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The Role of Development in the Evolution of Life Histories

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Abstract. The aspects of development that interest students of life history evolution are plasticity, canalization, and constraint. Plasticity has both an adaptive component modifiable by gene substitutions and a chemically and structurally inevitable component. Plasticity may be either continuous or discrete. Continuous plasticity in life history traits is shaped by major trade-offs among growth, reproduction, survival, and differentiation. Allocations among these competing needs can be understood as problems in optimization within constraints set by the developmental system. More is known about what evolution ought to produce, in this respect, than about what developmental systems can produce. Discrete plasticity - the environmentally induced production of alternative forms, such as environmental sex determination - seems to be selected when there are discontinuities in the environment or where intermediate shapes will not work, and when environments vary mostly between, rather than within, generations. In different ways both plasticity and canalization uncouple the gene pool from selection, implying that the organism is a privileged level of selection and promoting stasis within species. We know little about developmental constraints and their implications for evolutionary ecology. Organisms appear to be a mosaic of relatively recent adaptations which we can understand in terms of optimality theory, embedded in a framework of relatively old constraints which we would like to understand in terms of developmental mechanisms.

INTRODUCTION

Two major approaches to biological understanding are the adaptationist and the mechanist. The first attempts to answer the

question: What should natural selection favor? The second attempts to answer the question: How does the organism work? Until recently life history theory has been an exclusively adaptationist field. This paper attempts to demonstrate that the mechanist approach is necessary to fill in large gaps in our understanding.

The emergence of life history theory as an independent field has drawn attention to the mechanisms of growth and differentiation from a new perspective and with new force. A brief historical sketch will make clear why.

Population genetics was the first attempt at a predictive quantitative theory of evolution. Its founders made a series of simplifying assumptions that had the effect of reducing the objects of study to changes in gene frequencies: the organism disappeared from view, and with it went the phenotype, the ecological interactions of the phenotype with the environment that determine fitness, and the developmental interactions with the environment that produce the phenotype.

Life history theory emerged in the 1960s out of the dual traditions of comparative demography and population regulation. One can view it as a reaction to the lack of empirical content forced upon population genetics by its simplifying assumptions. Along with other optimization approaches in evolutionary ecology, it constitutes the second attempt at a predictive quantitative theory of evolution.

Whereas population genetics underrates the organism, life history theory underrates the gene. The simplifications of one field are the complexities of the other. In life history theory, one studies how phenotypic traits interact to affect some measure of fitness. All fitness measures are somehow related to reproduction and to survival. Thus the life history traits most frequently analyzed include: age at maturity, number of offspring, age-distribution of reproductive

effort, longevity, and age-specific mortality rates. The major conceptual tools in the analysis are the equations of demography and the trade-offs among reproduction, growth, and survival.

Some limits of both population genetics and life history theory are in sight, if not reached (39). These fields are limited, in part, because they ignore each other and, in part, because they ignore development. Thus the perspective of life history theory motivates a new look at development because developmental mechanisms could connect population genetics with life history theory to form a predictive theory of evolution more powerful than either of the first two attempted.

To the developmental biologist or geneticist, what a life history theoretician means by "development" may not fit easily into the classical categories of the subject. By that term I mean not only traditional whole-organism patterns of growth and differentiation, but also the mechanisms that produce age-specific patterns of survival and reproduction. In the whole organism, these mechanisms produce dynamic patterns recognized as plasticity, canalization, and constraint.

Phenotypic plasticity denotes the capacity of a single genotype to produce a range of environment-dependent phenotypes: the scope of reaction for that genotype. Canalization is a near antonym of plasticity that describes the observation that many different genotypes may produce the same phenotype. It refers to the buffering of the phenotype from mutations, recombination, and the environment.

