying a type I group selection model is that there be differences in group-individual fitness among groups.

> *Candidate 1:* Differential group-individual fitness is due to an irreducible group property whenever groups possessing the property have higher group-individual fitness than groups lacking it.

Under this candidate, our hypothetical beetle case would pass as an example of group selection because groups with the property "all members fly" have higher group-individual fitness than groups having the property "no members fly". But as Williams (1966, p. 16) argues, differences in mean reproductive rates among groups are often merely the summative result of fitnesses of individuals contained within groups: a group whose members each possess property P (a P group), for example, may have a higher group-individual fitness than a group whose members lack P (a not-P group) simply because individuals with P are more fit than individuals lacking P.

A stronger candidate for group selection would require that the group property responsible for the differential in group-individual fitness not be "reducible" to properties that attach to single individuals, but be attributed to the group (or population) as a whole; it must be a property of the group *context*. So a second candidate might be:

[5]Arnold and Fristrup (1982, p. 117) make an analogous distinction. They distinguish "a 'group (treatment) effect on individual fitness,' vs. 'selection between groups as units'" (Arnold and Fristrup 1982, p. 117). Their model of group selection however, framed in terms of analysis of covariance, differs markedly from ours.

Candidate 2: Differential group-individual fitness is due to an irreducible group property whenever it is dependent on (having or lacking) a specific group context.

But as Sober demonstrates (for example, 1984, pp. 270, 274), such context dependencies may merely reflect mathematical correlations between context and fitness values, leaving open the question of their causes. If a model of group selection is to hope to address the issue of causality, it must go beyond the necessary conditions provided by Candidate 2. Sufficiency requires an additional constraint that will avoid labeling as group selection cases where differences in group-individual fitness, though *correlated* with a shared property, are not *caused* by the sharing of the property (that is, the group context).

4. Sober's Model of Type I Group Selection. Explicitly recognizing the need to treat group selection as a causal process, Sober proposes the following model of type I group selection:[6]

> [G]roup selection occurs in a set of populations exactly when there exists some property P such that
>
> 1. Groups vary with respect to whether they have P, and
> 2. There is a common causal influence on those groups that makes it the case that
> 3. Being in a group that has P is a positive [Pareto] causal factor in the survival and reproduction of organisms. (1984, p. 314)

That is, according to Sober,

> Group selection, on the construal defended here, requires that a group property be a positive causal factor in the survival and reproductive success of organisms. The Pareto interpretation of positive causal fac-

[6]Sober makes it clear that he intends his definition to address the issue of the influence of group properties on individuals (that is, the aims of a type I model as distinct from the aims of a type II model). He says, for example:

> . . . the idea of group reproduction standardly used in discussing group selection is that of groups founding numerically distinct colonies. But there is no need to restrict our attention to this process to the exclusion of considering the dynamics of population growth. (Sober 1981, p. 19)
>
> . . . the definition of group selection used here does not require that groups go extinct or found new colonies. (Sober 1984, p. 318)

The final chapter of Sober 1984 briefly considers a type II model.

> tor requires, in turn, that the group property *help at least one individual in the group and not harm any*. (1984, p. 330; emphasis added)[7]

4.1. Clarifying Examples. To unpack Sober's model, consider two examples he discusses; the first, imaginary; the second, real.

(i) Groups with different average heights. Consider a system of populations such that a) each consists of individuals with identical heights, and b) taller individuals have higher individual fitnesses than shorter ones. But suppose an individual's fitness is determined, not by its own height, but by the *average height* of the group it is in.

> Here organisms do better or worse in virtue of the phenotype of the group they belong to. In this case, I would argue, we have a genuine case of group selection. Groups are selected for their average height. (1984, p. 259)

(ii) T-allele in the house mouse. This widely discussed case concerns a group or deme of mice, all of whose males are sterile because they are homozygous for a gene called the *t-allele*.[8] Since mice mate only within their deme, female mice in a deme where *all* males are sterile cannot reproduce either, and the deme becomes extinct. The group property, P, sterility of all males in the deme, guarantees that all members of the group have fitness 0.

> [B]elonging to a group in which all males are homozygous for the *t* allele is a negative causal factor in reproduction. So this is a case of group selection, in the sense of our definition. (1984, p. 317)

Cases *(i)* and *(ii)* share a special type of context dependence:

> Just as in the hypothetical example of selection for groups according to their average height, we see that group selection in the mouse case involves an important sort of *context dependence*: Otherwise identical individuals may have different fitnesses because they live in different

[7]The general view of causality in the model Sober endorses is this:

> The necessary and sufficient condition is simply that a positive causal factor must raise the probability of the effect in at least one causal background context and must not lower it in any (1984, p. 315).

In the case of group selection, the effect is survival and reproduction of group members.

[8]See for example Arnold and Fristrup 1982, Brandon 1982, Lewontin 1970, and Williams 1966. Though this case is often cited as a possible example of selection at three levels (gametic, mouse, and deme), we ignore here the level of gametic selection for the *t*-allele.

