Neophenogenesis: A Developmental Theory of Phenotypic Evolution

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Abstract:

An important task for evolutionary biology is to explain how phenotypes change over evolutionary time. Neo-Darwinian theory explains phenotypic change as the outcome of genetic change brought about by natural selection. In the neo-Darwinian account, genetic change is primary; phenotypic change is a secondary outcome that is often given no explicit consideration at all. In this article, we introduce the concept of neophenogenesis: a persistent, transgenerational change in phenotypes over evolutionary time. A theory of neophenogenesis must encompass all sources of such phenotypic change, not just genetic ones. Both genetic and extra-genetic contributions to neophenogenesis have their effect through the mechanisms of development, and developmental considerations, particularly a rejection of the commonly held distinction between inherited and acquired traits, occupy a central place in neophenogenetic theory. New phenotypes arise because of a change in the patterns of organism-environment interaction that produce development in members of a population. So long as these new patterns of developmental interaction persist, the new phenotype(s) will also persist. Although the developmental mechanisms that produce the novel phenotype may change, as in the process known as "genetic assimilation", such changes are not necessary in order for neophenogenesis to occur, because neophenogenetic theory is a theory of phenotypic, not genetic, change.

Article:

INTRODUCTION

A central problem for evolutionary biology is to explain the origin of phenotypic diversity among organisms. In its early years, before the rediscovery of Mendel's genetic work, evolutionary theory was almost entirely a theory of phenotypic change. Darwin's formulation of natural selection required that phenotypic variations exist in a population, but offered no account of the origin of such variations, beyond postulating "a tendency to vary, due to causes of which we are quite ignorant" (Darwin, 1872: 146). The idea that evolutionary change might involve anything other than change in the observable characteristics of organisms had to await Johannsen's (1909, 1911) distinction between the genotype and the phenotype, and the rediscovery of Mendel's (1866) experiments on inheritance in the early 20th century.

As the science of genetics advanced, Darwin's "tendency to vary" became identified with the processes of mutation and recombination. This opened the door for theories of population genetics, which explained evolutionary change in terms of selection among genetic variants, rather than among phenotypic variants as proposed by Darwin. With the discovery of DNA by

Watson & Crick (1953), the genetic theory of natural selection was placed on a molecular foundation and the current neo-Darwinian synthesis was completed. In the process, however, something had been lost; namely, the focus of evolutionary explanation on the phenotype. As Ho & Saunders (1979: 575) remark, neo-Darwinism "is primarily a theory of genes, yet the phenomenon that has to be explained in evolution is that of the transmutation of form". Neo-Darwinism treats phenotypic change solely as the outcome of genetic change, the result of natural selection among members of the population. Although genetic change is no doubt important in the changing phenotypic makeup of an evolving population, in neo-Darwinian theory it has become the only source of phenotypic evolution (Saunders & Ho, 1982).

In this article, we propose that it is more useful to view the natural selection of genetic variants as but one component of a broader process of phenotypic change that we call neophenogenesis, the origination of novel phenotypes that persist over evolutionary time. The changes that evolutionary theory attempts to explain are primarily changes in the phenotype—in the anatomy, physiology, or behavior of organisms over long periods of time. Such change may, of course, be brought about by the natural selection of genetic variants, but there are other mechanisms of neophenogenetic change (Novak, 1982b; Socha & Zemek, 1982), and it is these extra-genetic mechanisms, and their relationship to genetic change, that are our primary concern here.

Developmental Mechanisms and Evolutionary Change

An important current theme in evolutionary biology is that explaining phenotypic change requires us to pay close attention to the mechanisms of development (e.g. Alberch, 1980; Alberch et al., 1979; Bonner, 1982; Fallon & Cameron, 1977; Gould, 1989; Gustafson et al., 1985; Hall, 1975, 1984; Oster et al., 1988; Raft & Kaufmann, 1983; Shubin & Alberch, 1986). Developmental mechanisms are responsible for producing the phenotype and so, as de Beer (1940) pointed out long ago, evolutionary change in the phenotype can only come about by change in development (for an even earlier statement, see Mivart, 1871). But the developmental theory that underlies much of the current work is deeply problematic, because it accepts a relatively strong version of the distinction between inherited and acquired traits. Inherited traits are attributed to the developmental action of the genes, acquired traits to environmental influences experienced during the course of individual development. There are compelling arguments, summarized below, against this view of development and in this article we show how an alternative, and better supported, developmental theory leads to a quite different explanation of evolutionary change in the phenotype. The inherited/acquired distinction, however, is deeply rooted in the history of modern evolutionary theory, growing out of the division between Darwinism and neo-Darwinism that arose in the late 19th century, and that eventually banished Lamarckian, or quasi-Lamarckian, mechanisms from evolutionary biology. It is a central component of modern evolutionary theory, albeit one that is only rarely made explicit.

Darwin's view was that "natural selection has been the main, but not the exclusive means of modification" (Darwin, 1872: 483). In addition to selection among the spontaneous variations that are now attributed to mutation and recombination, Darwin believed that the effects of use and disuse could be inherited, a Lamarckian evolutionary mechanism that came to assume progressively greater prominence in successive revisions of the Origin, and that culminated in his theory of pangenesis (Darwin, 1868). This theory, along with others that proposed the inheritance of acquired characters, was dealt a devasting blow by Weismann's (1893) theory of

the germ plasm, which erected an impenetrable barrier between the germ-cell line and the somatic tissues. According to Weismann, the somatic and germ-cell lines are entirely separate; no change in the former can ever be transmitted to the latter. The germ-plasm theory was eventually accepted by biologists and later received confirmation in the "central dogma" of molecular genetics, according to which information flows only from DNA (germ) to protein (soma) molecules, not in reverse. Weismann's theory produced a split among evolutionary biologists, separating those who believed (with Darwin) that processes other than natural selection are involved in evolutionary change from those who followed Weismann in arguing that the isolation of the germ-cell line means that natural selection among spontaneous heritable variants is the only mechanism of evolution. Thus, evolutionary biologists became divided into what Romanes (1897) called Darwinists (such as Darwin, Romanes, and Spencer) and ultra-Darwinists (such as Weismann, Wallace, and Lloyd Morgan).

In the ensuing years, ultra-Darwinism (or neo-Darwinism as it came to be called) gradually became pre-eminent, incorporating the findings of Mendel and later of molecular biology into the modern evolutionary synthesis. The hallmark of neoDarwinian theory was thus from the beginning a belief that acquired characters cannot be inherited and that belief requires, of course, the assumption that acquired and inherited characters can be distinguished in the phenotype. Starting from that distinction, the neo-Darwinian argument is that natural selection accounts for evolutionary change in inherited characters, selecting among their alternate forms as those forms are made available by mutation and recombination. Acquired characters are not subject to natural selection because they are transitory and have no genetic basis. Since they are not inherited (and cannot become inherited, according to both the germ-plasm theory and the central dogma), they must arise anew in each generation and do not evolve (e.g. Ayala & Valentine, 1979: 19). Thus, in neo-Darwinism, "evolution" has become synonymous with genetic change: "Evolution is a change in the genetic composition of populations" (Dobzhansky, 1951: 16; emphasis in italics added)*.