Constraints are features of ontogenetic mechanisms and morphogenetic design which limit the power of selection to mold phenotypic traits. They "should not be confused with trade-offs or costs, ... which share the characteristic that if the opposing selection forces were removed, the phenotype would be free to move beyond the point already attained" (37). The

most common constraints encountered in life history work are allometric and are defined within lineages. For example, weight at birth is strongly constrained by weight of parent in mammals, but the functions relating the two traits are different in primates and ungulates. The implication is that if selection acts to increase weight at birth, it must do so by increasing size of adult, and that if selection acts to increase size of adult, then size at birth will increase automatically, whether adaptive or not.

PHENOTYPIC PLASTICITY

Phenotypic plasticity has long been recognized as a major adaptation. Wright (51) stated that "it is not only of the greatest significance as a factor of evolution in damping the effects of selection ... but is itself perhaps the chief object of selection. The evolution of complex organisms rests on the attainment of gene combinations which determine a varied repertoire of adaptive cell responses in relation to external conditions" (p. 147). W.D. Hamilton has noted (personal communication) that under most realistic conditions an appropriately plastic type should beat any genetically fixed type in intraspecific competition. Plant ecologists take the view that "phenotypic plasticity represents a genetic response and may arise through local differentiation under conditions of disruptive selection" (48); they expect plasticity in colonizers and early successional species (7,16). In plants, the plasticity of a trait seems to be specific to that trait, caused by particular environmental factors, is specific in direction, is under genetic control, and is capable of being altered by selection (7). While this view is largely correct for the plants studied, evidence to corroborate it does not exist for most animals. Certainly the exclusive emphasis on plasticity as adaptation needs qualification.

The Nature of Plasticity

The term "phenotypic plasticity" covers a variety of complex phenomena. Plant ecologists emphasize that plasticity is

adaptive, is genetically based, and is appropriate to the stresses encountered. They have evidence to back this view. For example, in common groundsel individuals differ heritably in plasticity itself (1), and phenotypic variability may be plastic in one population and genetic in another (2). Such evidence is sparse in the literature. Although not sufficient to force the conclusion that plasticity is adaptive, it does make that view more plausible.

Thus part of what we see as a plastic response is arguably an adaptation. However, some plastic responses also inevitably result from the physicochemical nature of organisms. Although much of the information required to construct an organism unquestionably resides in DNA, a significant portion of it resides not in the genes but in the dynamic properties of the chemicals used to make the organisms. Consider the remarkably appropriate pattern of reinforcing fibers seen in the cross-section of the head of a mammalian femur: they reflect precisely the lines of stress induced by patterns of loading placed on the bone during function (42,46). Nothing about the arrangement of the fibers is encoded in the genes; it is a convenient property of the whole bone system, including collagen fibers, apatite, and bone cells, that it forms reinforcing fibers along lines of stress during remodeling. This is at least a property of mammalian bone; it may also be the case in other vertebrates, but this point is not as well established.

Second, consider Odell, Oster, Alberch, and Burnside's (26) model of gastrulation. They showed that, given a hollow ball of cells with an arrangement of fibers resembling the microfibrils of real blastulae, gastrulation could occur spontaneously without a signal from the genes. The context is in part created by the genes; once it is set up, the event follows inevitably. If their model is correct, it implies that the genes control gastrulation indirectly, by specifying the nature and distribution of microfibrils. The key point is that in both cases changes in bone reinforcement or gastrulation could be brought about only within limits imposed by the mechanism used. Genes must use such

mechanisms to build organisms, and each mechanism implies a specific set of opportunities and a specific set of constraints.

These cases point to a general property with special relevance to phenotypic plasticity. Not all plasticity need have originated as an adaptation. Adaptive plasticity presupposes the existence of a developmental system with heritable variability for a norm of reaction which selection could modify. Plasticity originated straightforwardly in chemistry. Life began as a complex chemical system, and the rates and directions of chemical reactions are sensitive to temperature, pressure, pH, and substrate concentration. The far-reaching consequence of those simple facts is that any organism which is not buffered against those changes in the physical environment to which chemical reactions are sensitive will inevitably be phenotypically plastic. There is no genetic control or "adaptation" involved in this component of plasticity, which is as much an encumbrance as an opportunity.

