Positive Heuristics in Evolutionary Biology

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1 INTRODUCTION
Most philosophical discussions of biology traditionally concentrate on the products of biological research. For example, the structure of biological laws and theories is often compared to paradigmatic cases, usually in physics. The results are often rather embarrassing for the biologist (Smart [1963], Manser [1965], Peters [1976], or the more sympathetic Beckner [1959]), though this seems to be changing as more carefully studied case histories are conducted (Ruse [1973], [1977], Hull [1974], Wasserman [1978]). Such discussions are usually concerned with products of science (i.e., laws, theories, explanations) rather than the process of scientific change. Given the recent concern in philosophy of science with scientific change (Kuhn [1970], Lakatos [1970], Feyerabend [1975]), it is particularly instructive to focus on the growth of one biological field to see if it can be characterised in a philosophically respectable way as scientific or 'rational'. It will be argued here that the area of biology known as population genetics can be historically reconstructed according to Lakatos's model of 'scientific research programmes' [1970] in a natural manner.

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1 Some notable exceptions are Hull [1973], Green [1971], Lee [1969] and especially Provine [1977]. Provine's excellent article stimulated the work presented here.
Population genetics is the cornerstone of modern evolutionary biology\(^1\) (see, for example, Simpson [1944], Ruse [1973], Wilson [1975] or Roughgarden [1979]). Initially, the incorporation of genetics, especially population genetics, allowed evolutionary biology to grow and develop. This incorporation of genetic principles occurred early in this century with the celebrated 'evolutionary synthesis' effected mainly with pencil and paper by Ronald Fisher, J. B. S. Haldane and Sewall Wright. This synthesis of Darwinism and modern genetics resulted in the Neo-Darwinian or Synthetic Theory of Evolution. The development of mathematical genetic models (the so-called 'beanbag' genetic models to be discussed in 2d) helped Darwin's theory overcome the original anomalies which it faced (Provine [1971], [1977]). These anomalies were so blatant in the latter half of the nineteenth century that a strict Popperian would have claimed the theory to be refuted (see, for example, Lee [1969]). Such a theoretical feat is, of course, the essence of the 'positive heuristic' in science: to turn

... one counter-instance after another into corroborating instances, primarily by overthrowing the original observational theories in the light of which this 'contrary evidence' was established (Lakatos [1970], p. 133).

More recently there has been a shift in population genetics to incorporate ecological models and principles. This shift began in the late 1960s with the synthesis of population genetics, population ecology and evolutionary biology into 'population biology'. This synthesis is still incomplete and research in this area continues to intensify. It will be shown below that the same kind of models which guided the celebrated 'evolutionary synthesis' of the 1920s function in a similar manner today in the developing synthesis of population biology. Demonstrating this theoretical continuity and growth constitutes the bulk of this essay.

2 THE POPULATION GENETICS RESEARCH PROGRAMME

The subject of Lakatos's model is not a theory but a succession of theories which, because of some continuity, form a research programme. Research

\(^1\) By the term 'evolutionary biology' or 'evolutionary theory' I want to restrict my attention to those fields of biology concerned with studying the mechanisms of evolution. Dobzhansky ([1970], p. 28) states 'as the study of evolution proceeded, two main approaches were employed. The first concentrated on unravelling actual evolutionary histories, that is, phylogenies of various groups of animals and plants. The methods used are those of systematics, comparative anatomy, comparative embryology, and especially paleontology... The second approach emphasised studies of the mechanisms that bring evolution about, of causal rather than historical aspects. Genetics, especially population genetics and ecological genetics, has supplied the basic concepts and the experimental as well as observational methods.'
programmes are characterised by two heuristics, the negative and positive, which provide for continuity within the programme. The negative heuristic characterises the hard core of the research programme. This core is irrefutable by the methodological 'decision' of the scientists who work within the programme. This decision to 'accept' the hard core is not necessarily (and usually in fact is not) a conscious decision on the part of the individual scientist, but rather the hard core exists implicitly in all he or she does. Acceptance arises more from a process of indoctrination into a field rather than from any rational decision. The scientist does not attempt to establish or refute the hard core since the negative heuristic discourages this path of research. Rather scientific investigation proceeds by building a protective belt around the hard core. This protective belt consists of subsidiary hypotheses which buffer the hard core from refutations. The construction of this protective belt is guided by the positive heuristic of the research programme which provides a direction for research within the programme.

(a) The Hard Core

The basic elements of Lakatos's model are all clearly identifiable within the population genetics research programme. For the population geneticist, the common denominator of all evolutionary forces is their effects on gene frequencies. In other words, gene frequency changes are evolution. This proposition, the hard core of population genetics, is best summarised by Sewall Wright in the conclusion to volume II of his treatise (Wright [1969], p. 472):

... the species is thought of as located at a point in gene frequency space. ... Evolution consists of movement in this space.

This point of view is the basis of the population genetics approach to evolution. This is as true today as it was during the synthesis of the 1920s and 30s.

While gene frequency changes are sufficient for evolution (genetic drift of neutral alleles), several authors have pointed out that they are not theoretically necessary. For example, Brandon [1978] notes that genotypic changes can logically occur without gene frequency changes.\(^1\) Based on this and other considerations of theoretical adequacy, Brandon ([1978], p. 107) offers a definition of evolution 'as the expansion of similarity

\(^1\) Consider for example, two loci each with two alleles \((A, A' \text{ and } B, B')\) in equal frequency. Assume also that the population is in linkage equilibrium. Letting \(p\) be the frequency of \(A\) and \(q\) be the frequency of \(B\) we have \(p = q = 0.5\). Assume selection operates equally against gametes \(AB\) and \(A'B'\), due to some epistatic interactions. Evolution will result in a decrease in frequency of these gametes but with no change in gene frequency.
classes of genotypes over time'. While I agree with Brandon that this definition is more precise theoretically than the gene frequency notion, I feel it contributes little, if anything, to capturing the historical development of the Synthetic Theory. The two important characteristics of the Synthetic Theory concern the genetical and statistical basis of evolution. The gene frequency approach captures both and does, I feel, accurately portray the conceptual framework within which population geneticists have worked.

I should stress that my concern in this paper is primarily to identify those concepts which have been historically and conceptually operative and less with their theoretical adequacy. I do feel the maxim 'gene frequency changes are evolution' captures in essence the population genetics approach as well as providing the basis of a formal framework within which population geneticists as well as other evolutionary biologists work. I claim this in full knowledge of its theoretical inadequacies.

Another criticism of the gene frequency approach stems from its mistaken equivalence with the single locus, gene frequency approach of 'beanbag genetics', which I will discuss in detail shortly (Brandon [1978]; Mayr [1977]). Single locus theory, or beanbag genetics, is only one aspect of the gene frequency approach, albeit it has proved to be the most operationally useful aspect, as I hope to demonstrate. Single locus, multi-locus, quantitative inheritance and even the phenotypic models all rely equally on the gene frequency approach to evolution. The theoretical adequacy of the single locus approach is, indeed, questionable as Lewontin [1974] has stressed. However, its usefulness as a heuristic is another matter. As I will develop in more detail shortly, beanbag genetics has functioned as a strong positive heuristic in evolutionary biology. Although this heuristic power of beanbag genetics depends upon the hard core, it is a mistake to confuse the two.

Recently there has been some questioning, especially among students of macroevolution, of the gene frequency approach to evolution. Consequently, I would like to broaden the scope of the analysis beyond population genetics proper and discuss the evolutionary generality of the population genetics hard core as identified above.

Some of the problems involved in pushing for the generality of the population geneticist's approach to evolution surface when definitions of evolution are advanced. I should note, however, that the hard core need not appear explicitly in the definitions of a field. Indeed, definitions are more likely to be concerned with theoretical adequacy (as viewed from current methodologies) rather than any underlying thread of historical and conceptual continuity. However, one may at least get some insight
into the basic issues and presuppositions of a field by examining the various definitions offered.

The traditional textbook definition is usually very similar to the hard core of population genetics identified above.\textsuperscript{1} Some authors are careful to include other genetic changes besides changes in the frequency of genes. For example, Eldredge and Gould ([1977], p. 26) state that

Evolutionary change is the modification through time of genes and gene frequencies.

This move to include other genetic changes within the hard core is certainly more realistic. After all, many plants have speciated via polyploidy. Likewise, in addition to gene frequency and chromosomal changes, White ([1978], p. 8) identifies two other categories of genetic change as being important in speciation: changes in the amount of DNA and changes in the composition and amount of satellite DNA's. Thus if one's interest is speciation, the gene frequency approach appears, on the surface, to be inadequate.