Sometimes, however, evolutionists adopt a different position; namely, that evolutionary theory must ultimately explain phenotypic change, and that although genetic models are an important part of that explanation, they cannot provide the entire account. For example, Simpson (1953a: 5) wrote that "genetic factors are not important to us for their own sake, but only because they are among the various determinants of phenotypic evolution" (emphasis in italics added). More recently, this view has been echoed by Lewontin (1974: 19), who suggests that "the real stuff of evolution" are changes in phenotypic, not genotypic characters. In a recent "post- synthesis clarification," Mayr (1988: 530) has expressed sympathy with the position of evolutionary naturalists that evolution "is not merely a change in the frequency of alleles in a population, as the reductionists asserted, but is at the same time a process relating to organs, behavior, and the interactions of individuals and populations."

The position expressed by Simpson, Mayr, and Lewontin, and by some other authors (e.g. Bock, 1979; Futuyma, 1979: 21; Lambert & Hughes, 1984; Ho & Saunders, 1979, 1982), may be summarized as follows: Evolutionary theory must ultimately explain phenotypic change, and

* Although Dobzhansky's definition is canonical, essentially the same one can be found in other leading statements of neo-Darwinism spanning 30 years (e.g. Simpson, 1959: 15; Grant, 1963: 125; Mettler & Gregg, 1969: 59; Dawkins, 1976: 48; Dobzhansky et at, 1977: 8; Ayala & Valentine, 1979: 18; Lumsden & Wilson, 1981: 371).

although genetic models are an important part of that explanation, they cannot provide the entire account of change in the phenotype over evolutionary time. That task will require a theory that incorporates all of the mechanisms that may produce phenotypic change and, in particular, that explains the relationship between genetic and extra-genetic sources of such change[†].

The interest in development shown by evolutionary biologists over the past few years is an important step towards bridging the gap between genotypic and phenotypic change in a population. But that bridge will only stand if it is buttressed by a secure developmental theory. In many evolutionary discussions, development is represented as the unfolding of a genetic program (Alberch, 1982; Mayr, 1974; Smith-Gill, 1983). According to this programmatic view of development, some characters (those that evolve) develop under genetic control, whereas others depend on input from the environment. From this perspective, the task of developmental studies is to reveal the mechanics of such developmental unfolding, showing how the genes act on developmental processes rather than directly on adult phenotypic characters, and how development itself is constrained. But the development of evolving characters is always seen as being under tight genetic control, as it must be if the neo-Darwinian distinction between inherited and acquired characters is to be preserved. The importance of the distinction can be further appreciated by noting the existence in the neo-Darwinian lexicon of terms that explicitly distinguish inherited (genetic) traits from acquired (environmental) ones, such as the phenocopy (an environmentally induced phenotypic copy of a mutant genetic trait) and the ecophenotype (a novel phenotype produced by the environment rather than the genes). The existence of such terms presupposes the view that inherited traits can be distinguished from acquired traits (Oyama, 1981).

The problem is that the inherited/acquired distinction itself is invalid. It has produced innumerable confusions, errors, and omissions in developmental theory (especially in the development of behavior; see Gottlieb, 1976; Johnston, 1987, 1988; Kuo, 1967; Lehrman, 1953, 1970; Oyama, 1982, 1985; Schneirla, 1956) and its retention in evolutionary biology can only lead to similar problems there. The theory of neophenogenesis is an attempt to incorporate an alternative view of development into evolutionary biology, but doing so will require that we abandon the neo-Darwinian distinction between inherited and acquired characters.

CRITICISMS OF THE INHERITED/ACQUIRED DISTINCTION IN DEVELOPMENTAL THEORY

Perhaps the clearest and most forceful exposition of the inherited/acquired distinction in developmental theory is to be found in the literature on behavioral development, where it is usually presented as a dichotomy between learned and innate behavior. For example, Lorenz's (1935, 1965) theory of instinct required an absolute distinction between those elements of behavior that are specified by the genes and those that arise in the course of individual experience. The neo-Darwinian origins of Lorenz's distinction can clearly be seen in his treatment of behavioral evolution (Lorenz, 1937), in which he forcefully and explicitly rejects any evolutionary connection between the two kinds of behavior. Lorenz's learned/innate

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[†] In a recent "post-synthesis clarification", Ernst Mayr has noted the conflicting views of naturalist and reductionist biologists regarding evolutionary change. According to the naturalists, evolution "is not merely a change in the frequency of alleles in a population, as the reductionists asserted, but is at the same time a process relating to organs, behaviors, and the interactions of individuals and populations" (Mayr, 1988: 530).

dichotomy was vigorously criticized by developmentalists such as Lehrman (1953, 1970), Schneirla (1956, 1966), Jensen (1961), and Gottlieb (1970) who, building on Kuo's (1921, 1929) pioneering insights, argued that all behavior, and indeed all phenotypic characters, arises in development as the result of an interaction between the animal and its environment. The genes play a role in this interaction, one that is still hard to specify in any detail, but they do not directly determine any aspect of the phenotype. Lorenz (1965) responded that, to the contrary, the genes encode information that requires only the environmental conditions necessary to sustain life in order to determine in detail those components of behavior called "innate" or "instinctive". This information is in the form of a genetic program (see also Mayr, 1974) that unfolds mechanically in the course of strictly determined maturation.

The view that development involves a programmatic unfolding of the phenotype is entirely consistent with the neo-Darwinian account of evolution, because it allows phenotypic characters to be divided into those that are specified (programmed) by the genes and those that depend on the environment. The interactionist view, however, which denies that any such division can be made, is much harder to reconcile with neo-Darwinian thinking because it rejects the distinction between acquired and inherited characters. This may account for the tremendous resistance to interactionist developmental thinking in the behavioral sciences, which in their modern form grew out of the neo-Darwinian evolutionary biology of the late 19th century (see Johnston, 1987, 1988 for documentation of this resistance). None the less, the interactionist position is a powerful and compelling alternative to the dichotomous view characteristic of neo-Darwinian thinking. Our current understanding of gene action in development does not allow for direct genetic specification of any phenotypic character beyond the level of protein structure (and even that specification is influenced by intracellular environmental factors such as pH and temperature; Pritchard, 1986); and the route from protein structure to gross anatomy and behavior is long and exceedingly complex. If the interactionist position (in some version) is accepted as a more adequate account of development than the dichotomous view, then evolutionary biology can hardly maintain the distinction between inherited and acquired characters as it attempts to integrate the results of developmental analyses into its account of evolutionary change.