> groups, and otherwise dissimilar individuals may have identical fitnesses because they live in the same group. (1984, p. 263)

But slight modifications of the above examples yield cases that would be excluded by Sober's model as cases of group selection. Consider, for instance, a slight modification of the average height example:

(ia). Suppose that the average height, say being in a tall group P, increases the fitness of group members (as before) except for a single member who, if himself tall, would have a higher fitness were he *not* in a tall group. (We might imagine that this one member is more successful at evading a shared predator if in a group of shorter individuals.) The fitness of this single member may still be causally affected by the group's average height, or perhaps by the constellation of heights, but *in a different direction* than the other members. Thus, example *(ia)* exemplifies a case where a single individual's fitness was reduced, not increased, by the group property P. The Pareto interpretation of positive cause that Sober favors (that is, at least one has a fitness increase, and none has a fitness decrease), requires him to deny in such cases that P is either a positive or a negative causal factor. He says about such cases:

> Membership in a group having a given composition is *not* a positive (or a negative) causal factor, because the group phenotype raises some individual's chances of survival and reproduction and lowers those of others. (1984, p. 319)

Because case *(ia)* fails to satisfy the Pareto interpretation in Clause 3 of Sober's model, it fails to be an example of group selection for Sober. Yet, the role played by context in case *(ia)* seems to exemplify the very feature Sober gives as the reason why cases *(i)* and *(ii)* *should* be viewed as group selection. That is, in case *(ia)*, as in case *(i)*, it is the group, and not the individual phenotype, that causes the fitness effect. (See above quotation from Sober 1984, p. 263.) The difference between cases *(i)* and *(ia)* is so slight as compared to this strong similarity, that to suppose they illustrate objectively distinct selection processes seems to run counter to biological intuition.

Conversely, cases *(i)* and *(ii)* can be substantially modified so as to exhibit almost no context-dependent effects on individual fitness, while still passing Sober's definition of group selection.

(iia). Imagine (in a modified version of example *(ii)*) that a group property decreases the fitness of one member of the population, but has no effect on the fitness of any other member. Such a case would satisfy the Pareto interpretation of negative causal factor. And despite the fact that group properties play almost no role, this case would pass as an example

of group selection as decisively as does example *(ii)* under Sober's model (provided Clauses 1 and 2 are also met). For, as Sober makes clear in discussing the *t*-allele example, group selection for him does not require that the fitnesses of all members be affected:

> Homozygote males are unaffected by the sort of group they are in; they have a fitness equal to zero in any case. . . . It suffices simply that each female has her chance of reproducing diminished by belonging to such a group. (1984, pp. 317–318)

Cases like *(iia)*, then, would be formally equivalent to a *t*-allele case (that is, example *(ii)*) where only a single female was present in the deme. While it seems that a case in which all members were affected by a group property warrants being distinguished from a case in which all but one (or a very small number of members) are affected, Sober's model provides no grounds for making such a distinction. Both cases are categorized as group selection. It does not matter how many are affected, as long as those that are affected are influenced in a unidirectional manner.

Even without considering extreme cases like *(iia)* (where only one member is negatively affected, all others are unaffected), Sober's model appears to give counterintuitive results.

(iii). Consider a case where the common cause (affecting a set of groups) is some shared threat to survival; a "predator", or a threat such as cancer or starvation. (Thus, Clause 2 of Sober's model is satisfied).[9] The ability to avoid starvation among members of the U.S. population of 1985, for example, is far better than among members of the Ethiopian population. The group context P might be the context of Ethiopia in 1985, while not-P, the context of the U.S. in 1985. Regardless of the intrinsic properties of an individual, the ability of an Ethiopian individual (a member of a group with P) to avoid starvation would be much lower than that of an American individual (a member of a group with not-P). Such a case appears to satisfy Sober's requirements for group selection because the property P is a negative causal factor in surviving starvation (under the Pareto interpretation). But does such a case really warrant recognition of a special causal process beyond that normally called upon to explain fitness differences among individuals? We suggest that biologists (and probably Sober as well) would tend to interpret the case as a typical example of individual selection in which individuals in their interactions with the

[9]From Sober's examples (for example, 1981, p. 111; 1984, p. 319), it seems clear that he regards sharing a common "predator" or threat to survival as satisfying his requirement in Clause 2 for a common causal influence.

environment are responsible for observed fitnesses. Yet it does not seem that Sober's model as it stands would rule out such cases.[10]

4.2. Descriptive Irreducibility of Type I Models of Group Selection. Though the model proposed by Sober defines a criterion of irreducible type I group selection, it will often result in characterizing cases in a manner that conflicts with biological intuition. Without further grounds for thinking these intuitions misplaced, we raise the question as to why one should accept the particular, and not uncontroversial, notion of causality upon which his model is based.[11] Moreover, there is reason to question whether any notion of how group properties cause differential group-individual fitness can do more than *describe* some special manner in which group properties influence group-individual fitness, rather than characterize an objectively distinct group-level causal selection process.