Adaptive plasticity resulted from the genetic modification of the primordial plasticity imposed by chemistry. Wherever we see adaptation in plasticity, it is layered upon and limited by the nature of the materials out of which organisms are built. Thus one major research question in the evolutionary ecology of development is this: How much of any particular plastic response in a life history trait has evolved, and how much is inevitable?

The Types of Plasticity

Bradshaw (7) recognized three types of plasticity: continuous, graded responses to continuous environmental change (Schmalhausen's "dependent morphogenesis"), discrete modifications with no intermediates (Schmalhausen's "autoregulatory dependent morphogenesis"), and fixed phenotypic variation, as is found in the germination requirements of desert plants and the hatching requirements of annual fishes (cited in (36)). Most plasticity

in life history traits represents continuous responses to continuous changes in environmental variables like salinity, temperature, and food: plasticity in growth, condition, reproduction, and longevity. Discrete plastic responses, called "developmental switches" by Levins (23), seem to be less general, but are known in rotifers, aphids, and cladocerans, may exist in fish, and function in the production of castes in ants.

Continuous plasticity. We already know a great deal about continuous plasticity from work done in environmental and comparative physiology. However, the implications of that work have not been made general through connections to development and evolution. So much mechanism and structure underlie "plasticity" that one wonders if the word, with its connotation of freedom in scope of response, misleads. At least three trade-offs shape plasticity in life history traits. Their causes, relationships, and impacts all need exploration. The implicit assumption in all discussions of trade-offs is that the economic principle of allocation of scarce resources to competing needs applies to the case at hand.

1) Growth versus Reproduction: This is the first of two trade-offs that define the Cost of Reproduction, a central concept in life-history theory. Its importance was emphasized by Williams (50), who recognized its equivalence to trading present reproduction for future reproduction via growth. The effect can perhaps be most clearly seen in organisms with storage organs, such as plant tubers or salamander tails. For example, Maiorana (24) showed that salamanders with bits of tail missing delayed maturity, if juveniles, and either skipped reproduction for an entire season or reduced reproductive efforts, if adults. Salamanders are long-lived. The pattern Maiorana found is consistent with the idea that for a long-lived organism with juvenile mortality much higher than adult mortality, adults are "worth" more than juveniles, and that excess energy, up to a point, should be shunted to maintenance or growth rather than reproduction.

Hirshfield (17) has also nicely demonstrated the trade-off of growth and reproduction in medaka, a small cyprinodont fish from Japan. In medaka, increased temperature with no change in food intake results in increased reproduction and decreased growth. Calories invested in reproduction are balanced almost exactly by calories taken away from growth. This diversion of an increased proportion of fixed resources into reproduction from growth represents a reaction on two levels. Maintenance may be less efficient at higher temperatures, increasing the probability that the adult will die for physiological reasons. Also, increased temperatures may indicate increased mortality risks to the adult from external changes in habitat or community composition. These data suggest that the allocation of energy depends both on the impact of environmental changes on the condition of the organism and on the information that current condition carries about future changes in adult and juvenile mortality.

Spight and Emlen (35) studied the plastic responses of snails to an increase in food supply, where energy could go either into growth or reproduction. The allocation of energy to function seems to be well understood within the adaptationist context of life history theory. A snail species which had high adult mortality put increased food into increased reproduction, whereas a second species with low adult mortality put increased food into growth (future reproduction).

Whether organisms have determinate or indeterminate growth also depends on the trade-off of growth and reproduction. Many annual plants, nearly all birds and mammals, and most insects first grow, then reproduce. Most perennial plants, fish, amphibians, and reptiles continue to growth throughout life. Male mosquitofish stop growing at maturity; females of the same species do not. Theorists have tried to explain determinate growth by using optimal control theory to predict age-specific allocations to growth and reproduction in annual plants (45) and bees (27). When fitness increases with size, but at a decreasing rate, Oster and Wilson (27) predict a shift from pure growth to pure

reproduction at a fixed size. When fitness increases with size, but at an increasing rate, a graded response is selected. They also noted that stochastic variation in system parameters always results in a graded response.