This is especially disconcerting since there can be no doubt that the origin of species is one of the most central problems of evolutionary biology. 'To Darwin, evolution was synonymous with the origin of species' (Dobzhansky et al. [1979], p. 7). Yet in an often quoted passage Lewontin ([1974], p. 12) states,

While population genetics has a great deal to say about changes or stability of the frequencies of genes in populations and about the rate of divergence of gene frequencies in populations partly or wholly isolated from each other, it has contributed little to our understanding of speciation . . .

For these reasons, Dobzhansky et al. ([1979], p. 8) feel (Eldredge and Gould would certainly agree), that definitions formulated solely in terms of gene frequencies 'neglect the transspecific aspects of evolution'. Therefore they (ibid.) augment their definition to include all genetic changes along with some comments concerning adaptation.

Likewise Mayr ([1977], p. 45) refers to the gene frequency approach as 'reductionistic' and claims that it

is inappropriate and misleading, particularly for three reasons:

(1) It places its main emphasis on a by-product of evolutionary change rather than on the evolutionary events themselves which are effects of selection pressures on the phenotype, and

(2) It treats purely stochastic phenomena on an equal footing with genic changes

\textsuperscript{1} For example, Futuyma ([1979], p. 7) states 'biological evolution can be defined more precisely as any change from one generation to the next in the proportion of different genes.'
caused by natural selection, and

(3) It implies that genes rather than individuals are the primary target of natural selection.

Mayr’s first criticism is itself misleading for he has abstracted out only part of what is usually a circular, cause-effect loop. Selection pressures on a trait (gene) will usually depend upon, among other things, the frequency (and/or density) of the trait (gene) in the population. Consequently, whether the traits and the ensuing selection are regarded as effects of the genes (being in a certain frequency),¹ or the genes (being in a certain frequency) are regarded as effects of selection on the traits, is, I think, really immaterial.

In regards to Mayr’s third criticism, it is simply not the case that if gene frequency changes are the common denominator of evolution, selection must operate at the genic level. Genes come packaged in different ways depending upon the population structure. There is no necessary relationship between the units perceived by selection and the fact that selection (at whatever level) is reflected in the frequencies of genes. It is, however, interesting to note that by taking a “gene’s eye” perspective of evolution, population genetics has revolutionised the theory of evolution of behaviour (Hamilton [1964], Wilson [1975], Dawkins [1976], see section 3(e)).

Mayr’s second criticism concerning adaptation is shared by other workers. It is often objected that gene frequency changes are only part of evolution and that adaptation is as central to evolutionary biology as are gene frequency changes. While it is true that the fascinating array of adapted life forms often serves as motivation for the study of evolution (it did for Darwin), adaptation is not a necessary consequence of evolution. There are a variety of (theoretically and empirically) well documented gene frequency dynamics which decrease adaptation, and these dynamics are certainly part of evolution. A central component of most evolutionary models is $W$ the population fitness or state of adaptation of the population (see equation [2] below). Since $W$ is a function of the gene frequencies (e.g., [3]), one of the first questions asked of such a model is whether the gene frequency dynamics (e.g., equation [5]) act to increase or decrease $W$. Often the gene frequency dynamics don’t maximize $W$ and sometimes they decrease it. In addition if adaptation were an essential part of evolution’s hard core, certainly no evolutionary biologist could seriously entertain a neutralist theory of evolution. Yet they do, and the neutralist theory is an integral part of modern evolutionary biology. These considerations

¹ Here the cause being referred to is the frequency of the gene itself and not the fact that the gene is expressed as (and hence causes) the trait.
show that while gene frequency changes are an essential part of any evolutionary question (at least implicitly), adaptation is not. While I agree that adaptation is the most interesting result of gene frequency changes, it is not a necessary result. For these reasons, I feel it is best to put the process of adaptation, along with the other various effects and causes of gene frequency dynamics, in the protective belt and not the hard core.

A related line of attack on the generality of the gene frequency approach comes from the work of Eldredge and Gould ([1972] and Gould and Eldredge [1977]) and Stanley ([1975a], [1975b] and [1979]). These authors have argued that macroevolution is 'essentially decoupled' from microevolution and that evolution within populations has nothing to do with evolution of higher taxa. The argument for this decoupling depends upon two claims. First, that little change occurs between speciation events (i.e., during phyletic evolution) and second, that the speciation event is random:

The direction taken by any particular speciation event or succession of events must be determined largely by historical accident. . . . If rapidly divergent speciation interposes discontinuities between rather stable entities (lineages) and if there is a strong random element in the origin of these discontinuities (in speciation), then phylogenetic trends are essentially decoupled from phyletic trends within lineages (Stanley [1979], pp. 186–7).

This is a controversial proposition and debate on this issue continues to intensify. However, its resolution really has no direct bearing on the issues under discussion here. On the surface, Stanley's claim appears to undermine the gene frequency approach, since the formal results of population genetics are usually associated with phyletic evolution. However, on closer analysis Stanley's work supports the primacy of population genetics. For if we push beneath the surface to ask what events occur during the speciation process (what Stanley terms 'species selection'), we find exclusive use of standard population genetic concepts. The founder principle, genetic drift, inbreeding, adaptive topography and population structure are all traditional population genetic concepts, defined and elucidated by the gene frequency models to be discussed shortly. These very concepts, with their traditional meaning, are used, for example, in Stanley [1979] to define and discuss species selection and quantum speciation. Indeed Stanley ([1979], p. 143) acknowledges that the punctuational model does not invoke mechanisms of evolution that are unrecognized in the Modern Synthesis, but simply places different degrees of emphasis on various elements of change.

As Eldredge and Gould and Stanley often point out, their work is directly descended from the work of Simpson [1944] and Mayr [1963] which is permeated by population genetical concepts, themselves elucidated by the
study of gene frequencies (see Provine [1977] for discussion). In agreement, Lewontin later ([1974], p. 159) qualifies the previous quote of his to say,

I do not mean to say that the theory and observations of population genetics have not influenced and even permeated theories of species formation. One has only to read Dobzhansky’s *Genetics and the Origin of Species* [1951] or Mayr’s *Animal Species and Evolution* [1963] to see how population genetics informs modern ideas about speciation.

Consequently, there are, I think, good reasons for insisting on the evolutionary generality of the population genetics approach to evolution. The gene frequency approach provides an underlying framework for the various subdisciplines of evolutionary biology within which problems can be conceptualised and defined (see also Ruse [1973] for a discussion of this role of population genetics). On the other hand, there can be no doubt that other genetic changes are equally important, especially in the speciation process. However, an argument could be made that these other genetic changes will, more often than not, be reflected in gene frequency changes. For example, over thirty percent of the genera of flowering plants have speciated via polyploidy (Dobzhansky et al. [1979], p. 224). Two reasons are given (*ibid.*). First polyploidy ‘contributes characteristics that by themselves have intrinsic value’. If this were the only reason, it is conceivable (though not likely) that no gene frequency changes would ensue. However, the second important reason is that ‘polyploidy results in the fixation of heterozygous gene combinations . . .’. Consequently the ensuing gene frequency changes are thought to be an important factor in the evolutionary significance of polyploidy. In other cases, the gene frequency changes triggered by other genetic events may be only a second order phenomenon, not a primary result. In these cases the gene frequency approach would certainly prove inadequate, although it remains to be seen how common these cases are.

(b) *Empirical Progress*

The other important component of Lakatos's model of scientific research programmes is the positive heuristic. My main thesis here is that a series of simple genetic models, termed 'beanbag genetics', has served and continues to serve as a positive heuristic in evolutionary biology. Two things will be shown to substantiate this claim. First that beanbag genetics consists of 'a chain of ever more complicated models simulating reality' and, most importantly, that these models provide for direction within the research programme by containing
Second it must be shown that the development of beanbag genetics is ‘empirically progressive’.

The criterion used for empirical progress is whether the theoretical development of beanbag genetics has anticipated new facts. A new theory has some excess empirical content over its predecessor . . . if it predicts some novel, hitherto unexpected fact . . . (and) a series of theories is empirically progressive if some of this excess empirical content is also corroborated ([1970], p. 118).