Development and Evolution in Neophenogenesis

Any account of change in the phenotype over evolutionary time must recognize that the characteristics that change are themselves the product of development. Thus, to account for phenotypic change, we must consider and integrate all of the ways in which changes in development may be brought about. Neo-Darwinian theory incorporates development by distinguishing two kinds of phenotypic traits (inherited and acquried) and offering an account of evolutionary change in only one of these. Our task, by contrast, is to offer an account that proceeds from the position that no such distinction is possible or necessary.

The development of an organism is determined by interactions among the various components of the organism and its environment, in which genes, hormones, diet, physical factors, exercise, sensory experience, social interactions, and numerous other factors play important roles (Bateson, 1987; Gottlieb, 1976, 1981; Lehrman, 1953, 1970) (see Fig. 1). A change in any of these components may modify the phenotype; from the interactionist perspective, there is no justification for making a priori judgments as to which of them are most likely to produce adaptively significant changes in the phenotype. The relevant factors can only be determined by

experiment, and will likely be found to vary from species to species and from time to time during development. In particular, there is no warrant for singling out genetic change as being more relevant to the analysis of phenotypic change than

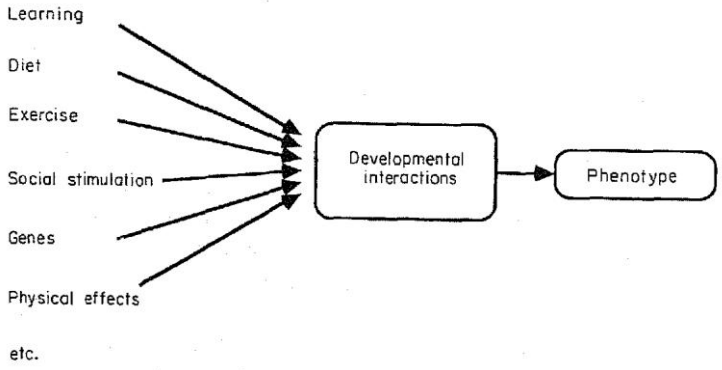

Fig. 1. Development of the phenotype results from interactions among numerous components of both the organism and the environment. Altering any of these contributing factors, not only the genes, may produce change in the phenotype; if the alteration persists, the phenotypic change may persist long enough to be evolutionarily significant.

are changes in any other of these factors. Neo-Darwinian evolutionary theory, of course, does make such an a priori assessment of evolutionary relevance, in asserting that only genetic changes produce true evolutionary change. So long as evolution is defined as change in the genetic makeup of populations, this assertion is necessarily true (by definition), but since neophenogenesis is defined differently and more broadly, such a priori assessments need not, indeed cannot, be made

There is a terminological issue that needs to be addressed directly here, because it may result in the arguments we present being unfairly dismissed as inconsequential. Although formal definitions of evolution in neo-Darwinian theory invariably specify genetic change as being necessary for evolution to occur, less formal use of the term frequently refers to any phenotypic change that persists over a relatively long period of time. This latter sense of "evolution" is in effect when we read a description of "evolutionary change" in the primate brain, for example, based on evidence from comparative anatomy and fossil reconstruction. We have no idea to what extent such changes in phenotype involved genetic change in the populations involved, and so they should really be referred to as "phenotypic changes that may, to some extent, be evolutionary". Of course, no one is likely to use such a clumsy circumlocution, and so changes of this kind are almost always referred to as evolutionary, even though evidence about the mechanisms that brought them about is rarely available (Hailman, 1982). Thus, "evolutionary change" has come to have two meanings that are hardly ever distinguished, except when the explanatory hegemony of neoDarwinian theory is threatened. If we offer an account of some change in the phenotype that clearly (at least by hypothesis) does not involve genetic change, a neo-Darwinian evolutionary biologist is likely to retort that such changes, not being "evolutionary" in the formal sense, need not concern him/her. But that same biologist is likely to turn around and describe as "evolutionary" in the informal sense many phenotypic changes whose origin is in fact unknown. Because of this terminological ambiguity, neo-Darwinian theory succeeds in defining for itself two explanatory domains: a formal domain whose extent is largely unknown because we rarely know what genetic changes have taken place in natural populations; and an informal domain that encompasses all phenotypic changes not specifically

shown or assumed to be extra-genetic in origin. Unless this problem is explicitly recognized, a theory of neophenogenesis (such as is proposed here) runs the risk of being dismissed because it fails to address evolutionary problems. This is only true if "evolutionary" is construed in the formal sense; in the informal sense of "evolutionary" . It is clear that neo-Darwinian theory itself fails to address many "evolutionary" problems that might be encompassed by a theory of neophenogenesis.

A change in the environment of a population may alter the phenotypes of individuals developing in that environment without being a source of natural selection; that is, without changing the relative reproductive successes of genotypes in the population (see also Novak, 1982a, b; Socha & Zemek, 1978). An environmental change that does have selective consequences may also affect phenotypic development, and its selective and developmental consequences are likely to interact in complex ways that are at present very hard to predict (see further below). Let us illustrate our view of neophenogenesis by considering an environmental change that we presume not to have any selective consequences; later we will add selective consequences to the picture.

A NEOPHENOGENETIC SCENARIO-DIETARY CHANGE IN A RODENT POPULATION

Suppose that a population of rodents whose diet consists mainly of soft vegetation encounters a new food source in the form of hard but highly nutritious seeds. Evidence from studies of food selection in rodents (Kalat, 1985; Richter, 1947) suggests that the animals will initially sample small amounts of this new food, and then gradually increase its representation in their diet, especially if the seeds provide a rich source of some important nutrient. Because young rodents typically acquire their initial food preferences from their parents, especially their mothers (Galef, 1985), the new food habit will tend to stabilize as it spreads through the population, so long as the seeds remain available. Because animals will be eating these seeds during much of their lifetime, the new diet may have developmental effects on the phenotype that go beyond simply the establishment of a new food habit. Diet has consequences for body size and composition, fecundity, age of sexual maturation, nervous system development, and other aspects of the phenotype with far-reaching consequences for the animal's adaptation to its environment.

As well as these direct effects of diet on development, there are also indirect effects produced by the animals' interaction with their new diet. For example, as the diet changes from relatively soft to much harder items, the mechanical stresses exerted on growing jaw tissues during development will change. Patterns of bone growth are partly determined by forces exerted on the growing bone (e.g. Frost, 1973; Herring & Lakars, 1981; Lanyon, 1980), and so the skeletal anatomy of the jaw will be different in animals that experience relatively hard and relatively soft diets during early life. Functional demands such as this, which arise out of the interaction between the developing animal and its environment, are central to the theory of neophenogenesis being presented here. To that extent, the theory resembles Lamarck's theory of evolution, which also emphasized the role of animal-environment interactions in producing phenotypic change. However, whereas Lamarck proposed (following what were then widely accepted beliefs; Burkhardt, 1977; Richards, 1987) that the effects of such interactions could be inherited by subsequent generations, our theory requires no such mechanism. It may be, as we discuss below, that the developmental mechanisms that produce the phenotypic character in question (such as the form of the jaw in our hypothetical example) may subsequently change, perhaps as a result of natural selection in the population. But our account, by denying the distinction between acquired

and inherited (or genetic) traits, is not required to postulate "genetic assimilation" of developmental modifications, in the manner of Baldwin (1986), Cope (1887), Matsuda (1982, 1987), Morgan (1896), Osborn (1986), Schmalhausen (1949), and Waddington (1957). In that respect, our account differs from some other recent critiques of neo-Darwinism (e.g. Rosen & Buth, 1980; Steele, 1979, 1981), many of which also require a mechanism by which developmental modifications may eventually become inherited. To reiterate our position: Changes in either genetic or other influences on development may lead to relatively enduring transgenerational change in the phenotype which, in our definition, constitutes neophenogenesis. Before describing how we can incorporate natural selection into our account of neophenogenesis, let us consider some objections that might be raised against our position thus far.