Our argument is not an indictment of Sober's model, if its intention is to describe a special (that is, unidirectional) way in which individual fitness is influenced by properties of the group. Recognizing the influence of group properties on individual fitness is often important. For example, without recognizing the effect of group context in the t-allele case (example *(ii)*) one would have predicted a higher proportion of t-alleles than actually found (see Lewontin 1970). This is often the basis for characterizing the t-allele case as an example of group selection, as Robert Brandon makes explicit:

> [H]ere group reproductive success cannot be satisfactorily explained in terms of the aggregate of the group members' adaptedness values. That would be the case if we were dealing with groups all of whose members were sterile. But here only the males are sterile. That fact explains the group extinction only when conjoined with other facts about the group structure, such as, females mate only with males from their own group. (Brandon 1982, p. 320)

That knowledge of the mating behavior and group structure was re-

[10]It might be possible to rule out such cases by adding further stipulations to what may be regarded as a "group". But as far as we can tell, Sober has not specified how groups are to be defined. He has emphasized that his definition "does not require a reification of the group; it does not force one to suppose that groups are something above and beyond the interactions of their member individuals and the environment" (Sober 1981, p. 113).

[11]It is possible to interpret a positive causal factor in a manner different from Sober (defined in note 7). On Giere's (1980) causal model, for example, it would be sufficient that the group property P raise the fitnesses of individuals on the *average*, instead of in all cases. (For a defense of Giere's model against an important criticism raised in Sober (1982), see Mayo (1985).) Our point is not that employing Giere's notion rather than the Pareto interpretation Sober favors would result in an adequate model of group selection. It is, rather, to note that Sober's model is tied to a specific notion of "positive causal factor".

quired to explain the t-allele frequency correctly, seems only to mean that the original predictions were based upon an incomplete understanding of the relevant biological interactions, for example, the fact that females mate only with males from their own deme. If the biological context of each organism was correctly understood originally, then the fitness value of each organism could, in principle, have been correctly determined.

In the sense of explicit recognition of the influence of group context upon individual fitness, a model of type I group selection is instructive. And because properties of group context are not properly attributed to individuals, there is a sense in which the *properties* are irreducible to individual properties. But this again leads at most to what Gould refers to as a *descriptive* criterion of irreducibility.[12] The fact that group properties influence individual fitnesses in special ways does not, however, seem to provide grounds for distinguishing type I group selection as a group *process* objectively distinct from the way group properties influence individual fitnesses in cases of ordinary individual selection.

TYPE II MODELS OF GROUP SELECTION

5. Reducible versus Irreducible Type II Models of Group Selection. While the causal processes described by Type I models of group selection do not appear to be strongly irreducible to processes acting at the level of individuals, those that come into the purview of type II models of group selection, we shall argue, often do. In contrast to type I models of group selection, the focus of type II group selection models is not differences among groups in their average individual fitness (that is, *differential group-individual fitness*), but rather differences among groups in average rate of production of distinct new groups (that is, *differential group-group fitness*). As formulated in subsection 2.2.b.:

> *Type II group selection* occurs when an irreducible group property or process results in group-group fitness differences among groups.

And the aim of a type II model is to provide a criterion for the concept of irreducibilty employed.

Many proposed Type II models seem to allow that irreducible group selection may be invoked whenever a group property is responsible for differential group-group fitness. After all, group multiplication is a pro-

[12]If the aim is to arrive at a descriptive sense of group selection, Sober's model would still need to address the examples that to us seem counterintuitive. If we are correct, for instance, that an example like *(ia)* illustrates significant context dependency, then its not passing under Sober's model would suggest that, as a descriptive tool, his model was too restrictive.

cess that happens to groups, not individuals in groups; speciation, for example, happens to species and not species members. As Gould notes:

> [T]rends that are powered by differential origin must often include an important component of species selection. Propensity to speciate is not generally a property of individuals. . . . (1982b, pp. 96–97)

But to invoke species selection for this reason may in fact be at odds with Gould's own demand for a strong concept of irreducible species selection. For, as we shall argue, processes of group multiplication and their causes differ; not all of them identify processes objectively distinct from traditional natural selection acting on individuals within groups. Not all satisfy a strong criterion of irreducibility.

5.1. Reducible Group-Group Fitness. One case where differential group-group fitness is reducible to individual selection is when group multiplication occurs as a direct result of the population size of the group. For instance, in the example of groups with homogeneous heights, (example *(i)*), Sober imagines that once a group reaches a certain size it sends out "excess individuals" as migrants that then found a new group (1984, p. 258). Since tall groups are more fit than short groups, tall groups found more groups than do short groups. Had the tall groups not exceeded the short groups in their group-individual fitness they would not have had a higher group-group fitness (given the stipulated manner in which these groups multiply). Thus, the differential group-group fitness is a direct result of the differential group-individual fitnesses of groups having or lacking tallness.