The stronger condition of content increase sometimes imposed by Lakatos ([1970], pp. 120, 124) is much more difficult to show as it must be shown that all the old facts as well as some new ones are explained by the new theory. I have doubts whether this could ever be shown in general for two, let alone a series, of theories (Laudan [1977]). Indeed much of the time the old data are simply forgotten as the field moves around them. However in the context of a particular problem there is usually some overlap in the data explained by two successive theories and, in this restricted domain, it is, I think, possible to show content increase (see section 3(e)). For example, Hamilton’s theory of kin selection discussed in 3(e) and the older group selection arguments (used by Darwin) both explain the existence and some associated facts of sterile casts in the social insects, but kin selection goes farther to explain the association of sociality with the haplodiploid genetic system (i.e., a new fact). However, it would be foolish to argue that the data explained by group selection are included in the domain of kin selection or that kin selection explains “more” than group selection. This could never be determined and on this point I agree with Laudan [1977]. In any case, what I intend to show is that the development of beanbag genetics has anticipated new facts which have since been corroborated.

(c) Background for Population Genetics

Our discussion begins at the turn of the century when Mendel’s laws were rediscovered by deVries in 1900. This is a natural starting point, since most models in population genetics begin explicitly with Mendel’s Laws.\footnote{For example in the late 1950s and early 60s many workers in population genetics were calculating $B/A$ ratios, which are the ratios of genetic loads (see 3(b)) between outbred and inbred populations. These ratios were supposed to decide the kind of genetic variation present in a population. Nothing seems to have resulted from the large amount of field and experimental data involving $B/A$ ratios. For a number of reasons, the field seems to have moved around these data.}

\footnote{See next section. This claim could be made stronger to include all models in population biology, if only an implicit presupposition of Mendel’s Laws and their consequences (especially the conservation of variation) is relied upon. See also Ruse [1973].}
Various forces are then added and evolutionary results can be deduced. The successive addition of these forces represents the heuristic mentioned above and discussed in more detail below.

The period beginning with the publication of Darwin’s book in 1859 up to the turn of the century was one of great controversy for Darwin and his theory (see especially Hull [1973]). However, although there were many scientific and public debates during these years, little progress was made in the theory of evolution until genetic principles were included. Indeed, it would not be difficult to make a strong case for the assertion that evolution doctrine as a whole would be sounder today if all speculation concerning it had been banned between the publication of *The Origin of Species* and the second or third decade of the present century (Shull [1936], p. 210).

I now turn to this period and summarise some of the anomalies which Darwin’s theory faced. This summary is based on Provine’s [1971] and Shull’s [1936] account of this period. In the next section I show how the advent of beanbag genetics allowed evolutionary theory to progress around these and other anomalies.

At the turn of the century, most biologists believed that selection of continuous variation (Darwin’s small ‘individual differences’) was simply too weak a force to be responsible for evolution. Often biologists (e.g., Punnett, see quote from [1915b] below, and Morgan) would de-emphasise the role of selection and emphasise the importance of mutational leaps. Until the use of mathematical models (see next section) to explore the quantitative consequences of Mendelism and the advent of population genetics, most biologists were quite unconvinced that selection of small differences was sufficient to explain evolution. Many biologists (e.g., Mayr, Rensch, Sumner) were still neo-Lamarkians in the early 1920s.

Galton’s work on the phenomenon of regression had convinced most biologists that selection of continuous variation soon reached a limit due to the counteracting force of regression towards the population mean. Hence each new selected variant would regress towards the mean the next generation. It appeared that only discrete ‘leaps’ would be stable in the face of such regression, and mutation was thought to be the source of such stable new variants in a population.

Mutations, as we understand them today, are simply changes in the nucleotide sequence of the DNA molecule and as such represent change in the informational content of the genome. Recombination between two DNA molecules can also change the nucleotide sequence of the molecule and create new variations. Mutations arise because of mistakes made in
replicating the DNA and consequently represent the ultimate source of genetic variation. However, historically the term 'mutation' was used in a different way as a progressive force in evolution.

Before the advent of modern genetics with the concomitant observational shifts, it was commonly thought that two types of variation existed. The first was Darwin's 'individual differences' which were small and almost continuous variations between individuals. The second consisted of discrete phenotypic classes (such as sports) which were termed by deVries 'mutations'. DeVries [1901] argued that this second class was largely responsible for evolutionary change and that the 'individual differences' were relatively unimportant. He further argued that new species must arise by such mutations:

The latter (Darwin's theory of selection of individual differences) assumes that the usual or the so-called individual variability is the starting point of the origin of new species. According to the mutation theory the two (individual and mutational variabilities) are completely independent. As I hope to show, the usual variability cannot lead to a real overstepping of the species limits even with a most intense steady selection (deVries [1901], quoted out of Dobzhansky [1970], p. 42).

Of course, according to modern genetics, this distinction between the two types of variation is spurious. The resolution lies in Mendelian genetics and multifactorial inheritance as will be discussed shortly.

However, deVries had provided empirical support for such large 'mutational' variation through his work on Oenothera, the evening primrose, by observing the production of offspring which differed drastically from their parents. These 'mutant' changes which often resembled differences between two species of the same genus were permanent, and so it appeared that selection was not necessary for evolutionary change—especially selection of numerous, small variations resulting in gradual change. In addition Johannsen's work on pure lines showed that many of the small phenotypic variations, which Darwin had emphasised as important, were not heritable. Hence selection could not occur within these pure lines. Of course, in light of present knowledge, Johannsen's [1903] work and that of deVries are far from presenting anomalies or counterexamples to Darwin's theory. However, at the time the general impression was just the opposite because the theories used to interpret phenotypic variation did not emphasise the distinction between genetic and environmentally caused variation. Furthermore

no one had seriously considered the question of whether . . . (mutations) could of themselves, without the aid of any other process, really initiate a new species (Shull [1936], p. 208).
It is worth noting that problems concerning the genetic control of phenotypic variation have always existed in evolutionary biology. The shift to a model of multifactorial inheritance discussed in the next section is a case in point. Currently there are controversies surrounding the genetic theories underlying the enzyme polymorphisms found in natural populations.

The proponents of the evidence discussed above which appeared to run counter to the Darwinian selection hypothesis belong to the discontinuous school. The rediscovery of Mendel’s work in 1900 lent further support to the discontinuous school. If genes were discrete and non-blending as Mendel showed, then it appeared “obvious” that evolution should be discrete and not gradual as Darwin had argued. This “obvious” connection between Mendel’s discrete inheritance and the discontinuous school was made and defended vigorously by Bateson and opposed as being anti-Darwinian by the biometricians. The biometricians (especially Pearson and Weldon) studied continuously varying characters, such as height, and developed correlation tables for these characters between relatives. Mendelism was interpreted as a threat by the biometricians since they immediately associated it with discontinuous evolution:

To those who accept the biometric standpoint, that in the main evolution has not taken place by leaps, but by continuous selection of the favorable variation . . . there must be a manifest want in Mendelian theories of inheritance. . . . To complete a Mendelian theory we must apparently associate it for the purposes of evolution with some hypothesis of “mutations”. The chief upholder of such a hypothesis has been deVries . . . (Pearson [1906], p. 39, taken from Provine [1971], p. 69).

Many biologists were convinced that Mendelism went hand in hand with discontinuous evolution even though the connection was spurious, soon to be overcome by the positive heuristic of the emergent population genetic programme (see next section). However, this connection was reasonable at the time, especially in light of the fact that Darwin himself had depended on a blending theory of inheritance in his theory of evolution by selection of continuous variation.

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1 For a very current example involving the ‘hidden’ genetic variation being discovered by new molecular techniques, consider the effects of post-translational modification of structural proteins on the neutralist-selectionist controversy (Finnerty and Johnson [1979]). Such modification allows for combinatorial effects between loci in producing ‘observed’ enzyme variants. These effects reduce the absolute number of segregating alleles which must be explained by selectionist theories and represent a shift in the observational theories underlying genetic variation.
(d) **Beanbag Genetics**

Besides serving as an introduction to beanbag genetics, this section will discuss the role of beanbag genetics in the synthesis of the 1920s. The following sections will demonstrate the continuing role of beanbag genetics as a positive heuristic in contemporary evolutionary biology.

A general heuristic tool in theoretical population genetics is to assume a specific model of inheritance for a trait and then to deduce possible evolutionary results. Examples include single locus, multilocus, quantitative inheritance and phenotypic models. Evolutionary results are obtained in the following way. The dynamics and equilibria for the model are first investigated without any outside forces operating. The equilibria attained are the gene frequency distributions which do not change over time. Subsequently, the gene frequency dynamics and equilibria are investigated with forces acting on the system. The standard forces include selection, sampling effects, mutation, migration, gene conversion, etc. This theoretical methodology can provide insights about how genes and their traits evolve as discussed shortly.