OBJECTIONS TO NEOPHENOGENESIS

Objection 1

A new functional demand merely elicits a different developmental response from an unchanged organism; it does not produce real change in the organism itself.

The cogency of this objection depends on what is meant by "the organism". From the standpoint of neophenogenesis, the organism is the phenotype and new functional demands certainly can produce change in the phenotype. Only if "the organism" is taken to refer to the genotype does this objection carry any force, but as already noted, the aim of neophenogenetic theory is to explain phenotypic, not genotypic change. At any stage in the phylogeny of a lineage, normal development of the individuals that it comprises depends on their having both a normal genotype and a normal functional context for development. Enduring changes in either the genotype or the functional context may produce stable, transgenerational phenotypic change, and the effects of both require explication. All such phenotypic changes are "real" changes, regardless of their source.

The appeal of this objection depends quite strongly on one's view of the role played in development by the normal environment. On one view, the normal environment may be seen as having an essentially passive or "permissive" role in development, merely allowing the endogenous maturation of a normal phenotype (Lorenz, 1965; cf. Gottlieb, 1970). On that view, it is the genotype that is primarily responsible for the characteristics of the phenotype, and only genetic changes will appear to be of fundamental importance in producing phenotypic change. Alterations to the environment simply block or interfere with normal development, producing developmental aberrations of little or no interest. As argued above, this view finds little support from modern developmental theory, which emphasizes the paramount importance of functional interactions with a normal environment during development. Although the role of such interactions in particular instances of development continues to be debated, no adequate theory of development can exclude them from consideration. The task of a theory of neophenogenesis will be to work out the implications of this fact for explaining change in the phenotype. It is clear from the outset that if we grant that normal functional interactions play an important role in constructing the species-typical phenotype, then we must also grant that a change in those interactions may play an important role in changing the phenotype, and in maintaining that change in subsequent generations. Those are the defining characteristics of neophenogenesis.

Objection 2

Functional demands on the organism are readily reversible and so their developmental effects are transient and of little long-term significance; genetic changes are more permanent.

The issue of the irreversibility of evolutionary change is fraught with all sorts of problems, but there is clearly no consensus that change must necesarily be irreversible in order to be evolutionary (Simpson, 1953a; Futuyma, 1979). Although the effects of altered functional demands are more likely to be reversible than are those of genetic changes, this is no reason to consider them on that account as insignificant contributions to neophenogenetic change. The important point is whether changes in functional demands are necessarily, or even typically, transient. If such changes do in fact endure for appreciable periods of time, then they may indeed contribute significantly to long-term changes in the phenotype. It is true that phenotypes themselves do not persist—they must be constructed anew in each generation. However, a changed phenotype will continue to recur in subsequent generations to the extent that the same developmental factors prevail that gave rise to it originally in some previous generation. Of course, since genes do not, in and of themselves, make phenotypes, this same developmental contingency also holds for the trans- generational stability and persistence of phenotypic changes that result from genetic change.

Although many changes in functional demands are undoubtedly too transient to be of much long-term significance, others may be identified that are clearly very long-lasting. The dietary change considered in the preceding section is one such example: The change in functional demand will persist for as long as the new food continues to be a part of the animals' diet, which may be many hundreds or thousands of generations. Another example involves the altered functional demands imposed by the transition from an aquatic to a terrestrial habitat. One major change is that the skeleton of a terrestrial animal is subject to a completely different set of mechanical influences during development because of its changed locomotor patterns and the increased load-bearing demands it experiences. It is well known that the stresses and strains produced by muscle contraction and Ioad-bearing during normal locomotion in young animals are important in determining the mature form of the skeleton (Frost, 1973; Murray & Selby, 1930; Saville & Smith, 1966; Storey, 1975; Thompson, 1942: 975). Such functional demands have been a part of the normal developmental context for terrestrial animals for a very long time, and must have played some role in the modifications of the vertebrate skeleton that occurred during the transition from water to land during the Devonian period. A similar point can be made in regard to the change from quadrupedal to bipedal locomotion that occurred during the evolution of many different lineages, including reptiles, marsupials, and primates. If some members of such a species change their locomotor habits, perhaps to allow invasion of a new niche or adaptive zone (Mayr, 1963: 604), then they will experience a new set of stresses and strains that will contribute to the development of a different skeletal anatomy (Amtmann, 1974; Appleton, 1922, 1925; Gordon et al., 1989; Kiiskinen, 1977; Lanyon & Bourn, 1979; Saville & Smith, 1966; Simon, 1978).

It is important to note that we do not claim that new functional demands are all that is involved in changes of this sort. Quite clearly, any change as major as that from a quadrupedal to a bipedal style of locomotion will almost certainly involve genetic change as well. Our point is simply that the developmental effects of changes in functional demand are real and important constituents of neophenogenesis, and must be incorporated into any account of how phenotypic change occurs.

Their role as a pervasive and important factor in long-term change in the phenotype cannot be dismissed on the grounds that they are in principle more readily reversible than genetic changes, because many changes in functional demand have in fact not been reversed.

Objection 3

The effects of new functional demands cannot be inherited.

This objection is usually presented as a corollary to the one just discussed: Functional changes only have transient effects because they cannot be inherited. We have already presented arguments against a view of development that distinguishes acquired from inherited traits and those arguments should be borne in mind when evaluating this objection. Interactionist theory implies that there are no "inherited" traits, if by that is meant traits that arise solely from the genes. If the inherited/acquired distinction is abandoned, this objection loses much of its force because the idea of an "acquired" change becoming "inherited" requires a rather radical reinterpretation. One such interpretation might rephrase the objection as follows: There is no mechanism by which a phenotypic change that is initially evoked by some specific environmental stimulus can come to arise in development without the need for the originally evoking stimulus. However it is phrased, this objection is the one that biologists who claim the importance of function and individual development in evolution have tried the hardest to overcome, because it seems to be the most damning. Although Lamarck (1809, 1815) simply presumed, in keeping with the conventional wisdom of his day, that acquired changes could be inherited, others have proposed a variety of mechanisms for the inheritance of acquired characters (e.g. Cook, 1977; Darwin, 1868; Gorczynski & Steele, 1981; Ho et al., 1982; Matsuda, 1987; Rosen & Buth, 1980; Steele, 1979; Vosburgh, 1981; Waddington, 1953, 1957). Associated with these attempts are rebuttals by neoDarwinian theorists, who argue that the proposed mechanisms are unworkable in principle, refuted empirically, or unimportant to the process of evolutionary change (e.g. Brent et al., 1981; Fitch, 1982; Nisbet-Brown & Wegmann, 1981; Simpson, 1953b; Smith, 1981; Weismann, 1893; Williams, 1966).