To summarize, theory suggests that the shift from growth to reproduction depends on the demographic environment and the relationship of fitness to size; the data reveal a rich diversity of patterns of growth versus reproduction which are only beginning to be explored. Theory emphasizes the importance of measuring the dependence of fitness on size in pea aphids, which have determinate growth (28) and conveniently short generations, or in poeciliid fish, where one sex has determinate growth and the other has indeterminate growth. We should also entertain alternative hypotheses: organisms may stop growing because larger size carries increased mortality risks from size-selective predation.

2) Reproduction versus Survival: This is the frequently modeled cost of reproduction to the adult in the form of increased mortality (4). More precisely, increased reproduction reduces the likelihood of surviving to reproduce again in the future. In most models, the shape of the relationship between reproductive effort and residual reproductive value is critical: predictions change qualitatively when that curve changes from concave to convex, for example (19). Only Snell and King (33) and Law (21) have measured the shape of the relationship in rotifers and grass, respectively, but extensive evidence (9, 36) indicates that many angiosperms, arthropods, and chordates experience a cost of reproduction.

Calow (9) suggests that the mortality cost of reproduction can be resolved into ecological and physiological components. Moving to a different level of biological organization, I would add Williams' (49) genetic hypothesis: genes with beneficial effects on early fitness but pleiotropic deleterious effects on

late fitness components will be favored by natural selection. Experiments on Drosophila (31) and Tribolium (25,34) support the pleiotropy hypothesis. Note that the two hypotheses are not competitors, and that one would expect to see physiological competition between reproduction and maintenance caused by pleiotropic effects of the sort described.

Although the evidence is scanty, demographic pressures appear to have molded genetic mechanisms that have physiological effects on the ontogeny of reproduction and survival. The effect most directly related to phenotypic plasticity is reproductive compensation, demonstrated in experiments with Tribolium (6,25). If one stresses flour beetles by depriving them of food or by increasing their density on a fixed food resource, they decrease their reproductive effort and live longer. Beetles stressed early in life have higher post-stress reproductive rates later in life than do similarly aged unstressed beetles. The effect is not general; stressed flatworms increase their reproductive rate, presumably because juveniles are more resistant to starvation than are adults (9). As with the growth versus reproduction trade-off, the reproduction versus survival trade-off has an adaptationist explanation couched in terms of the differing impacts of environmental change on adults and juveniles. If stressed adults survive better than stressed juveniles, they should decrease reproduction; if not, they should increase it (18). Such effects, if demonstrated generally, will imply that natural selection has shaped developmental mechanisms capable of responding adaptively to specific stresses in specific demographic environments.

3) Growth versus Differentiation: This trade-off gives us the clearest view of the contrast between adaptationist and mechanist interpretations of the same phenomena. The questions at risk are: How fast should an organism grow? How rapidly should it differentiate? How many different morphological stages should it have in its life history, how long should it

spend in each, and how large should it be at the transition between each stage? One comparison that prompts the questions contrasts altricial birds and altricial mammals, which require parental care, with precocial birds and precocial mammals, which can feed themselves from the day of hatching or birth. Why are most carnivores altricial and most herbivores precocial (H. Horn, personal communication)? Similar patterns dominate comparisons among amphibians, insects, and marine invertebrates: some produce free-living larvae which either subsist on yolk or feed themselves, others are brooded and undergo metamorphosis either in an egg retained by the parent or in association with the parent.

Lack, an adaptationist, explained growth rates in birds as an optimal balance between rapid growth to reduce the juvenile period of vulnerability, and slow growth to reduce the energy requirements of the young (20). Ricklefs (29) countered with a mechanist explanation: all birds grow as fast as they can, but growth rates are set at different levels by the different constraints that operate in each group. He saw the major constraint as a conflict between growth and differentiation at the level of the tissues themselves. Only careful quantitative work will disentangle the relative contribution of these alternatives to a balanced explanation.