There can be little doubt that the most widespread genetic model assumed is a single Mendelian locus. Such models were termed 'beanbag genetics' because they ignore interactions between loci by assuming a single 'genetic factor' controlling the phenotype. Beanbag genetics is so widespread, not because it is generally realistic (it sometimes is), but because this approach has been and continues to be so fruitful in suggesting new avenues of research. This heuristic power of beanbag genetics has been demonstrated repeatedly and some examples will follow shortly. The other genetic models have been less pervasive in this respect and I will not discuss them here.

Interactions between loci are, of course, important in the genetic control

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1 The Castle-Hardy-Weinberg law discussed shortly is an example.
2 This genetic factor is usually thought of as a single locus. However it could be a group of loci between which there is no recombination and which are inherited as a unit. Examples include supergenes and inversions.
3 It is possible to justify this approach. For example, Williams states ([1966], pp. 56-57). 'Obviously it is unrealistic to believe that a gene actually exists in its own world with no complications other than abstract selection coefficients and mutation rates. The unity of the genotype and the functional subordination of the individual genes to each other and to their surroundings would seem, at first sight, to invalidate the one-locus model of natural selection. Actually these considerations do not bear on the basic postulates of the theory. No matter how functionally dependent a gene may be, and no matter how complicated its interactions with other genes and environmental factors, it must always be true that a given gene substitution will have an arithmetic mean effect on fitness in any population. One allele can always be regarded as having a certain selection coefficient relative to another at the same locus at any given point in time. Such coefficients are numbers that can be treated algebraically, and conclusions inferred for one locus can be iterated over all loci. Adaptation can thus be attributed to the effect of selection acting independently at each locus.'
of many traits. However, first order approximations along with the initial insights to evolutionary problems are still obtained by taking the beanbag genetic approach. This is as true today as it was in the 1920s. Complexity and realism are usually attained, not by assuming more interacting loci, but by assuming a richer repertoire of forces operating on the single locus. To add complexity by increasing the quantity of interacting loci controlling a trait, one must usually sacrifice complexity in the quality of forces acting.

The term 'beanbag genetics' was offered (rather disrespectfully!) by several quite prominent experimentalists who doubted the value of these models in evolutionary biology. However, an analysis of the role of these models in the evolutionary synthesis of the 1930s and 40s concluded that these models (i) demonstrated the sufficiency of Mendelism and weak selection as a cause of evolution, (ii) indicated which paths of research would be unproductive, and served as a framework for (iii) the reinterpretation of data and (iv) directing future research (Provine [1977]). An underlying motif of Provine's article is that these models functioned in subtle ways to modify the thinking of experimentalists (especially Dobzhansky, see his [1937]) and that the results of these models were quickly, and almost imperceptibly, incorporated into the observation language of evolutionary biology.

The basis of beanbag genetics is the Castle–Hardy–Weinberg (C–H–W law) equilibrium law, first formulated by Castle in 1903. It states that if no outside forces operate on a population, gene frequencies will remain constant. This law is analogous to Newton's first law in physics (Ruse [1973]), and it is a direct logical deduction from Mendel's laws (e.g., see Ruse [1973], pp. 32–37 for the deduction). This law is important because it provides, not only a foundation for the mathematical investigation of the dynamics of gene frequencies, but also a null hypothesis in the investigation.

It has been suggested recently that even weak interactions between loci have important effects on the dynamics of genes. These interactions can build up in a synergistic way and this can lead to joint distributions of alleles which are dramatically different than those predicted by the marginal frequencies at each locus (Franklin and Lewontin [1970], Lewontin [1974]). This was an important insight and would certainly undermine the beanbag genetic approach. However experimental work has failed to find the predicted linkage disequilibrium (a measure of the statistical association of allele frequencies at different loci). It appears now that the power of recombination was underestimated. Further theoretical work on the Franklin and Lewontin [1970] model with slightly higher values for recombination and lower values for the selection parameters (obtained from estimates in nature) has turned up the opposite results (Clegg [1978]). In systems which favour heterozygotes, selection can actually increase the rate of decay of linkage disequilibrium. Interestingly enough the effect that recombination has in breaking down statistical associations between loci, thereby allowing selection to act on 'net gene effects' (see p. 13, n. 3), is now thought to be one of the more important advantages of the sexual cycle (Felsenstein and Yokoyama [1976], Maynard Smith [1978]).

Especially Ernst Mayr. Provine [1977] has a nice discussion of this controversy as well as the relevant references.
gation of natural and experimental populations. If we assume one locus with two alleles $A$ and $a$, where the frequency in gametes of $A$ is $p$ and the frequency of $a$ is $(1-p)$, then the genotypic array under random mating (i.e., random association of gametes) is

$$\begin{array}{ccc}
AA & Aa & aa \\
p^2 & 2p(1-p) & (1-p)^2 \\
\end{array}$$

If there are no outside forces the C-H-W law states that the population will remain at this composition. Dynamics are introduced by hypothesizing the outside forces mentioned previously. In this essay only the incorporation of selection will be discussed. The philosophical points made here hold for the other forces as well.

The first and most basic model of selection is constant viability selection. We hypothesise a constant ‘fitness’ $W$ for each genotype which measures the probability that the individual will survive to reproduce. It is implicitly assumed that no differences in mating success, fecundity, etc. exist between the three genotypes. Since we are only interested in frequencies of genes, the fitnesses are usually expressed relative to a fitness of 1 for the heterozygote. It is sometimes also customary to express the fitness $W$ as one minus the selection coefficient $s$:

$$W = p^2(1-s_1) + 2p(1-p) + (1-p)^2(1-s_2)$$

is the ‘average fitness’ of the population. The new frequency of the $A$ allele after selection is

$$p' = \frac{p^2(1-s_1) + p(1-p)}{W}$$

This is also the frequency among offspring the next generation since there is assumed to be no other form of selection operating. The mating cycle
itself cannot change the frequency unless some genotypes do better than others in obtaining mates. Consequently the change in gene frequency over one generation is

\[ \Delta p = p(1-p) \frac{s_2(1-p) - s_1 p}{W} \]  

This process can be repeated as many times as desired (substituting \( p' \) back in for \( p \) in (4)) to calculate the gene frequency dynamics recursively.

This model is the first in a long series of beanbag models used in population genetics. All such models share the underlying assumption of Mendel’s Laws and seek to establish deductive consequences of these laws when they are coupled with other assumptions concerning possible evolutionary forces. This purpose was shared by Fisher, Haldane, Wright and Chetverikov, the main figures around which the neo-Darwinian theory evolved, as is evidenced by many explicit statements in their papers.

When in the 1920s and 30s Fisher, Haldane and Wright began building these models to determine the quantitative consequences of Mendel’s laws, they knew more or less what was being left out at each step. Every force which could conceivably disturb the Hardy-Weinberg equilibrium was to be investigated mathematically. For example, Haldane understood initially the complicating forces he wanted to include:

It is proposed in later papers to discuss the selection of semi-dominant, multiple, linked, and lethal factors, partial inbreeding and homogamy, overlapping generations, and other complications (Haldane [1924], p. 40).

and he viewed the problems as mainly ‘mathematical rather than empirical’ (Lakatos [1970], p. 136):

indeed the mathematical problems raised in the more complicated cases to be dealt with in subsequent papers seem as formidable as any in mathematical physics (Haldane [1924], p. 19).

Very rarely was any reference made to the empirical content or testability of the models, except in the weak sense of suggesting what parameters and variables might be operative in nature.

The model above (2) is usually false, since fitness is not constant and genotypes often differ in reproductive success. However, as the quote from Haldane above indicates, one hopes to remove these assumptions at a later date. These models will now be seen to turn ‘one counter-instance after another into corroborating instances’ (Lakatos [1970], p. 133).

Beginning with model (2) it is possible to calculate the time in generations needed to change an allele from one frequency to another, given values of the selection coefficients \( s_1, s_2 \). When this was done (first by Norton in Punnett [1915a], also reprinted in Chetverikov [1926] and
Provine [1971]), biologists were astonished by the relatively short periods of time needed to change allele frequencies:

Evolution . . . may be a very much more rapid process than has hitherto been suspected, for natural selection, if appreciable, must be held to operate with extraordinary swiftness . . . (Punnett [1915a], p. 96).