While skepticism towards some of these mechanisms may be in order, our argument does not stand or fall on the outcome of this debate. Our concern is with the process of neophenogenesis, with long-term change in the phenotypic makeup of populations, not with changes in their genetic makeup. Species-typical development requires both a normal genotype and a normal environment (including a normal set of functional interactions) and change in either of these may alter the development of the phenotype. The regular production of a modified phenotype in successive generations qualifies as a neophenogenetic change, whether the modification was brought about by a genetic change in the population, by a change in the functional demands on members of the population, or by a combination of the two.

Objection 4

Most developmental responses to a change in the environment are not adaptive.

Williams (1966: 75) raised this objection, among others, to Waddington's theory of genetic assimilation as a component of evolutionary change. Waddington (1953, 1957) showed that the phenotypic effects of heat shock and ether administration in *Drosophila* (respectively, the lack of a posterior crossvein in the hindwing, and the production of a second thorax) eventually

appeared spontaneously, without the environmental stimulus, in stocks that had been selected for a strong developmental response to the stimulus. Williams pointed out that since the crossveinless and bithorax phenotypes are not adaptive responses to the stimuli that produced them, their occurrence would be of no adaptive consequence for the population.

We agree with Williams that these particular phenotypes, and doubtless many others, are not adaptive responses to the changes that produced them. We would point out, however, that most mutations (the basis of neo-Darwinian explanations of phenotypic change) are not adaptive either. The neo-Darwinian argument rests on the claim that some mutations are adaptive and will be favored by selection when they occur. Not all evolutionary change is adaptive; neo-Darwinian theorists accept that some (indeed most) populations become extinct, partly because of a lack of adaptive genetic variation. Similarly, not all neophenogenetic change is adaptive; populations may become extinct because their members respond to a change in the environment by producing a maladaptive phenotype.

Objection 5

Phenotypic changes that do not involve genetic change are likely to be small and of very little evolutionary significance.

The question of how much change must occur in order to be considered significant is problematic from any theoretical perspective and clearly depends, to some extent, on one's interests. A taxonomist may find the small changes in CNS organization and muscle anatomy associated with a change in feeding habits relatively insignificant, whereas those same changes may be of primary importance to a behavioral ecologist interested in explaining the adaptation of a population, to a new food supply. This will be true regardless of whether the anatomical changes were caused by a point mutation or by an altered set of functional demands on the developing organism. Questions of what constitutes -significant change", it seems, must be settled by both neo-Darwinian and neophenogenetic theory, but cannot be used to decide between them. In any event, only systematic experimentation can determine the magnitude of phenotypic changes that are not accompanied by genetic change.

The first three of the objections just discussed all derive from the idea, deeply embedded in neo-Darwinian theory, that genetic change is somehow more "real" or "fundamental" than phenotypic change. The theory of neophenogenesis being advanced in this paper adopts a very different perspective, in which phenotypic change is the primary fact to be explained and genetic change is only one of many contributing factors. It must not be thought that we are advocating a view in which genetic change, and the natural selection of alternative genotypes, is irrelevant to neophenogenesis. But neo-Darwinian theory places natural selection at the center of its account of change in the phenotype, whereas our theory assigns a different, athough still significant, role to selection (cf. Fulk, 1989; Saunders & Ho, 1982; Michaux, 1988).

To understand the role that natural selection plays in a theory of neophenogenesis it is necessary to consider two important distinctions: That between the organism and the population, and that between the sources and the consequences of variation in the population. Neither of these distinctions, of course, is original. Both are central to the neo-Darwinian framework. In neo-Darwinism, the significant component of evolutionary change (natural selection) takes place in

populations; organisms are simply the carriers of the variations among which selection occurs (see Dawkins, 1976, 1982 for especially forceful statements of this perspective). The variations in question are genetic variations (modifications of DNA sequences brought about by mutation and recombination) having, as their evolutionarily significant consequence, different probabilities of survival and reproduction in the population. Although these different probabilities exist because of the characteristics of individual phenotypes, the neo-Darwinian focus is sharply on the population; organisms (phenotypes) are in many respects passive bystanders in the evolutionary play, their changing character the reflection of hidden but fundamental reshapings of the population's gene pool.

A theory of neophenogenesis, by contrast, shifts the focus to the organism, because in this theory the fundamental reshapings are reshapings of the phenotype itself; restructuring of the gene pool, if it occurs, is an outcome of developmental changes in the phenotypes of organisms that make up the population. Although it is conventional in neo-Darwinian theory to explain changes in phenotype as the outcome of genetic change in a population (especially changes produced by natural selection), in fact a phenotypic change must come before natural selection is possible. This is not as radical a statement as it may sound; it amounts to no more than a recognition that variation must exist in a population before selection among the variants can occur. In neo-Darwinian theory, the source of all evolutionarily relevant variation is genetic (mutation and recombination)—necessarily so, because neo-Darwinian evolutionary change is defined as change in the genetic composition of the population. For most neo-Darwinian theorists, the origin of this variation is unproblematic and genotypes having the requisite phenotypic effects are typically proposed with abandon (Axelrod & Hamilton, 1981; Maynard Smith, 1984; see Johnston, 1984). In neophenogenetic theory, however, it will not do simply to postulate the prior existence of needed variation in order to explain phenotypic change. If we wish to explain why organisms in some taxonomic group possess a particular phenotypic character, we must explain its first appearance in at least one organism in a population. That problem is a developmental one, which may or may not involve genetic change.

Once it has been shown how the character first appeared, the question can then be raised whether there is heritable variation for its development in the population and, hence, whether it can have spread in the population by natural selection. Notice that even if there is no such heritable variation, and even if there is no selection (because no differential reproductive success is associated with possession of the character), our theory can still explain the perpetuation of the character by pointing to developmental processes that will produce it under some altered set of conditions. The explanatory scope of neophenogenetic theory is thus considerably broader than is that of neo-Darwinian evolutionary theory. For example, several authors (Alberch, 1981, 1982; Gould, 1980; Maynard Smith et al., 1985; see Gould, 1989; Stearns, 1986) have recently pointed out that developmental mechanisms constrain the range of phenotypic variation on which natural selection can act. Such constraints lie outside the explanatory domain of neo-Darwinism (Gould, 1989); they define the starting conditions under which neo-Darwinian evolutionary mechanisms operate, but do not explain why those particular conditions exist. In neophenogenetic theory, the developmental mechanisms become part of the explanation for change in the phenotype over evolutionary time. However, from a neophenogenetic perspective it would be inappropriate to consider these mechanisms as constraints on anything. They are integral to the process of phenotypic change and their operation explains the origin and diversification of new phenotypes.