Case (10) comprehensively reviewed growth rates in vertebrates. He saw constraint dominating comparisons among higher taxa and adaptation dominating comparisons among closely related forms. The basic constraint on growth rates in vertebrates is ectothermy: the maximum growth rate of animals that maintain a consistently high internal temperature (endotherms) is an order of magnitude higher than the maximum growth rate of any animal that does not maintain a high internal temperature (ectotherms). Altricial birds grow about twice as fast as any mammal; precocial birds and eutherians grow at about the same rate. Within mammals, the level of constraint appears to be at the level of the order or the family. Within orders or families, growth

rates appear to be adapted to the feeding requirements and mortality rates of juveniles and adults. Robbins and Robbins (30), in a detailed review of growth rates in ungulates, emphasize constraint, not adaptation: they saw very basic controlling mechanisms common to all ungulates and subungulates that produce similarities in fetal and neonatal growth.

The literature on marine invertebrates reveals a similar contrast of adaptationist and mechanist views. Vance (44) and Underwood (43) both emphasized stage-specific food availability and mortality rates in explaining yolk-fed versus self-fed larvae. They differed strongly with each other on the details. Out of Strathmann's (41) synthesis again emerges a picture of constraints operating among higher taxa, and adaptation dominating comparisons among closely related forms.

The same contrast is seen in the literature on amphibian metamorphosis. Wilbur and Collins (47) emphasized the role of food availability, competition, and size-selective predation in determining length of larval period and size at metamorphosis. However, Smith-Gill and Berven (32) saw a basic constraint operating in the temperature-dependence of the differentiation rates involved. They claimed that growth rate depends on stage of differentiation attained, but that differentiation does not depend on growth rate or size attained. Because differentiation is more sensitive to temperature than is growth, differentiation is more frequently rate-limiting. Because metamorphic stages, not being particularly well adapted to either the juvenile or the adult habitat, are especially vulnerable, organisms should pass through them as rapidly as possible. Arnold and Wassersug (3) have demonstrated, for example, that garter snakes congregate in great numbers to feed on metamorphosing toads. Organisms should pass as rapidly as possible through those ontogenetic stages in which they suffer high risk. If differentiation is rate-limiting in such a stage, then it constitutes a real developmental constraint.

In summary, people working on higher vertebrates, on marine invertebrates, and on amphibians have produced literatures with similar patterns. The adaptationist interpretation of growth rates and size attained at transitions between stages is this: organisms should grow rapidly through sizes or stages of high risk and should attain the stage of differentiation that permits independence from parents when the benefits of doing so exceed the risks incurred. The benefits are determined primarily by the food available and the growth that results; the risks are determined primarily by physiological competence and by predation. For example, an organism may suffer high mortality in some larval stage because it has a morphology and physiology that promote rapid growth. The morphological specialization is for feeding rather than defense; the additional mortality is caused by predation. This suggests that organisms can specialize for feeding or defense but not for both (H. Horn, personal communication). The mechanist interpretation of the same phenomena emphasizes the incompatibility of growth and differentiation at the level of the tissues, and the dependence of growth on differentiation. These trade-offs need experimental analysis. Clearly, some types of growth are also aspects of differentiation - for example, the expansion of a tissue layer. Just as clearly, some types of differentiation depend on growth (19).

Evidence of developmental constraints in comparisons of higher taxa is not surprising; in many cases, higher taxa were recognized as different because they are differently constrained. Organisms are a mosaic of relatively new adaptations embedded in a framework of relatively old constraints. Our view of the general selective forces operating on growth and differentiation is increasingly clear, but our understanding of the mechanisms underlying constraints needs attention. Studies such as Lawlor's (22) on pillbug moulting and reproduction exemplify the productive combination of mechanist and adaptationist approaches that should succeed elsewhere.