But this difference in group-individual fitness may itself be due to different things. It may simply be due to individual selection for tallness; or it may be due to a group property like average height, as in Sober's example *(i)*. On these grounds Sober distinguishes the latter case from the former (identifying only the latter as group selection). However, as we have argued above, there seems to be no objective basis for identifying a particular subset of the ways differential group-individual fitness can arise as indicative of a process biologically distinct from ordinary individual selection. A distinction based on the cause of group-individual fitness does not seem to correspond to a distinction between the group and the individual acting as the unit of selection. Thus, whenever differential group-group fitness is the direct effect of differential group-individual fitness (that is, the average individual fitnesses of group members), it will not be irreducible to individual selection in the *strong sense of irreducible*. This suggests the following definition:

Differential group-group fitness is *reducible* (that is, not strongly irreducible) to individual selection if it is directly caused by differential group-individual fitness.

When is differential group-group fitness directly caused by differential group-individual fitness? We have just argued that the above case exemplifies one way in which this can occur, namely:

Differential group-group fitness is directly caused by differential group-individual fitness if the reason why one group, say A, has a higher multiplication rate than another group, say B, is that the average fitness of members of group A is higher than that of group B.

We refer to this as the case of *simple difference in group-individual fitness*.

In contrast, however, consider the following case (real examples of which we shall consider in section 6.1.). Suppose the groups that differ in group-group fitness multiply by sending out migrants that settle in separate locations, but that one group, call it group A, tends to do this much more often than some other group, call it group B. The reason for this difference in group-group fitness, let us imagine, is that such migration events are more advantageous to the survival and reproduction of members of group A, than such events are to members of group B. (Perhaps members of group A, but not of group B, have certain dietary needs that can be satisfied best by plants distributed in a patchy way.) In this case the reason group A has a higher multiplication rate than group B is *not* that group A has a higher group-individual fitness than group B. Suppose that the two types of groups have identical group-individual fitnesses (or perhaps group B has an even higher group-individual fitness than group A.) Nevertheless, it still seems that the differential group-group fitness in such a case is directly caused by differential group-individual fitness, provided this is appropriately qualified. For, as stipulated, *the reason* group A has a higher rate of group multiplication than group B is that the events involved in group multiplication (colonizing migrants), which we may abbreviate as M-events, confer a greater fitness advantage (on average) upon *members* of group A than upon *members* of the group B. As a result, the factors responsible for M-events become more frequent in group A than in group B, causing group A to multiply at a higher rate than group B, on average. Since we shall be referring to this type of case in the remainder of the paper, it will be helpful to have a convenient reference for it. In contrast to the first case, which we denoted as *simple difference in group-individual fitness*, this case may be referred to as an example of a *comparative difference in group-individual fitness benefit*.

An adequate characterization of a reducible difference in group-group fitness should capture both types of cases. We propose the following:

DEFINITION 5.1. Differential group-group fitness is directly caused[13] by differential group-individual fitness if the cause of (or reason for) the higher mean frequency of group multiplication events (M-events) in P groups than in not-P groups, is that *either* of the following hold:

CASE 1. the average fitness of members of a P group is higher than that of a not-P group. (That is, *simple differential group-individual fitness.*)

CASE 2. M-events confer a higher average fitness benefit on members of a P group than those events confer on the average fitness of members of a not-P group. (That is, *comparative difference in group-individual fitness benefit.*)

The reason we consider both cases as being caused by (and so reducible to) group-individual fitness, is that the differential group-group fitness described would not have occurred unless either case 1 or case 2 obtained.[14]

5.2. A Model of Irreducible Type II Group Selection. The interest in type II group selection models arises in explaining a trend, that is, differential representation of a property P across groups, that results because of a corresponding difference in group-group fitness.[15] The above considerations suggest that a successful type II model invoke group selection in explaining a trend only when it results from a case of differential group-group fitness *not* among those classified as reducible by definition 5.1. Since group multiplication may occur in different ways, we add the further requirement that the groups under comparison multiply by the same type or *mode* of group multiplication process, that is, by undergoing M-events.

[13]The relevance of adding "directly" to the causal statements in our model is to rule out factors that may be indirectly related to the effect, perhaps separated by several generations, as well as factors that are merely correlated with the effects. See note 14.

[14]Thus, we are using the notion of cause here as necessary cause. It is meant to distinguish factors and events that are merely necessary conditions for bringing about the differential group-group fitness. Just as oxygen, though a necessary condition of the Chicago fire, say, is not its cause, a minimal level of individual fitness among group members, while a necessary condition for group-group fitness, may not be its cause.