Indeed Chetverikov ([1926], pp. 181–9) devotes a full eight pages to discussing the implications of Norton’s one table for . . . these conclusions have a very great importance for a correct understanding of the evolutionary process. . . . Here Darwinism . . . received a completely unexpected and powerful ally in Mendelism . . . (for) . . . one of the most substantial difficulties of Darwinism has always been the difficulty in imagining the process by which minute improvements (accumulated).

Haldane’s first paper [1924] contained similar calculations for many other situations. Such calculations helped to delineate the relative effectiveness of selection in different situations and, in so doing, helped to resolve old anomalies. For example, the existence of deleterious traits had always been perplexing to the Darwinian programme, a programme which emphasised selection of favourable traits. But Norton’s and Haldane’s calculations on the rate of elimination of deleterious, recessive mutations from a population indicated that long periods of time are required . . . and we need not wonder that recessive sports still occur in most of our domestic breeds of animals’ (Haldane [1924], p. 24).

Such mathematical calculations convinced most biologists that selection on small variations could explain evolution. As Provine [1977] notes

This conceptual difficulty with Darwin’s small selection pressures was removed by the new mathematical models of selection. Small selection pressures did not appear to be capable of changing populations rapidly enough to fit the geological record, but in fact (due to the models) they could. The problem was in the minds of evolutionists, not in the small selection pressures.

Hence, it was not by the accumulation of new data (in fact, there was no change in the data) that the field progressed, but by a conceptual revolution prompted by a simple beanbag genetic model (2).

In 1918, Fisher demonstrated mathematically that Mendelian genetics was consistent with apparently continuous phenotypic variation and hence the compatibility of the two was finally established. Again, Fisher used a Mendelian genetic model. However, instead of just one locus controlling a trait (as above), many were assumed to affect the same trait, each obeying Mendel’s Laws but behaving independently (i.e., many “beans” in the

1 These different situations are all variations of the basic Mendeian model (2) and need not concern us here.
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"bag"). Fisher's model modified the relationship between Mendelian variation and phenotypic variation by positing many discrete Mendelian genes, each having a small cumulative effect on the same trait (e.g., height). With this auxiliary hypothesis, Fisher was able to derive the statistical properties of such a trait and reproduce many of the correlations between relatives which had been the domain of the biometricians in their studies of continuous phenotypic variation. These results resolved the apparent anomaly between continuous phenotypic variation and discrete Mendelian genes by modifying the relationship between the phenotypic data and the theory of genetic control which had given rise to the anomaly in the first place.

The priority of theory in effecting this shift was not complete, however, and experimental evidence had been produced previous to 1918 suggesting a multifactorial theory of inheritance.1 However, as Sewall Wright recalls:

The geneticists seem to have paid no attention, at least until Fisher elaborated the reconciliation in 1918 . . . ([1977b], p. 683).

Indeed in 1915 the problem was still very much out of focus. For example concerning the evolution of mimicry in butterflies, Punnett ([1915b], p. 146) argued for the 'Mendelian view (that) natural selection merely conserves the (mimic) form once it has arisen (by mutation) . . . rather than the modern Darwinian view (that) natural selection gradually shapes (the mimic) . . .'. Indeed, Fisher, in discussing his 1918 paper a few years later, states ([1922], pp. 321–2, the italics are mine):

At that time, two misapprehensions were generally held in regard to this problem . . . it was widely believed that the results of biometrical investigation ran counter to the general acceptance of the Mendelian scheme of inheritance. . . . At the time when the paper of 1918 was written, it was necessary, therefore, to show that the assumption of multiple, or cumulative, (Mendelian) factors afforded a working hypothesis for the inheritance of such apparently continuous variation as human stature. This view is now far more widely accepted.

Fisher's mathematical demonstration served to concentrate experimental work in this direction for

Mendelian research has with increasing frequency encountered characters which are evidently affected by many separate (Mendelian) factors. In some fortunate circumstances . . . it has been possible to isolate and identify the more important of these factors by experimental breeding on the Mendelian method (ibid.).

1 (i) William Castle demonstrated the effectiveness of selection on a continuously varying character. (ii) Nilsson-Ehle and Edward East demonstrated that Mendelian factors could account for some continuous variation. (iii) T. H. Morgan demonstrated that Mendelian factors might have very small effects (Provine [1971], pp. 108–30). However these results were provisional, at best, with little generality: 'Unfortunately . . . although I feel it extremely probable that variations in some characters that seem to be continuous will prove to be combinations of segregating characters, it is exceedingly difficult to demonstrate the matter beyond a reasonable doubt' (East [1910], p. 74, his italics).
However, empirical demonstration of Mendelian genes is not really necessary. Such data, though reassuring, are really only of secondary importance since logically, within the context of the models, there no longer exists any problem:

more frequently, however, no such analysis has been achieved. In these cases we can confidently fall back upon statistical methods, and recognize that if a complete analysis is unattainable it is also unnecessary to practical progress (ibid.).

With incorporation of both mutation and selection, in mathematical models (especially by Haldane) like (2) above, the relationship between selection and mutation as evolutionary forces was clearly delineated. Before this was done, many evolutionists believed that mutation pressure was the important factor in evolutionary change. However, the mathematical models showed this not to be the case and consequently removed many of the objections of the early mutationists (e.g., deVries, discussed in the last section) to evolution by selection of continuous variation.

In 1922, Fisher hypothesized \( s_1, s_2 > 0 \) in model (2) above (i.e., heterozygote superiority) and showed that an equilibrium was reached at which gene frequency remained constant maintaining both alleles in the population (termed a 'balanced polymorphism'). To obtain this constant gene frequency, set \( \Delta p = 0 \) in (5) and solve for the equilibrium \( p \):

\[
\frac{s_2}{s_1 + s_2}
\]

It had often been thought that selection would tend to purify a population of its variation.¹ Now just the opposite becomes a possibility. This result on heterozygote superiority stimulated workers to go to nature and look for balanced polymorphisms,² and the concept of balanced polymorphism is now central to evolutionary biology.

The available examples are numerous. By the successive incorporation of dominance, assortative mating, population structure, migration, finite sampling effects, etc. into the framework provided by (1) above, the theory progressed and in so doing 'provided a guiding light for rigorous quantitative experiment and observation' (Dobzhansky [1955]). It is important to stress that these advances in most cases were not precipitated by new data:

¹ See, for example, Chetverikov's discussion of Norton's table (Chetverikov [1926], p. 186) or Darwin's discussion of polymorphic species [1859], pp. 46, 81).
² Especially Dobzhansky, Ford and their students. See the preface to Ford's [1971] for his debt to Fisher. Chetverikov and the Russian school were also preoccupied with the large amount of cryptic variability preserved in natural populations by selection (Chetverikov [1926], Adams [1968]). The debt of Chetverikov to the mathematical models is explicit in his [1926].
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The experimental work, that should test these mathematical deductions is still in the future, and the data that are necessary for the determination of even the most important constants in this field are wholly lacking. Nonetheless, the results of the mathematical work are highly important, since they have helped to state clearly the problems that must be attacked experimentally if progress is to be made (Dobzhansky [1937], my italics; see also Chetverikov [1926], p. 182).

It is also worth noting that implicit in all the above models is the assumption that gene frequency changes are evolution. This hard core, identified previously, gives justification to the models. Without this core, the models would have no import in evolutionary biology at all.

The few examples discussed above should clearly show that although Mendel's Laws were rediscovered at the turn of the century, their consequences for the theory of evolution were not understood until they were investigated mathematically with the beanbag models:

Since 1900, a body of knowledge on the mechanism of heredity and on mutation has been built up by experiment that challenges any field in the biological sciences in the extent and precision of its results. The implications for evolution are not, however, immediately obvious. It is necessary to work out the statistical consequences (Wright [1942], pp. 223, italics mine).

Though Mendel's principles were rediscovered in the year 1900, and though every evolutionist from Darwin on—and none more than Darwin—recognized the important relation existing between heredity and evolution, no appreciable influence of Mendelian theory upon evolutionary speculation was discernible in the early years of the present century. Expressions of surprise have sometimes been called forth by the failure of evolutionary writers to make more use of Mendelian phenomena during this period (Shull [1936], p. 211).

Mendel's Laws are not evolution, and data on segregation ratios mean nothing to evolution unless set in a conceptual framework which exposes their meaning. The construction of this framework was and is the function of the positive heuristic.

I think enough has been said concerning the role of the mathematical models of the beanbag variety in the evolutionary synthesis. I turn now to the continuing role of beanbag genetics in evolutionary biology.