While no one has published a model that considers the evolution of plasticity from the point of view of these trade-offs,

we do have some data. Berven, Gill, and Smith-Gill (5) analyzed the apparently plastic response of the green frog which grows more slowly and metamorphoses at a larger size at higher elevations and lower temperatures. One would expect that of an ectotherm on mechanical grounds alone, but they showed that frogs from high elevation populations actually grew faster and matured at smaller sizes than one would predict from the growth of low elevation animals. Their result indicates that in this case the plastic response was not fully adaptive and had been countered by selection.

Discrete plasticity. Levins (23) commented that developmental switches are "the optimum strategy for a fine-grained environment when the range of the environment exceeds the tolerance of the individual phenotype." There is more to the adaptationist interpretation than that, as will be seen after we consider several examples. We also have partial descriptions of the mechanisms underlying some switches.

1) Heterogont life cycles of aphids, rotifers, and cladocerans: These organisms are facultatively asexual and normally reproduce parthenogenetically. When they receive a cue that indicates either predictable upcoming stress or a marked rise in the unpredictability of the environment, they shift from producing asexual to sexual eggs. In rotifers and cladocerans, the sexually produced eggs differ morphologically, can diapause, and are resistant to heat, cold, and drying. In aphids, the sexually produced offspring are winged; asexually produced offspring lack wings. There is a considerable literature on the environmental cues that induce the switch.

2) Environmental sex determination: Charnov and Bull (12) suggest that sex should be environmentally determined whenever the offspring enters a patchy environment away from the parent, in which some patches strongly favor males over females (and vice versa), and when neither offspring nor parent has control over which patch type the offspring enters. Examples

include parasitic wasps, nematodes, reptiles, fishes, echiurids, isopods, copepods, and orchids. Sequential and simultaneous hermaphrodites are also known for labile sex changes that usually depend on size and the relative fitness of each sex. The expanding adaptationist literature on environmental sex determination is largely consistent with optimization and lack of constraint. Note, however, that reptiles with sex chromosomes do not show environmental sex determination, whereas those that do not have sex chromosomes do show environmental sex determination (8). Sex chromosomes seem to have evolved recently, since they appear independently in several lineages. Thus in reptiles, environmental sex determination appears to be the ancestral condition, and the evolution of sex chromosomes places a genetic constraint on prior plasticity.

3) Nongenetic female polymorphism in the rotifer Asplanchna: Transitions between the two or three forms are triggered by the level of vitamin E (tocopherol) in the environment; it seems to serve as a cue to the existence of larger prey, and triggers production of offspring that grow to a larger size, have a different shape, and are capable of eating such prey. In this case, Gilbert's (14) elegant experiments have given us some insight into the mechanism of, as well as the reasons for, the switch.

4) Size of offspring in mosquitofish: Mosquitofish from estuaries in Texas have a developmental sensitivity to their early environment. If they encounter fresh water in the first week of life, they give birth, about 160 days later, to offspring that are 16% larger than the offspring produced by fish that encounter brackish water during the first week of life (38). In the field, the difference in size of offspring is also 16%, and in the same direction. Salinity may indicate the type of community encountered later in life, and larger offspring may be favored in the simpler freshwater community. The underlying mechanism is unknown, but given the close association

of excretory and reproductive tissue during development, it is likely that if there is a switch it is set early in life via the developing kidneys or gonads.

Why should an organism evolve a developmental switch, rather than a graded response? The theory is incomplete but developing rapidly. Discontinuities in development appear to be associated with several things: (a) discontinuities in the environment, such as a switch from fresh to brackish water; (b) discontinuities in morphology, such as winged versus wingless aphids, or males versus females, where intermediate forms would not function; or (c) environments that vary mostly between, rather than within, generations (36). The ontogenetic mechanisms that control switches are not well-understood, and stand as a challenge to researchers.