[15]Property P might be any factor (group or individual) used to *identify* the groups of interest that differ in group-group fitness. It need not itself be causally connected to this group-group fitness difference. Only the factors involved in M events need (by definition) be so causally connected, and these may differ from P.

Type II Model for Group Selection. Group selection occurs exactly when

(1) There is a shared mode of group multiplication among the set of groups; label this mode as M.

(2) There is a heritable difference in group-group fitness among a set of groups. (For example, groups with some property P have a higher mean frequency of M-events than groups with not-P.)

(3) The differential group-group fitness is caused by an irreducible group process. (That is, its necessary cause is not differential group-individual fitness in the sense of Case 1 or in the sense of Case 2) as defined in *definition 5.1.* above.

Thus, under our model, the question of whether to invoke group selection centers upon the cause of differential rates of M-events.[16]

5.3. A Model of Species Selection. The model of type II group selection proposed in section 5.2 is intended to be entirely general, and attempts to extend Gould's (1982 a, b) concept of irreducibility in the strong sense to every level in the evolutionary hierarchy. If adequate, our model should succeed in distinguishing reducible from irreducible group selection at any level. The question of whether to invoke *species selection* arises when trying to explain correctly differential group-group fitness among species, as well as resulting trends. Our model translates this question into the specific question of whether the differential group-group fitness is directly caused by differential average fitnesses of individuals within species (that is, by differential species-individual fitnesses), where this is characterized in clause *(3)* of our model. If the cause is differential rates of extinction, then the differential group-group fitness would appear to be reducible under *(3)* Case 1 above, because the extinction of a species results from the death of its members (see Gilinsky 1986). Thus we shall focus our attention, in the case where the groups are species, upon differential rates of species multiplication, that is, *speciation*. Our model of species selection is this:

Species selection occurs among species that speciate by mode M exactly when conditions *(1)* and *(2)* of our model of group selection (in 5.2) hold (where the group now is a species), and in addition

(3) The direct cause of the differential speciation rate (that is, the

[16]Unlike some models, our type II model of group selection does not require that groups be engaged in competition. The precedent for not requiring interaction for selection goes back to Darwin: "Two canine animals, in a time of dearth, may be truly said to struggle with each other which shall get food and live. *But a plant on the edge of a desert is said to struggle for life against the drought . . .*" (Darwin 1872, p. 101; emphasis added).

reason that M-events are more frequent among species A than among species B) is neither that

CASE 1. the average individual fitness of members of species A is higher than that of members of species B (that is, *simple difference in species-individual fitness*), nor that

CASE 2. M-events confer a higher average fitness benefit on members of species A than they confer on members of species B (that is, *comparative difference in species-individual fitness benefit*).[17]

Since the question of whether or not to invoke species selection is a question about the causes of differential speciation rates, it seems clear that answering it requires examining the causes of speciation. Indeed, we shall show, distinguishing among the mechanisms or *modes* of speciation allows us to distinguish differential speciation rates that are reducible from those that are irreducible, and thereby to provide a criterion for species selection.

6. Modes of Speciation and Species Selection.

6.1. Adaptive Speciation. In speciation, a parent species gives rise to a new set of populations with which it is unable to reproduce successfully. Thus speciation may be characterized as the acquisition of *reproductive isolating mechanisms,* that is, mechanisms that prevent interbreeding with members outside the species (Mayr 1970, p. 324). One type of hypothesis to explain the acquisition of isolating mechanisms is that they either *are* properties that rendered individuals in a species more fit than those lacking the properties, thereby resulting in a higher representation of the properties (Dobzhansky 1970; Bush 1975), or that they are pleiotropic *correlates* of such properties (Templeton 1981). Properties that increase individual fitness may be characterized as individual adaptations, and trends

[17]Other models of group selection typically treat Case 1 as reducible to individual selection, but consider Case 2 as genuine species selection (for example, Gould 1982 a, b). The tendency to do so stems from the fact that Case 2 does involve a certain "decoupling" of microevolution and macroevolution. As Vrba (1980, p. 81) points out:

> the theory of how a character becomes genetically fixed as a within-species adaptation clearly cannot on its own explain why a trans-specific trend should evolve with respect to that character.

However, with Vrba, we share the view that this type of decoupling does not require invoking species selection; it is merely the effect of individual adaptations and is reducible under her "Effect Hypothesis" (1980, p. 80), and reducible under our type II model (clause *(3)*, case 2). We differ with Vrba, however, over when invoking genuine species selection is warranted.

that result are caused by the *process of adaptation* of individuals. As Carson (1971, p. 66) points out:

> Evolution proceeds through two major processes, adaptation and speciation. In their most powerful form, both processes must operate in and be subject to, the laws of population genetics. . . . [M]any evolutionists appear to hold the view that there is no essential difference between them and conclude that the conditions which promote adaptation must equally promote speciation.