Of course, it may be that some of these phenotypes are reproductively more successful than others and if that is the case, and if the variation is heritable, selection will occur and the frequency of the existing variants in the population will change. But for further change in the available phenotypes to occur, there must be additional modifications in the developmental processes that bring them into being; that is, neophenogenesis, not simply selection, must continue for continued phenotypic change to be possible.

Selection is often viewed as a process that can shape or mould phenotypes by acting on existing variation, which is assumed to be random (though possibly constrained). This view ignores the individual organisms whose characters we are trying to explain and focuses solely on the population. Selection can indeed shape the distribution of phenotypes in a population, but it cannot shape the individual phenotypes themselves, because it is a consequence, not a cause, of phenotypic change. If our aim is to explain the origin of phenotypic characteristics (such as wings, eyes, or patterns of behavior) rather than just to explain changes in their distribution in the population, a neophenogenetic analysis, involving both developmental change and natural selection, is essential.

The developmental and selective components of neophenogenesis are not independent of one another, because the environmental change that elicits a novel developmental reponse from members of a population may also produce selection among them (e.g. Barnett & Dickson, 1986; Reznick & Bryga, 1987: fig. 3). Consider the dietary change discussed earlier (p. 477) as an example of extra-genetic neophenogenetic change. A change from relatively soft to relatively hard food may not only produce developmental remodeling of muscular and skeletal anatomy without any accompanying genetic change, but may also favor the reproduction of some genetic variants over others. For example, genotypes that exhibit a particularly adaptive developmental response to the new diet may be selectively favored over others that exhibit less adaptive responses (cf. Baldwin, 1896; Morgan, 1986; Waddington, 1957). Such "genetic assimilation" (Waddington, 1957) may or may

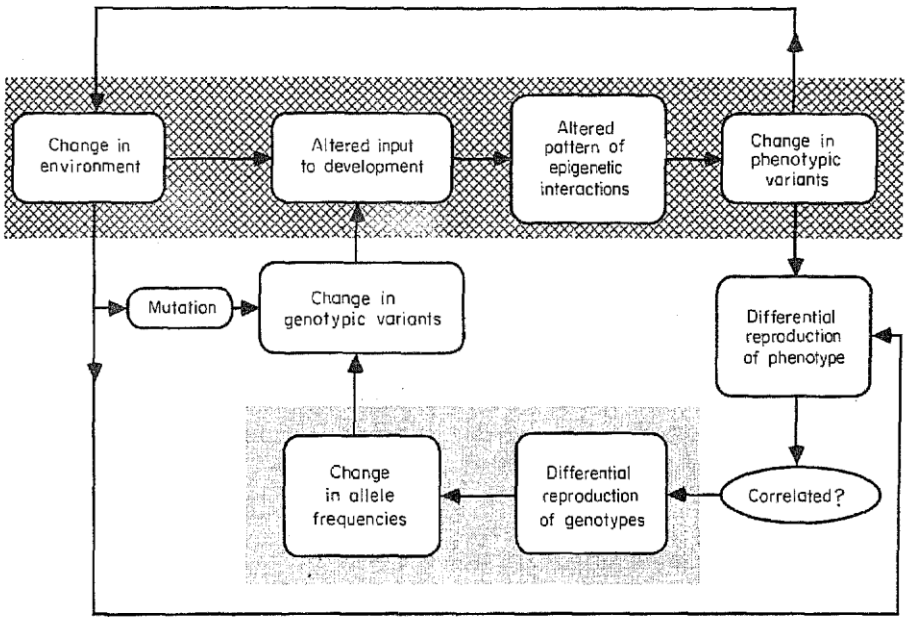

FIG. 2. As described in this article, neophenogenesis (defined as a persistent transgenerational change in the phenotypes of a population) incorporates both genetic and extragenetic change. All phenotypic change requires a change in the pattern of interactions that constitute development; such a change in development may occur if either the genetic or the environmental inputs to development are altered. Neophenogenesis requires only that the changed input to development persist across generations; this may occur without genetic change if the environmental change persists (hatched rectangle). If some genetic variation is correlated with the phenotypic variation produced by a change in development, then natural selection may result (shaded rectangle) and subsequent phenotypic change may occur because the pool of genotypic variants in the population has changed. Notice that natural selection is depicted as a consequence of phenotypic change and that neophenogenesis does not require that environmentally produced changes in phenotypes become "genetically assimilated".

not take place as phenotypes change over evolutionary time; its occurrence is neither required nor ruled out by our theory. Thus, extra-genetic and genetic (evolutionary) change, both brought about by an altered diet, may co-operate to produce a change in the phenotypes in the population. Neophenogenesis incorporates both kinds of change (Fig. 2).

The Extra-genetic Component of Neophenogenesis: Empirical Evidence

Our argument thus far has been largely theoretical. We have proposed that current developmental theory, by denying any basis for a strict division between inherited and acquired phenotypic traits, casts doubt on the adequacy of natural selection as a sole explanation of phenotypic change. We now present some empirical evidence suggesting that significant phenotypic change can indeed be brought about by environmental effects independently of any genetic change in the population. This section is not intended as an exhaustive review of the literature but rather as a survey of examples that illustrates the range of neophenogenetic changes, including both developmental responses to new functional demands and the direct effects of a changed environment, that can be explained without invoking genetic mechanisms such as natural selection. We have deliberately excluded any studies in which the phenotypic change might involve change in the genotype, because our aim is to show that much important phenotypic change can be explained without an appeal to accompanying genetic change.

Of greatest significance to our argument are those kinds of developmental response that give rise to persistent novel phenotypes. As argued above, such responses both provide phenotypic variation for natural selection and in themselves constitute a mechanism of neophenogenetic change. For this reason, we will exclude from detailed consideration those developmental responses that appear to be specific adaptations to recurrent environmental circumstances, such as seasonal or ecotypic variation. For example, buttercup seeds (Ranunculus spp.) can produce either of two quite different kinds of plant, depending on whether they germinate under water or on land; the two forms show differences in leaf and stem morphology that reflect the different adaptive demands of these two habitats (Cook & Johnson, 1968). Such adaptive polyphenisms have been described in a wide range of species, especially among plants (Bradshaw, 1965; Hickman, 1975; Teeri, 1978) and insects (Hoffmann, 1978; Shapiro, 1976; Watt, 1968) but they will not be discussed here.