3 NATURAL SELECTION AND THE CONCEPT OF FITNESS

(a) Introduction

In this section, I first (3(b)) discuss the concept of genetic load. This concept has been very important in evolutionary biology and stems from beanbag genetic models. In section 3(c), I discuss the role of beanbag genetics in observing and quantifying natural selection through estimation procedures which rely on these models. Next 3(d), I discuss some changes which have
occurred in the concepts of natural selection and fitness, as well as in ways of observing them. In all cases these changes are triggered by the development of beanbag genetics. I have devoted the final section 3(e) to another shift in the concept of natural selection, kin selection. This section demonstrates the heuristic power of beanbag genetics in creating new subdisciplines of evolutionary biology (e.g., sociobiology).

(b) Genetic Load
The concept of genetic load refers to the loss in fitness of a population by the presence of less fit genes. These genes can be maintained by forms of 'balancing selection' and it is with this kind of load, 'segregation load', that I shall be primarily concerned. There are other kinds of 'load' in the evolutionary process, for example, 'mutation load' (Haldane [1937]) and 'substitution load' (Haldane [1957], Felsenstein [1971]) or more recently 'lag load' (Maynard Smith [1976]). In all cases the concept concerns the effect of gene frequencies on the average, population fitness $W$. All these concepts have been very important in evolutionary biology and all originated in beanbag genetic models. Genetic load is usually defined as the difference in fitness between a population and a hypothetical population composed of some optimal genotype. In the case of segregation load the concept is a direct result of the constant fitness model (2).¹

It is difficult to convey how important the concept of genetic load has been in population genetics. In the late 1950s and early 60s many researchers were calculating $B/A$ ratios, which are the ratios of genetic loads between outbred and inbred populations. These ratios were supposed to decide the kind of genetic variation present in a population.² The concept also forms the basis for a recent paradox which involves the large amounts of genetic variation observed in natural populations. In the late 1960s up to the present, much time has been spent in population genetics discovering and explaining the large amounts of genetic variation uncovered by the technique of gel electrophoresis. The explanation of this variation has polarized the field into two camps, the 'selectionists' and the 'neutralists'. The initial paradox (Lewontin and Hubby [1966]) involved arguing that the variation was being maintained by natural selection. The paradox was due to the model of selection assumed: constant fitness, heterozygote superiority at each variable locus. To explain the variation

¹ First, assume heterozygote superiority, which is a form of balancing selection since it maintains both alleles in the population. Since the heterozygote is the optimum genotype (it has the highest fitness of 1 and $s_1, s_2 > 0$ by assumption) and $W = p^2(1-s_1) + 2p(1-p)(1-s_1)$, the load becomes $1 - W = p^2s_1 + (1-p)^2s_2$. Clearly the larger the intensity of selection and the greater the advantage of the heterozygote (i.e. the greater $s_1$ and $s_2$), the greater the genetic load.

² See Lewontin ([1974], pp. 74–82) for an especially clear explication of this procedure.
using this model, strong selection (e.g., $s_1, s_2$ large) must be assumed, with the accompanying huge amount of genetic load. For if weak selection were assumed (i.e., $s_1, s_2$ small but positive), the variation would be lost by random genetic drift. Neither were acceptable, hence

We then have a dilemma. If we postulate weak selective forces, we cannot explain the observed variation in natural populations unless we invoke much larger mutation and migration rates than are now considered reasonable. If we postulate strong selection, we must assume an intolerable (genetic) load of differential selection in the population (Lewontin and Hubby [1966], p. 607).

This dilemma then gave rise to the neutralist explanation of the variation as being nonadaptive or neutral with respect to selection (i.e., $s_1, s_2 = 0$). In other words, the variation detected by electrophoresis is just evolutionary noise.

These two explanations of the electrophoretic variation have really developed into two “mini” research programmes within population genetics, though both stem from models of the beanbag variety. The neutral models ($s_1, s_2 = 0$) still function as a heuristic in evolutionary biology, for experimentalists readily admit that these models have ‘greatly contributed to evolutionary genetics by inspiring and guiding (experimental) research’ (Ayala et al. [1974], p. 381). I will not go into these two programmes in any detail as I believe it is too early to make any comparisons between the neutralist and selectionist programme concerning progressing or degenerating programme shifts. However, I do think that a Lakatosian reconstruction will be the only way of assessing the two programmes. There has been much talk in the literature about ‘critical tests’ deciding between the two programmes.\(^1\) However, it happens that whenever a proposed ‘test’ occurs, new subsidiary hypotheses are added to save the “falsified” programme.\(^2\) The early neutralist models were welcome by experimentalists because it appeared that the models generated a large class of potential falsifiers (Ayala [1977]). However, with successive shifts in the neutralist programme, this class diminished to the extent that now ‘they do not tell us what observations might not confirm the theory’ (Lewontin [1974], p. 228). Such shifts are the lowliest form of scientific behaviour according to Popper’s view of proper scientific etiquette. However, according to

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\(^1\) See, for example, Ayala [1977] or Ewens and Feldman [1976].

\(^2\) For example, homogeneity of gene frequencies over a large geographical area would indicate selection as it is opposed to the random differentiation of populations predicted by the neutrality programme. However, with the addition of the force of migration to the neutrality repertoire this anomaly (for the neutralists) disappears. Now it would be interesting to see if this shift in the neutralist programme was \textit{ad hoc} or progressive. I do not propose to analyse this here. Lewontin ([1974], chapter 5), provides other examples of such shifts in the two programmes and would provide an excellent starting point for such a discussion.
Lakatos’s model, all this is really beside the point as the concern for critical tests and ‘instant rationality’ is entirely misplaced. It is only long after one programme has died away, with the benefit of hindsight, that certain ‘tests’ are hailed as critical. The history of the selectionist—neutralist dispute strongly favours Lakatos’s model:

Indeed [Lakatos’s] methodology confidently predicts that where the falsificationist sees [or desires] instant defeat of a theory through a simple battle with some fact, the historian will detect a complicated war of attrition, starting long before, and ending after, the alleged ‘crucial experiment’ (Lakatos [1971], p. 118).

The dilemma faced in the selectionist explanation above stems, not from any conflict with the hypothesis of selection per se and the ‘data’,¹ but rather from a particular model of selection. The problem concerning genetic load, and the apparent anomaly sketched above, is a result of the shortcomings of the constant selection model. In fact this anomaly disappears when other more ecological models of selection are considered.²

It is, however, important to understand that the shift away from constant selection was not precipitated by a conflict between the constant selection model and the electrophoretic data. True, there is a conflict, but the shift was anticipated much earlier and is more properly seen as part of developing the positive heuristic. The shortcomings of the constant selection model were known previously. It functioned as a heuristic and in so doing resolved old anomalies (see section 2) while creating some new ones. However the main point of this section concerns the concept of genetic load, its important role in research and its origin in the positive heuristic.

(c) Estimating Fitness

In addition to explaining the mechanisms of evolution, beanbag genetics is an instrument for estimation of selection in natural and experimental populations. This section deals with this important role. Not only do beanbag genetic models suggest new ways to understand evolution, they also function as an instrument for observing and quantifying the evolutionary process.

A model similar to (2) is assumed.³ The fitnesses of the genotypes are then assigned on the basis of the observed changes in gene frequency over

¹ Though perhaps there are problems with some of the observations as well. See p. 12, n. 1.
² Especially the ‘frequency dependent’ model of selection (see, for example: Wright [1969], chapter 5; [1977a], chapter 14) which has no load at equilibrium. However this model creates new problems (see Lewontin [1974], p. 260).
³ See, for example, Wright and Dobzhansky [1946] or DuMouchel and Anderson [1968] and Anderson, Oshima, Watanabe, Dobzhansky and Pavlovsky [1968].
time. As noted previously, equation (4) can be iterated indefinitely, substituting $p'$ back in for $p$. The estimation of the selective coefficients consists of fitting these iterations of the model to the observed temporal changes in gene frequency. This method is important because it allowed population geneticists to quantify natural selection for the first time. However as a method of measuring fitness it suffers from a fundamental epistemological weakness. It is circular. It's the perfect example of the empty tautology which philosophers continuously gripe about: those which are the most fit are the ones which survive to increase in frequency, and those which survive to increase in frequency are the most fit.