If they are right, then both adaptation and speciation will be reducible to individual selection and there is no need to invoke irreducible species-selection to explain trends. For, by the very definition of individual adaptation, in cases where speciation is a direct result of individual adaptations—a type of speciation that may be referred to as *adaptive speciation*–differential speciation rates are a direct result of differential species-individual fitness (as defined in 5.1 where the groups are species). This is straightforward when the reason for differential speciation rates is the difference in individual adaptiveness of the species in question, that is, "simple difference in species-individual fitness". The following illustrates how differential rates of adaptive speciation may be caused by a "comparative difference in species-individual fitness benefit", and thus be reducible to individual selection.

6.2. Differential Rates of Adaptive Speciation: An Example. Consider the proposed mechanism of speciation in a host specific maggot species of the genus *Rhagoletis*. The ancestral species, which infested hawthorn trees, gave rise to an apple-infesting descendant a little over 100 years ago (see Bush 1968 and Mayr 1970, p. 262). Bush has proposed the following scenario: host selection in *Rhagoletis* has a genetic basis. A mutation occurred in a locality where apples were available, and caused the mutants to be attracted to the new host. Host plant and mate selection are correlated because males set up and defend a territory on a single host fruit while awaiting the arrival of a female. Since the apple provided suitable nutrition for the mutants, those mutants that sought the apple host gave rise to a population of apple-infesting maggots. The key properties distinguishing the apple race from the hawthorn race, including earlier emergence from the pupa, earlier breeding season, and changed ovipositor length, arose as a result of individual selection. Mutants that possessed these properties conducive to life on the new host were more fit than those that lacked the properties and hence came to dominate in the apple-infesting population. After a few generations two populations were established, one adapted to the original hawthorn host, and a new one adapted to the apple host.

For these two races to evolve into distinct species, they had to acquire reproductive isolating mechanisms. However, these too, according to Bush (1968, pp. 247–248) arose as a consequence of individual selection:

> The evolutionary future of these host races would depend on the interrelationship between gene flow and selection. If selection can successfully eliminate introgressing genes between the two populations, then eventually the races may diverge genetically to a point where they are completely reproductively isolated from one another.

Thus, the factors that inhibit gene flow between the two races, and lead to reproductive isolation (speciation) are individual adaptations. Apple maggots that mate with hawthorn maggots may give rise to offspring that are *less fit* than those that mate with members of their own race (whose offspring will be born on a host with optimally nutritious fruit). Thus the process of speciation just described *is* a process of adaptation.

Assuming that speciation in the apple maggot did occur by the mechanism, M, as proposed by Bush, consider how our model of *species selection* applies to this case.[18] Suppose that species of the genus *Rhagoletis* with populations of mutants (P) (where mutation causes a change in host) have higher rates of speciation than species of the genus *Rhagoletis* with fewer or no such mutant populations (not-P); that is, suppose that P species have higher group-group fitnesses than not-P species. The M-events that lead to reproductive isolation in P species are also events and processes that provide a selective advantage to individual apple mutants. Such events are *selected for*, to use Sober's (1984) terminology. Were apple mutants to remain on the hawthorn host their offspring would be less fit. Among nonmutants, in contrast, M-events (for example, shifting to a new host) would be selected *against*, because without the mutation for shift in host preference, it would be disadvantageous to undergo a shift in host preference. Hence the cause of the higher frequency of M-events in P species as compared to not-P species is that M-events confer a greater fitness benefit in P species than in not-P species. Since the acquisition of reproductive isolating mechanisms (that is, speciation) among host specific maggots of this genus requires M-events, it follows (from Case 2 of our definition in 5.1) that the cause of the differential speciation rates between P and not-P species is *reducible* to individual selection.

[18]We use the *Rhagoletis* example to illustrate how our model makes a conceptual distinction between cases such as that discussed by Bush (where speciation is *adaptive* and *sympatric*) and cases of *founder speciation* to be discussed in section 6.4. In neither case is it our intention to pass judgment on the empirical validity of the hypothesized speciation mechanisms. Cases such as the *Rhagoletis*, however, are considered by Mayr (1963, pp. 262–263) to provide the strongest evidence of sympatric speciation which, in contrast to founder speciation, does not require geographic isolation.

Thus, if differential speciation rates are directly caused by the greater group-individual fitness benefit conferred by events responsible for speciation (M-events) in P species than in not-P species, then the differential speciation rates are directly caused by (and so reducible to) individual selection. More generally, if the shared mode of speciation is *adaptive speciation*, then condition *(3)* of our model for species selection will *not* be satisfied. Such cases fall under "comparative difference in group-individual fitness benefit", and thus are *reducible* to individual selection under our model.