RESPONSE TO A NEW FUNCTIONAL DEMAND

Throughout this article we have emphasized the role of function in development, and new functional demands on the individual constitute one of the most important extra-genetic mechanisms of neophenogenesis. Functional interactions between the organism and its

environment during development were essential to Lamarck's theory of evolution and are equally important to the argument being developed here. The role of locomotor function in skeletal development, already discussed, is an example of this category of environmental influences. Fossil skeletal remains have been such an important source of information about vertebrate evolution that an appreciation of the role of function in skeletal development would seem essential to understanding the mechanisms of vertebrate evolution. Several authors have discussed the mechanisms of skeletal evolution from a developmental point of view (Fallon & Cameron, 1977; Hall, 1975; Lande, 1978; Robb, 1935; Straus, 1927) but functional considerations rarely play an important role in these discussions. Since we know that function is critical to normal skeletal development, this seems an unfortunate omission (cf. Cope, 1887, 1896).

To illustrate, consider an example provided by Bock & Morioka's (1971) analysis of the ectethmoid-mandibular articulation (EMA) in the *Meliphagidae*, a family of tropical passerine birds commonly known as honeyeaters. The EMA is a secondary jaw articulation possessed by some members of this family in addition to the normal articulation between the mandible and the quadrate bone of the skull. Bock & Morioka conclude, on the basis of a detailed analysis of the anatomy and feeding habits of the species concerned, that the function of the EMA is to brace the mandible so as to facilitate opening the bill in a manner that permits the tongue to be coated with mucus as it slides in and out of the mouth during feeding. The EMA shows various degrees of elaboration in the species that possess it. In some, such as *Melithreptus albogularis*, the pronounced dorsal mandibular process forms a fully developed diarthrosis with the ectethmoid bone. In others, such as *Manorina flavigula*, the dorsal process is much smaller and the EMA correspondingly less well developed. But there is a considerable amount of individual variation within species, as Bock & Morioka (1971) illustrate in their fig. 8 showing the mandibles of seven specimens of *M. flavigula*. One of these specimens is especially interesting because the shape of the dorsal process of the mandible (which forms the EMA in contact with the ectethmoid bone) is quite different on the right and left sides of the mandible. Bock & Morioka attribute this situation to mechanical interaction between the bones of the skull during development:

> "Presumably this bird had some malformation or malfunction of its jaw muscles or of its quadrate articulations so that the mandible was pulled to the right [This produced an] abnormal rubbing together of the dorsal mandibular process and the fugal bar [which] resulted in a modification of the dorsal process via the mechanisms of physiological adaptation possessed by bony tissue" (p. 21).

Thus, in this abnormal specimen there is evidence of a role for mechanical interactions between the bones of the skull in the development of the EMA. But such interactions must also be involved in normal development among the Meliphagidae; their precise nature will depend on the behavior of the young bird and the movements of its bill and skull that occur during early development. Bock & Morioka (1971: 46) attribute the evolution of the EMA to the natural selection of genetic variants, but its initial appearance in the population and its subsequent elaboration into the complex structure seen in some species may well have been the result of new functional demands brought about by a change in the behavior of individuals in some ancestral population(s). Although these alternatives cannot be resolved definitively, because the populations in question are now extinct, the latter hypothesis can be investigated experimentally. If it turns out that development of the EMA in one or more species of Meliphagidae depends on

the normal pattern of mechanical interactions that occur during individual development, as shown in other species by Drachman & Sokoloff (1966), Herring & Lakars (1981), and Lanyon & Bourn (1979), or if the development of the joint can be altered by changing those demands (cf. Gordon et aL, 1989), then the hypothesis will be supported. It might also be possible to produce an experimental replica of the EMA in some species that does not normally develop one, demonstrating how the initial appearance of this phenotype might have been caused. Stebbins & Basile (1986) have argued that demonstrations of this kind (which they call "phyletic phenocopies") may provide a valuable tool for investigating the developmental basis of evolutionary change (see also Rosen & Buth, 1980), and we would argue that they are likely to be even more important for the analysis of neophenogenetic change.

The sensitivity of tooth and jaw development to changes in diet has been shown in both vertebrate and invertebrate species. Bernays (1986) reared caterpillars of the noctuid moth Pseudaletia unipuncta on both soft and hard foliage and found significant differences in the morphology of the heads and mandibles between experimental groups. These differences were not allometric side-effects of changed body size (resulting, perhaps, from different nutrient content of the two diets), because overall body size did not differ between the groups. Rather, the skeletal differences were specifically due to the mechanical interactions between the jaws and the food. Changes in diet have also been shown to affect the jaw and skull of rats (Beecher & Corruccini, 1981; Bouvier & Hylander, 1982; Moore, 1973), the teeth of primates (Corruccini & Beecher, 1982), and various hard structures in cichlid fish (Greenwood, 1965; Meyer, 1987). Such findings suggest that changes in tooth and jaw anatomy revealed in the fossil record are partly due to the mechanical effects of dietary change, and not entirely to natural selection, as is usually supposed. For example, Brace et al. (1987) argue that the reduction in hominid tooth size during the Late Pleistocene was due to relaxed selection pressure for large teeth following the advent of cooking. An alternative explanation is that at least part of the reduction was due to the change in mechanical demands on human teeth as soft, cooked food became more common in the diet.

The central nervous system, particularly in birds and mammals, is especially sensitive to the developing organism's interactions with its environment (Greenough, 1975; Renner & Rosenzweig, 1987). Thus, we might expect changes in such interactions to have played an important role in the changes in CNS structure and function that are such a prominent feature of vertebrate evolution (Gottlieb et al., 1982; Johnston & Toth, 1989). Neural differences between species are usually attributed, whether implicitly or explicitly, to the effects of natural selection (e.g. Eisenberg & Wilson, 1978; Radinsky, 1978), but such differences can be produced within a species by changing the conditions under which animals develop (Renner & Rosenzweig, 1987). Theories of CNS evolution (e.g. Jerison, 1973) have not considered the effects of changes in experience on CNS structure and function, even when they explicitly take developmental considerations into account (Katz, 1983; Katz, et al., 1981; but see Katz & Lasek, 1978).

Changes in physical features of the environment such as temperature, humidity, and salinity, as well as in social features such as crowding must be a common occurrence during phylogeny. Their developmental effects on a variety of organisms are well documented and some authors have explicitly drawn attention to their taxonomic or phylogenetic implications. For example, Sumner (1909) reared white mice at two different temperatures and measured the effects on

several morphological dimensions. He found that the tails of mice reared in a cold environment and, to a lesser extent, their other extremities, were shorter than those of mice reared in warmer surroundings. Sumner pointed out that "The modifications thus artificially produced are such as have long been known to distinguish northern from southern races of mammals" (p. 146; emphasis in italics in the original). Similar results of cold rearing, apparently mediated in part by parental behavior, have been reported by Barnett & Dickson (1986). Changes in rearing temperature have also been shown to affect meristic characters (such as the number of vertebrae) in fish (Brooks, 1957; Hubbs, 1922, 1926; Murray & Beacham, 1989).