This circularity results, in part, from the fairly restrictive concerns of population genetics. Traditionally, population genetics did not speak to the problem of adaptation but dealt only with the statistical dynamics of heritable change. Fitness was a theoretical construct employed in the equations of population genetics to determine the distribution of genotypes over time. Adaptation was taken for granted. Unless provided with bridge principles which connect this theoretical construct to the real world, the concept of fitness remains empirically empty. To construct the necessary bridge principles we must identify the relevant ecological constraints impinging upon the organism. It is only by taking an ecological perspective and identifying the ecological constraints and pressures independent of survival that one can break out of a tautological definition of fitness. The functional definition discussed in the next section (equation 7) does get us out of the circular definition of fitness, although the data required to use such a model is still in the future. Undoubtedly, such a model will eventually be used.

A step in this direction has been taken by breaking down the overall measure of fitness into its various components due to longevity, fecundity and even mating cycle parameters (for example, Clegg, Kahler and Allard [1978], Bundgaard and Christiansen [1972], Christiansen and Frydenberg [1973]). In this case the simplest selection scheme becomes

\[
\begin{array}{ccc}
AA & Aa & aa \\
\text{longevity} & l_1 & l_2 \\
\text{fecundity} & f_1 & f_2 \\
\end{array}
\]

and the overall selection, $s_1$, is some combination of $l_1$ and $f_1$. Likewise for $s_2$. This represents a more ecological perspective and an increase in empirical content since the times and means of sampling are dictated by the biology and life history of the organism.

The inferences one wishes to make by applying selection models to data concern the amount and type of selection operating. For Darwin,
natural selection concerned the struggle to survive, and the classical bean-
beanbag models (2) concentrated on survivorship as the only component of
selection. However, the use of such models, which employ one measure of
selection for each genotype, can lead to incorrect interpretations concerning
the type and amount of selection operating (Prout [1965], [1969]). Of
course, it wasn’t known that these interpretations were ‘incorrect’ until
the positive heuristic advanced. The total selection occurring in a gener-
ation is the important quantity in evolution and, consequently, it was
natural to first assign only one overall selection coefficient to a genotype.
However as beanbag genetics advanced to include the selection component
models outlined above, it soon became apparent that the classical models
were misleading in applications. It is not necessary to reproduce the
algebra which demonstrates these deficiencies and the advantage of a
selection components model, and only the conclusions will be given here.
The interested reader is referred to Prout’s seminal papers ([1965], [1969],
[1971a], [1971b]).

First, an overall selection coefficient underestimate the amount of
selection taking place within the life cycle as well as the portion of the
reproductive excess ‘used’ in evolution. This is the case because selection
can occur in different directions within the life cycle. The different
directions tend to cancel out so that the net selection appears to be small
while the total amount of selection may be much greater (Clegg, Kahler
and Allard [1978]). Second, misuse of these classical models can lead to the
wrong conclusion concerning the type of selection occurring, especially in
the case of frequency dependent selection. This is important for this is a
qualitative mistake rather than something merely quantitative, as for the
first deficiency. The mistake results when fitness estimates are obtained
by comparing the genotypic frequencies at the same stage of the life cycle
in successive generations. If selection is incomplete at the stage chosen


Although it is easy to observe the products of natural selection, it has
proven very difficult to observe the process. My main point in this section
concerns the important role of beanbag genetics in structuring the ob-
servations of the process of natural selection which are possible. This role
seems related to the role of what Lakatos terms the ‘observational theory’
in science. The observational theory provides the researcher with the facts
which must be explained by specifying the properties, artifacts and limits
of the instrument of observation. Lakatos further argues⁴ that the positive heuristic often modifies the observational theories as it advances. The example of natural selection discussed here seems to be a case in point.

(d) Fitness in Population Biology

The selection coefficients in (2) were initially assumed to be constants. Historically this was the case because biologists were concerned with the efficacy of selection in relation to other evolutionary forces (see section 2). Once this had been sorted out, it was necessary to analyse further the selection coefficients, for constant fitnesses are forbidden by ecological laws and models. The need for such an analysis has been realised by philosophers as well:

Population genetics thus uses a parameter, the selection coefficient, which must be further analyzed. . . . It is incomplete (though not incorrect) as a theory of evolution without them (Wimsatt [1970]).

To make any evolutionary sense, a model must not only be rooted in the laws of genetics, but must incorporate forces which act at the level of the phenotype. However, since the phenotype can be understood only in an ecological setting, it is necessary to incorporate ecological forces. Consequently ecological forces become identified with selection coefficients, and the explicit functional form of these coefficients can be taken from models in population ecology. For this reason, it may be better to speak of a 'creative shift' (Lakatos [1970], p. 137) in the positive heuristic, though this 'shift' was anticipated in the pioneering work of the 1920s⁵ and is clearly within the spirit of the basic framework (2). The basic framework of the beanbag genetic model (2) directs attention to the selection coefficients. It is through these variables that contact can be made, through additional hypotheses, with the actual ecological causes of fitness. It is in this way that beanbag genetics models are a powerful force in the ongoing synthesis of population biology.

The development of an ecological perspective on selection has taken many different forms. Prior to making fitness an explicit function of ecological variables, s₁ and s₂ were allowed to vary both in time and space. The first model was that of Levene [1953], in which he demonstrated that genetic variation could be maintained without heterozygote superiority if individuals dispersed into different habitats, underwent different selective forces in the habitats (i.e., there are two sets of fitnesses, one for each

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¹ See, for example, the quote in section I from Lakatos ([1970], p. 133).
² See, for example, Norton [1928] and Haldane [1927]. Though Norton's work was largely ignored for some 40 years, it has a decidedly modern ring to it.
habitat) and then reunited to form a random mating population before the next generation of dispersal. The selection scheme looks like

<table>
<thead>
<tr>
<th></th>
<th>AA</th>
<th>Aa</th>
<th>aa</th>
</tr>
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<tbody>
<tr>
<td>Environment I</td>
<td>$s_1^I$</td>
<td>1</td>
<td>$s_2^I$</td>
</tr>
<tr>
<td>Environment II</td>
<td>$s_1^{II}$</td>
<td>1</td>
<td>$s_2^{II}$</td>
</tr>
</tbody>
</table>

in Levene's model. The theoretical demonstration that environmental heterogeneity could maintain a polymorphism was an exciting discovery for it had often been thought that heterozygote superiority was the only form of balancing selection. This demonstration, especially through the work of Levins (reviewed in his [1968]), initiated the move in field and experimental population biology towards a concern for environmental heterogeneity and a rapid proliferation of research on this subject followed. In addition, other models (Haldane and Jayakar [1963] and Karlin and Liberman [1974]) demonstrated the importance of temporal variation.

However, these models do not attempt explicitly to represent the ecological causes of the environmental variation. The shift toward specifying the ecological causes of selection is important for two reasons. First, a functional representation of the selection coefficients is a move away from the tautological definitions of fitness which still pervade the estimation procedures in population genetics (discussed in the last section). The second reason, which is related to the first, is that this shift increases the empirical content of the models.

Newton's Laws have little empirical content until supplemented with subsidiary hypotheses, and none of these hypotheses follow directly from Newton's Laws (Kliener, personal communication). For example the abstract equation $F = ma$ has no empirical content until an identification of various causes are made (for example, electromagnetic, gravity, 'weak' and 'strong' forces, etc.) which produce the effect of acceleration. The same is the case in the theory of evolution. The concept of fitness, abstractly presented in model (2), has no empirical content until supplemented with ecological causes which effect genetic change (for example, competition, predation, mechanics of population growth, etc.). The identification of the various forces which can effect genetic change and 'buffer the core from refutations' is, of course, the function of the positive heuristic.

Ecological theory focuses on the changes in numbers of organisms (instead of gene frequency) and attempts to formulate these changes in

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terms of population interactions. If there are no interactions, numbers obey the following equation

$$\frac{dN_i}{dt} = r_i N_i(t).$$  \hspace{1cm} (6a)$$

Solving this equation yields

$$N_i(t) = C e^{rt}.$$

(6b)

Here $N_i$ and $r_i$ are, respectively, the number of organisms and the growth rate of the $i$th type. Equation (6b) means that if no outside forces come into play, organisms increase exponentially. Of course, this is one of the premises (due to Malthus) from which Darwin deduced his principle of natural selection.