6.3. Nonadaptive Speciation; Founder Speciation. One mechanism of speciation (acquiring reproducive isolation) that would satisfy our model of species selection is speciation via what is termed the "founder principle" (as originally proposed by Mayr 1954). The process of founder speciation begins with the origin of a small founder population by the chance transfer of a few individuals from the main population. These individuals represent a statistical sample of the genetic variability of the parent population. If the founder population remains isolated from the parent population, that is, if gene flow between founder population and parental population is prevented, then genetic transformations may occur in the isolate that yield reproductive isolating mechanisms (that is, yield speciation). Thus the key features of founder speciation are the *formation and maintenance of an isolate* and *genetic transformations* that occur in the isolate.

Under theories of founder speciation, a considerable amount of the genetic transformation that occurs is a consequence of the properties of the founder individuals that by chance form the isolate. Since the frequencies of genes in the isolate are at its inception different from their frequencies in the parental population, genotypes that comprise the founder population experience an instantaneous change of genetic environment, a change that may change the selective values of all genes at once (Mayr 1954).[19] Furthermore, continued inbreeding in the small isolate produces gene combinations that are very different from combinations that occur in the outbreeding parental population. These genetic changes may cause instabilities that lead to a breakup of the old co-adapted gene system, a breakup Mayr terms a "genetic revolution". If the genetically changed isolate manages to survive the instability associated with the genetic revolution, and a reduced level of competition and predation enables it to develop a new stable genetic system, it is highly unlikely that the new system would

[19]All of the theories of founder speciation discussed in Carson and Templeton (1984) involve a change of genetic environment, but each accords it a different degree of importance. Mayr seems to accord it a central role in the speciation process.

be compatible with the old one; that is, it would have developed one or more reproductive isolating mechanisms that render the descendant population a new species. While many properties of the new species will result from subsequent individual adaptations, the changes that would be expected to be the crucial ones with regard to the acquisition of reproductive isolating mechanisms are not these; rather, they are the genetic changes that immediately follow the founding event. As Carson (1971, p. 68) stresses: "[t]hese changes, not adaptive ones, represent the really critical point in the process of formation of the new species. Chance, not natural selection, plays a principle role in the initial arrival of the founder" and in the subsequent genetic tranformation. Therefore, "the key genetic shifts leading to the crucial species differences may be non-adaptive" (Carson 1971, p. 68). The new population would reflect changes that were

> initially unrelated to the forging of new adaptations and might indeed represent a basically non-adaptive phase of evolutionary change. Such an abrupt series of events might be an initial genetic move in the direction of speciation essentially divorced from the monitoring effects of natural selection. (Carson 1971, p. 60)

6.4. Differential Rates of Founder Speciation and Species Selection. We have followed Carson in hypothesizing a distinction between the events involved in founder speciation and those involved in natural selection on species members. The principal novelty that we propose, is to link this aspect of founder speciation to the process of irreducible species selection.

If speciation is by the mechanism of founder speciation, then the *M*-events required for speciation would be the events involved in the establishment and maintenance of isolates that result in the evolution of reproductive isolating mechanisms (that is, speciation). At first glance, the founding population appears to play a role in speciation analogous to that of the maggot mutant; both diverge from the main population giving rise to a new species. But the reason for the inhibited gene flow between the maggot mutants and the parent population was the fitness advantage it conferred upon the mutants; that is, the changes in the mutant colonies just *are* individual adaptations. But unlike the maggot case, the *M*-events that result in founder speciation (of the sort described by Carson) are not individual adaptations. Hence the reason for a difference in speciation rates (*M*-events) among species will *not* be that *M*-events confer a greater fitness benefit to the higher speciator. Therefore, it seems that the cause of differential rates of founder speciation is not reducible to individual selection in the sense of *comparative difference* in individual fitness ben-

efit (Clause *(3)* Case 2 of our model). But is it reducible to a *simple difference* in species-individual fitness (Clause *(3)* Case 1)? To argue that it need not be, it must be shown that the reason one species, say a *P* species, has a higher rate of founder speciation than another species, say not-*P*, is *not* that the former has a higher species-individual fitness than the latter. Such an argument follows from consideration of the conditions that have been substantiated empirically for founder speciation:

(I) If colonization were by multiple representatives from the parental population, that is, if there were *free gene flow*, then the difference between ancestral and descendant populations is likely not to be greater than that expected in subspecific differentiation.
(II) (converse of (I)): If colonization is via a few founders and if there is *little or no gene flow* from the parental population, then genetic reconstitution and thereby acquisition of reproductive isolating mechanisms would be expected (assuming the isolate survives at all).

Correspondingly, the higher rates of founder speciation (that is, higher incidence of *M*-events) in *P* species as compared to not-*P* species are directly due to genetic transformations associated with small population size and low or no gene flow in *P* species, and high gene flow in not-*P* species. And it is easy to imagine how this difference in rate of *M*-events could result *even if the P species did not have a higher species-individual fitness than the not-P species*.