Retardation or acceleration of somatic development can have profound effects on the adult phenotype and such changes in developmental rate have often been cited as a potent source of phenotypic change (Bolk, 1926; de Beer, 1958; Gould, 1977). Several cases are known in which external conditions may alter developmental rates sufficiently to produce marked change in the phenotype. For example, the normal temperature range of the water beetle *Rhodnius prolixus* is about 21-32°C. If fourth instar larvae are reared at lower temperatures (17-20°C), they molt as usual into fifth instar larvae but have a more juvenile morphology than normal. Those reared at higher temperatures (33-36°C) show a rather more adult morphology than normal (Wigglesworth, 1952). A similar situation is reported by Lynn (1961) for *Ambystoma tigrinurn*: Individuals living in cold Rocky Mountain lakes are normally neotenic, reproducing as aquatic larvae, but those reared in warmer water in the laboratory metamorphose into terrestrial adults (see also Snyder, 1956). The potential phylogenetic and taxonomic significance of these results is confirmed by field data on both living (Southwood, 1961) and extinct (Tihen, 1955) species. These data show that temperature-induced changes in morphology are not merely laboratory curiosities but regularly occur under normal ecological conditions as well.

Other environmental conditions than temperature have been shown to have important phenotypic effects that are of potential significance for understanding the processes of neophenogenetic change. Bullfrog larvae (*Rana catesbeiana*) reared under hypoxic conditions show a variety of physioloical and morphological changes in all of their organs of respiration (skin, gills, and lungs; Burggren & Mwalukoma, 1983). Larvae of the salamander *Ambystoma tigrinum* nebulosum develop into either typical or cannibalistic morphs, but the cannibalistic morphs only develop under crowded rearing conditions (Collins & Cheek, 1983). Morphogenetic effects of environmental changes have been demonstrated in the field as well as the laboratory. James (1983) transplanted red-winged blackbird (*Agelaius phoeniceus*) eggs between sites in Florida, Colorado, and Minnesota and found that much of the regional difference in morphology among these populations could be attributed to environmental rather than genetic differences. As James points out, geographic variation among avian populations is generally attributed to natural selection, or to other genetic mechanisms; her results reveal the importance of taking extra-genetic mechanisms into account in explaining such differences.

If the existence of effects such as these is not appreciated, it may erroneously be assumed that natural selection is responsible for all phenotypic change that occurs during phylogeny (Michaux, 1988). For example, Kellogg (1975) described a gradual change in shell width in the fossil radiolarian Pseudocubus vema over 2 million years of its history recorded in a single deep-sea core. She interpreted this phenotypic change as a result of selection for larger body size in a cooling environment. Gould & Eldredge (1977) challenged Kellogg's interpretation, pointing out

that migration of larger individuals from neighboring populations could also account for the change in shell width in her sample. They also suggested a third alternative, one that closely follows our argument in this paper:

> "And if the increase is phyletic (affecting the entire species, though only sampled in one spot), why must we invoke genetic change mediated by natural selection—as Kellogg does (p. 368). For basic dimensions of simple creatures, a purely phenotypic response of an unaltered genotype to changing environments seems just as likely. We must not make up stories about the power of natural selection, just because modern theory favors it as an evolutionary agent. In so doing, we do not strengthen the Darwinian cause, but only display our biases [p. 128-129]".

We are in full agreement with Gould & Eldredge's caveat and with their implied suggestion that we should be prepared to entertain a wider variety of mechanisms to explain phenotypic change than natural selection alone. However, we would also extend the scope of their remarks, for the environment has developmental effects on more phenotypic characters than just "basic dimensions of simple creatures", as the studies discussed here show.

CONCLUSION

The theory of neophenogenesis presented in this paper can be considered an organismic theory of phenotypic change. It differs in those respects from conventional neo-Darwinian theory, which has a populational rather than an organismic focus, and emphasizes genetic rather than phenotypic change. The theory has much in common with other accounts of phenotypic change that adopt an organismic perspective, including those of Ho & Saunders (1979, 1982; Saunders & Ho, 1976), Jamieson (1986), Lambert & Hughes (1984), Lewontin (1983), Michaux (1988), Novak (1982a), Reid (1985), Rosen & Buth (1980), Wake et al (1983), and Zemek & Socha (1982; Zemek et aL, 1985). The unifying feature of all of these proposals is their focus on the organism as the source of evolutionary innovation. Change in the population is seen as the outcome of a process that begins with organismic change, rather than as a mechanism for explaining organismic change, as is the case with neo-Darwinism. An alternative way of putting the distinction is that neoDarwinism takes the origin of novel (phenotypic) variations for granted, and concentrates on explaining their selective (and other) consequences; organismic theories treat the origin of the variations themselves as the primary problem to be solved.

Because of their focus on the individual organism, organismic theories of phenotypic change put developmental considerations at the center of the explanatory stage. Rosen & Buth (1980: 300) argue that "evolution, in the sense of the transmutation of species, is an epigenetic, not a genetic problem". In other writings, we have also emphasized the primacy of developmental mechanisms for understanding evolutionary change (Gottlieb, 1987, in press; Gottlieb et al., 1982; Johnston, 1984) and in this paper we have attempted to present a more thorough account of the consequences of that stance. In particular, we have emphasized that modern developmental theory requires us to abandon the distinction between inherited and acquired characters in explaining phenOtypic change. That fact requires us to adopt a much broader view of the mechanisms responsible for phenotypic change and we have adopted the term "neophenogenesis" to encompass both genetic and extra-genetic mechanisms of change. That does not mean that we propose the existence of two independent mechanisms of change, one involving natural selection of heritable variants, the other some mode of "cultural" or "non-genetic" change (e.g. CavalliSforza & Feldman, 1981); nor do we endorse the developmentally

naive theory of "gene-culture coevolution" proposed by Lumsden & Wilson (1981; see critique by Johnston, 1982). In our theory, the organism is treated as an integrated developmental system, and explanations of change may draw on any of the factors (both genetic and extra-genetic) that contribute to its development (Fig. 2).

Because neophenogenetic theory recognizes that phenotypic change may be produced without any alteration to the average genotype of the population, it does not depend on mechanisms for overcoming Weismann's barrier as do some other organismic theories of phenotypic change (e.g. Ho et al., 1982; Michaux, 1988; Rosen & Buth, 1980). We do not deny that such mechanisms may exist, but their existence is not necessary for our theory to carry the explanatory burden assigned to it. Buss (1983a, b) has argued that Weismann's barrier may be irrelevant to evolutionary change in many taxa whose genetics are quite different from those of vertebrates. We argue that even where Weismann's barrier exists, it does not constrain the phenotypic effects of environmental change as neo-Darwinism has assumed. A similar point is made by Socha & Zemek (1978: 85), who point out that if an environmental factor "act[s] permanently for a number of generations, the resulting [phenotypic] change can be of a phylogenetic character". We agree with Socha & Zemek (1978, 1982) and with Novak (1982a, b) that such changes are of fundamental importance for understanding the ways in which phenotypes have changed over evolutionary time. The theory of neophenogenesis developed here is offered as a contribution towards that understanding. -

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