Equation (6) is analogous to the C–H–W law in genetics or Newton’s first law in physics. It states that if no outside forces operate on a population, the growth rate won’t change. Potential outside forces are due to population interactions (e.g., competition, predation, etc.). These interactions can be incorporated into (6a) abstractly as

$$\frac{dN_i}{dt} = f_i(N_1, N_2, \ldots, N_m) \quad i = 1, 2, \ldots, m \quad \hspace{1cm} (6c)$$

where there are $m$ interacting types and the function $f_i$ describes explicitly the effects of the $m$ types on the growth rate of type $i$. It is not important to go into the explicit functional form in this discussion. Besides being functions of population size $N_i$, the $f_i$’s could be made functions of any other relevant ecological variable.

We are now in a position to propose an interlevel model.\(^1\) Selection is due, in part, to the interactions between individuals of different types. We can borrow the functional form of these interactions (the $f_i$’s above) from the models occurring in ecology in formulating the mechanisms of selection. In other words, in the gene frequency equations (2) we define

$$s_1 = f_1(N_1, N_2, \ldots)$$

$$s_2 = f_2(N_1, N_2, \ldots)$$

(7)

Consequently we now have a system of coupled gene frequency equations and number equations.\(^2\) Thus it is by making the selection coefficients functions of processes occurring in the various subdisciplines of evolutionary biology (e.g., ecology, ethology, sociobiology, etc.) that beanbag

\(^1\) Beckner ([1968], chapter III) notes that interlevel explanations are very important in biology and that it is the role of models to provide the framework for such an explanation.

\(^2\) There are too many references in this area of research to present here. An excellent introduction to this area is Roughgarden [1979].
genetics serves to integrate various subdisciplines of evolutionary biology. The functional form of the selection coefficients are the bridge laws which connect gene frequency changes with the environment and hence will go some way in explaining adaptation. They are important because they are a first step in assessing fitness independently of survival. In the next section, I continue this theme concerning the functional representation of selection in beanbag genetic models by discussing a new subdiscipline of evolutionary biology, sociobiology.

(d) Sociobiology

There is perhaps no clearer example of the positive heuristic power of beanbag genetics in the Neo-Darwinian Research Programme than in the new field of sociobiology. This whole field of evolutionary theory was initiated by the simple beanbag genetic model of Hamilton [1964a, b]. The “facts” which this field and Hamilton’s models explain, social behaviour, were once regarded by evolutionary theory as anomalies and were ‘by far the most serious special difficulty, which (Darwin’s) theory had encountered’ (Darwin [1859], p. 242). More recently, the classical models sketched above create this anomaly themselves, for

If natural selection followed the classical models exclusively, species would not show any behavior more positively social than the coming together of the sexes and parental care (Hamilton [1964], p. 23). 1

However, as shown below, these very same models provide the framework necessary to turn this counter instance into a corroborating instance, for today this once anomalous data is, in general, regarded as confirming the genetical theory of evolution. Again this shift was not triggered by any data but by developing the positive heuristic of the programme. Most of the relevant data is still in the future for ‘social biology is still very far from providing the multiple measurements of fitness and the coefficients of relatedness that would permit exact tests of the theory’ (Hamilton [1972], p. 194). However, these old facts take on new meaning in light of Hamilton’s model ‘and we should certainly regard newly interpreted fact as a new fact . . .’ (Lakatos [1970], p. 157).

Kin selection models are directly descended from model (2} above and differ only in the formulation of the selection coefficients. The details are relatively unimportant and will not be presented here, except to give the basic motivation and the conclusions. A clear description of these models

1 Hamilton’s papers are reprinted in Williams [1971] and the page numbers given are from Williams’ book.
is given by Charnov [1977] or the reader can refer to Hamilton's original papers [1964a, b].

In (2), we hypothesize that the $A$ allele confers the phenotype on its carrier of behaving altruistically towards some relative, who thereby benefits by the amount $b$. The act is altruistic and so it is produced at some cost, $c$, to the altruist. The $a$ allele confers no such phenotype, so that $AA$ individuals are altruistic, $aa$ non-altruistic, and depending upon the degree of dominance, $Aa$ individuals may or may not be altruistic.

Due to kinship, relatives may also possess the altruistic $A$ allele with a certain probability $r$. The fitnesses $W_{AA}$, $W_{Aa}$, and $W_{aa}$ are then made functions of $c$ and $b$ to take into account (i) the loss of fitness to the altruists due to $c$ and (ii) the increase in fitness to the relative due to $b$. From (5) it is possible to get conditions on the $W$'s for $\Delta p$ to be positive and for the altruistic gene to increase in frequency. This condition interpreted in terms of $c$, $b$ and $r$ requires in most cases,¹ that the following inequality hold: $c/b < r$.

This simple inequality, now termed 'Hamilton's Rule', and the related concept of inclusive fitness have revolutionised studies of the evolution of social behaviour (see, for example, Wilson [1975]). Armed with Hamilton's Rule, sociobiologists can go to nature, make pairwise comparisons of genetic relationship, and then make statements or explanations about how social behaviour should evolve (Hamilton [1964], [1972]; Wilson [1975]; Trivers and Hare [1976]; to list only a few). There can be little doubt that this rule and the related concept of inclusive fitness presently provide most of the direction and focus for sociobiological research. It has generated a growing body of new experimental and field research. The new facts it has generated are too numerous to list but include the relationships between genetic systems, population structure, mating systems and sociality. Such empirical progress is a clear demonstration of the heuristic power of beanbag genetics.

4 CONCLUSIONS

We have identified the hard core of population genetics which consists of the view that gene frequency changes are evolution. We then monitored the construction of a "protective belt" around this core which consists of the various possible evolutionary forces which cause gene frequencies to change. These forces were added primarily through the development of beanbag genetics which functions as a strong positive heuristic in

¹ But see Cavalli-Sforza and Feldman [1978] and Michod [1979].
evolutionary biology. It was also argued that these various forces were added historically in an empirically progressive way—anticipating new data and explain away old anomalies.

Consequently from the point of view of Lakatos's model, the history of population genetics has behaved remarkably well. The issue of more general interest concerns the Neo-Darwinian Research Programme and not simply population genetics proper. Only a few subdisciplines outside of population genetics proper have been discussed here: speciation theory, sociobiology, and aspects of evolutionary ecology. I have argued that, within each of these subdisciplines, the gene frequency approach provides the basis for a framework within which problems can be formulated and solved (see also Ruse [1973]). The example of sociobiology is especially revealing since the subdiscipline is so new and its theoretical underpinnings in the gene frequency approach are so blatant. It is worth stressing again that the successes of the gene frequency approach in elucidating new aspects of social behaviour relies on an extension of the population genetics hard core to this particular field. Indeed, the very reason Wilson [1975] claims that social theory has been brought within the Synthetic Theory of Evolution is that the gene frequency approach provides a framework for understanding social behaviour. In the case of speciation theory, the theoretical underpinnings in the gene frequency approach are less obvious. This is to be expected because there has been time, since the pioneering work of Simpson [1944] and Mayr ([1942], [1963]), for the concepts first elucidated by the study of gene frequencies to be firmly incorporated within the discipline. Consequently they often appear only implicitly. Nevertheless, there can be no doubting the fact that these concepts (discussed in section 2(a)) originated in, and are still defined by, the gene frequency models. On the other hand, these concepts are mobilised in ways which are unique to the particular problems of the subdiscipline.

Each subdiscipline in evolutionary biology has, for the most part, its own, distinct subject matter. Consequently there must exist methodologies which are in certain respects unique to each field. Using these methodologies, perfectly good science can be done within each subdiscipline concerning the proximate causes of the phenomena of interest without any reference to gene frequencies. For example, in the study of behaviour important questions may involve elucidating the hormonal control of behaviour or in molecular biology elucidating the mechanism of recombination. However, if evolutionary reasons are sought for why recombination or certain behaviours exist at all, each aspect of the phenomenon must be weighed for its possible adaptive significance in relation to other evolutionary forces. This procedure often explicitly involves positing
genes for the various alternatives and determining which genes will in-crease in frequency. Other times the gene frequency approach is implicit, as is the case with the strategic reasoning often used in evolutionary ecology.¹

However, to argue convincingly for the generality of the gene frequency approach, it must be shown that all contributions to modern evolutionary biology rely, either explicitly or implicitly, on the study of gene frequencies. Although I feel the examples discussed here support this view, I realise these few examples are insufficient for the task. Indeed I have some doubts whether it will be the case that a single hard core could be shown to apply in all areas of such a diffuse discipline as evolutionary biology and still maintain some historical integrity and continuity (Laudan [1977]). This, I think, requires further work.

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¹ This reasoning gets much of its justification from Fisher's Fundamental Theorem, a result of beanbag genetics which I have not discussed.
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