Uncouplings

Developmental plasticity uncouples the phenotype from the genotype and thus frees the gene pool from the immediate impact of selection, which acts on phenotypes. The adaptive advantage of this uncoupling to an individual genotype is immediately clear: any suboptimal genotype with the plastic capacity to produce an improved phenotype will be favored over other suboptimal types without that capacity (51). However, an evolutionary commitment to plasticity carries with it the cost of putting the phenotype at the mercy of the environment. Canalization also uncouples the gene pool from the effects of selection by ensuring that many different genotypes produce a single phenotype (which may be optimal). Canalization also makes the phenotype independent of the environment. Thus canalization and developmental plasticity differ significantly in the manner in which they uncouple phenotype and genotype.

Under what conditions should we expect plasticity to be favored over canalization? Collins (13) claimed that the traits to which fitness is most sensitive should be canalized and remain unaltered by stress, but Boyce (cited in (13)) pointed out that if the optimum value of a trait shifts predictably

under stress, then the components of fitness should be plastic in a specific way. More generally, if the environment is unpredictable from generation to generation, but if conditions later in life are predictable from conditions early in life or over an even shorter term, then plasticity is favored. If the environment is equally and highly unpredictable in the short- and long-term, within and between generations, then mechanisms to produce a variety of offspring, no matter what the immediate environment, appear to be favored, as is found in desert seeds and annual fish.

CONCLUDING REMARKS

The remarks that follow address several potential - and possibly controversial - implications of the point of view developed in this paper.

1) The major recurring theme of this paper is that developmental and demographic traits are co-adapted and co-constrained Just as development has a strong role in the evolution of life histories, life histories have had a strong role in the evolution of development. For example, one key theme in the life history literature is the explication of the forces operating to speed or to slow maturation, and we now have at hand a list of well-understood hypotheses on why any organism matures at the age observed (4,11,40). Age at maturity is a key trait in several senses. At maturity, organisms are committed to adult function through extensive morphological and physiological changes, often accompanied by habitat shifts. This complex network of change, extending over several levels of biological organization, must be coordinated and controlled by the developmental system. Thus the demographic environment may dictate that a certain age at maturity is optimal, but whether it is actually attained or not will depend on the flexibility of the developmental system.

2) The developmental plasticity and canalization of life history traits, the components of fitness, have at least two

implications for currently controversial topics in evolutionary biology. First, they imply that the organism must be an important, even privileged, unit of selection, in contrast to the more extreme positions taken in the discussion of "selfish genes" in sociobiology, which imply that the interests of the genes frequently overwhelm the interests of the organism. Most of the action in evolution has taken place in the design of the individual organism and its complex genetic, developmental, and life-historical architecture. Kin-selection has important effects on behavior, but has not touched the central problem of evolutionary biology: to provide a general explanation for the design of organisms.

Second, to the extent that plasticity and canalization are effective in uncoupling the gene pool from the selective impact of the environment, they promote genetic stasis within species and may very well be as important in this respect as gene flow. The relevance of these effects has not been adequately reflected in the debate over macroevolution (15). If in fact the major features of evolution are produced through a series of punctuated equilibria, with rapid evolution during speciation events followed by little change during the lifetime of species, then plasticity and canalization may be just those critical features that slow the rate of genetic change in the phyletic phase, and breakdown in plasticity and canalization may be necessary to spur a speciation event.

3) I have argued implicitly throughout this paper that developmental evolutionary ecology has sufficient content to be considered a viable specialty. What research strategies will increase that content most efficiently? No single approach will suffice, given the complexity of the issues, the multiple levels of organization involved, and the different points of view of the adaptationists and mechanists. We will see contributions from theoretical explorations of the trade-offs underlying plasticity, from experimental analyses of the mechanisms that produce those trade-offs, and from ecological

studies of the tactics of growth and differentiation. The single study I would most like to see done next would combine field studies of growth, reproduction, and mortality with laboratory studies of plasticity and the physiological and molecular mechanisms that produce it. Such a study should be done in two populations of a single species known to differ heritably in plasticity, to make possible subsequent genetic analysis.

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