6.5. Example: Planktotrophic versus Nonplanktotrophic Larvae. Consider a case where there is good evidence for (I) and (II), that is, for founder speciation as the mode of speciation of a group of species. There are two general modes of larval development in snails, planktotrophic and nonplanktotrophic. Planktotrophic larvae feed upon microorganisms in the water column and spend a prolonged period of time developing there before settling to the bottom where they live as adult snails. Nonplanktotrophic larvae do not feed in the water column and, if they spend any time there at all, the time is usually short. In general, snails that develop from nonplanktotrophic larvae have a lower capacity for dispersal than snails that develop from planktotrophic larvae (Jablonski and Lutz 1980). Hansen (1978, 1982) was able to document in several groups of snails evolutionary trends toward increased representation of species whose individuals have nonplanktotrophic larvae as compared to those whose individuals have planktotrophic larvae. He suggests that the reason for the trend may have been higher rates of speciation among nonplanktotrophic

species.[20] The reason for the differential seems to be that low dispersal capacity engenders a higher probability that founding populations (isolates) comprised of small numbers of individuals will arise, and that they will be maintained by low or no gene flow from the parental population. Thus *M*-events, the events needed for acquiring reproductive isolation in the isolate (that is, for founder speciation), occur far more often among nonplanktotrophic species (*P* species) than among the planktotrophic species (not-*P* species).

However, changes in the individual fitnesses of planktotrophic snails do not alter the fact that the antecedent of (I) holds (high gene flow and thus rare origin of *M*-events). Similarly, changes in the individual fitnesses of nonplanktotrophic snails do not alter the fact that the antecedent of (II) holds (no or low gene flow and comparatively common occurrence of *M*-events). Thus, we can argue as follows:

Higher speciation frequencies in nonplanktotrophic species versus planktotrophic species are due to comparatively low or no gene flow between isolates and the main population (*M*-events comparatively frequent) in the former, but high gene flow between isolates and the main population (*M*-events comparatively rare) in the latter. And this would be the case even if nonplanktotrophic species had a *lower* species-individual fitness than planktotrophic species. Thus, in this case, the higher speciation frequencies in nonplanktotrophic species versus planktotrophic species are caused *neither* by a greater group-individual fitness in nonplanktotrophic species than in planktotrophic species (simple differential group-individual fitness) *nor* by a greater group-individual fitness benefit conferred by *M*-events in nonplanktotrophic species than in planktotrophic species (comparative difference in group-individual fitness benefit). From Definition 5.1, it follows that higher speciation rates in nonplanktotrophic species than in planktotrophic species are *not* directly caused by differential fitnesses of snails of the two types of species (that is, they are not *not* due to differential species-individual fitnesses of nonplanktotrophic versus planktotrophic species). Therefore (since clauses (1) and (2) of our model of species selection in 5.3 hold), our model indicates the trend Hansen observed is caused by irreducible speciation and thus by *species selection*.

More generally, we may conclude that if the shared mode of speciation among species is founder speciation, then differential speciation rates among

[20]Hansen (1982) also suggests other possible explanations. One of these is that, perhaps, species with planktotrophic larvae tend to convert to species with nonplanktotrophic larvae in association with events of speciation. We are assuming, for purposes of this example, that such conversion from one larval type to another does not occur. Again, as indicated in note 18, our interest is in making the correct *conceptual* distinction and not in passing judgment on the empirical adequacy of proposed speciation mechanisms in given cases.

the species in question may not be directly caused by individual selection in either the sense of Case 1 or Case 2 of our model, but alternatively according to our model, are caused by *species selection*.

7. Conclusions. The literature on group selection is large and complex, but it can be reduced, we have argued, to the study of two principle concepts. One concept involves explaining differences among groups (however defined) in their respective rates of production of contained individuals. We refer to models that purport to explain this phenomenon as *type I* models of group selection. Though such models are of interest because they point to the need to recognize how group properties may influence the fitnesses of individuals within groups, we conclude that type I group selection models do not identify a causal group-level selection process distinct from individual selection, though most such models have this as their principal aim.

The second concept of group selection involves explaining differences among groups in their respective rates of production of distinct new *groups* (group-group fitness). We refer to models that purport to explain this phenomenon as *type II* models of group selection. We conclude that for proper explanation of the causes of differential rates of group-group fitness, a distinct group-level selection process must be invoked under certain circumstances. However, not all cases require recognition of a distinct group-level selection process, because differences among groups in their respective group-group fitnesses may be *reducible* to the process of individual selection. For strongly *irreducible* type II group selection, differences in group multiplication rates must not themselves be directly caused by differential fitnesses of individuals, in the senses we have defined. Thus, recognition of real instances of group selection is contingent upon careful analysis of the causes of group multiplication. We argued that if the mode of speciation is adaptive speciation, then the cause of the difference in speciation rates will be reducible to individual selection; while if the mode is founder speciation, the cause of the difference in speciation rates may require invoking species selection. Since, if we are correct, the importance of group selection in evolution depends in large part upon whether processes of group multiplication are directly caused by individual selection, further research into the causes of group multiplication, particularly speciation, is needed.

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