

**Novelty and Niche Construction**

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**for a symposium: The Making Up of Organisms: Mapping the Future of  
Biological Models and Theories, Paris June 8-10.**

**Draft of March, 2006**

## **1. The Targets of Macroevolutionary Theory**

Macroevolutionary theory is organised around two distinct though related issues: phylogenetic diversity and phenotypic disparity. The study of diversity is the study of the phylogenetic organization of the tree of life at different scales and levels of abstraction. Sometimes the focus is on particular events: the apparently co-ordinated radiation of the insects and flowering plants; the explosive radiation of the Cambrian fauna. Sometimes it is on repeated patterns; for example, the role of mass extinction events in reshaping the history of life. The second central issue is the evolution of novelty, especially complex novelties. In the last decade or so, (Maynard Smith and Szathmary 1995) focused attention on the evolution of vertical complexity, though gene selection theory has played an important role in first making this problem visible. Once genes (or gene-lineages) are seen as independently evolving lineages with their own fitness interests, the problem of explaining the evolution of the multi-celled organism is hard to miss. Even for those genes which are in germ-line cells, helping build an organism is a very indirect and expensive way of adding new links to a lineage. It both delays replication and, often, commits to a meiosis lottery in which each co-operating gene only has a 50% chance of success. Moreover, even if the fitness benefits of life through an organism are worth this investment, there is a trajectory problem: how is the alliance initiated, stabilised and policed.

Leo Buss took up this challenge (see (Buss 1987)), but Maynard Smith and Szathmary generalised it. They showed that the evolution of multi-celled individuality was one of a series of evolutionary transitions involving the fusion of previously independent evolutionary agents; the result is a new, more vertically complex agent. Horizontal complexity has been recognised as puzzling for much longer (Nitecki 1990): this is the problem of reconciling an incremental conception of evolutionary change with the evolution of qualitatively new structures. While it is easy to see how an existing trait could be elaborated incrementally, it is much harder to see there could be (say) an incremental, and incrementally adaptive, shift from a scaled bipedal dinosaur to a feathered and flying bird.

As with evolutionary biology in general, these macroevolutionary questions have been pursued from a broadly externalist perspective. There are exceptions: a coevolutionary conception of the diversification of flowering plants and insects is received wisdom. But for the most part, evolutionary change is typically seen as a response to independent environmental forces. (Eldredge 2003) is a typical recent example: evolutionary change is essentially driven by the intensity and geographical extent of physical disturbance. These models underplay the active role of organisms and populations in constructing their environment. Richard Lewontin was the first to emphasise the importance of organisms' agency in selecting and modifying their environment; recently, niche construction theorists have developed and extended Lewontin's insights ((Lewontin 1982; Lewontin 1985; Odling-Smee, Laland et al. 1996; Jones, Lawton et al. 1997; Odling-Smee, Laland et al. 2003)). I shall argue in this paper that it is impossible to understand macroevolutionary dynamics without taking niche construction into account. I shall focus on the role of niche construction in the evolution of novelty, and I begin with a short summary of the argument.

The argument begins by rehearsing an "evo-devo" argument: the mechanisms that generate variation cannot be black-boxed, for unless those mechanisms are isotropic, generating variation densely and evenly around a lineage's existing location in phenotype space, the mechanisms that generate variation will influence the trajectory of evolution (Raff 1996; Arthur 2004). And while there are many open questions about variation, there is no a priori expectation that variation is typically isotropic. I then link this now familiar line of argument to an emerging emphasis on the role of phenotypic plasticity in evolution. In different ways, Mary-Jane West-Eberhard and Marc Kirschner and John Gerhart have argued that plasticity is central to evolvability (Gerhart and Kirschner 1997; West-Eberhard 2003; Kirschner and Gerhart 2005).

Their idea is this. Phenotypic plasticity pre-adapts lineages to evolutionary change, by decoupling the development of distinct organ systems. If organisms are phenotypically plastic, then the functional integration of different systems — for example, those that determine bone growth in limbs and those that lay down the circulatory system — must be sensitive to the contingencies of their development. Limb development requires simultaneous and co-ordinated development in other

organs and tissue systems: cartilage, muscle tissue and attachment points, innervation of soft tissues; circulatory connections to tissues and bone marrow. If bone structure or muscle mass is plastic, responding to signals from the environment, co-ordinated systems must be plastic too, responding to signals from the systems developing with them. For the organism does not “know in advance” how much muscle tissue to build, or how densely the capillaries should be packed into the tissues that surround the bone. Co-ordination must be managed via cross-talk, as these systems develop. They must be able to fine-tune their own development in response to signals from the systems to which they attach. This same sensitivity of integration to the contingencies of development will make functional integration possible in the face of genetically-caused changes in crucial organ systems. The systems that build muscle tissues and attach those tissues to the limbs will do so appropriately in the face of genetically modified changes in bone size or shape. Hence plasticity pre-adapts for evolvability.

In adaptable organisms, environmental change generates novel phenotypes: organisms respond behaviourally, physiologically and morphologically to new stresses and opportunities. Novelty appears by environmental induction. Such novelties are not transcribed into the germline and thus inherited. But they can still have evolutionary effects. Once those novel phenotypes appear, they are subject to a selective sieve. Some will disappear. But if the environmental inducer continues to be present, some of these novelties will reappear in the next and subsequent generations, rebuilt by the same mechanisms of phenotypic response. One consequence will be genetic accommodation in its various forms. The novel trait itself will be fine-tuned in various ways by genetic change. Likewise, other aspects of the phenotype will be adaptively adjusted to the novelty. So evolutionary change (on this view) often involves a double pulse: new phenotypes often appear first as a result of phenotypic response to environmental novelty; then followed by genetic accommodation.

We now get the link between niche construction and the evolution of novelty. Phenotypic plasticity is sometimes a response to extrinsic changes in the environment. But it can also be a response to changes the lineage itself has caused. Niche construction — the effects organisms have on their own world — is important for the ways it modifies selection pressures, and hence the ways existing variation is sifted

(Odling-Smee, Laland et al. 2003). But it is also important in generating the variation on which the evolution of novelty depends.

## **2. Biased Variation and Evolution.**

Rudy Raff's The Shape of Life epitomized a major reorientation in evolutionary biology: the integration of developmental biology within contemporary Darwinism (Raff 1996). Developmental biology is relevant to evolutionary biology because developmental biology is our theory of phenotype possibilities: of the potential variation that is available to selection. Developmental biology is in the business of identifying the  $G \Rightarrow P$  map, and its potential permutations. It took some time for evolutionary biology to accept that it was crucial to understand the  $G \Rightarrow P$  map, because a crucial assumption was made about its nature. The working assumption of the modern synthesis was that variation in natural populations was typically isotropic: the variation in a trait is distributed densely and without bias around its current mean. If this assumption were right, while the mechanisms through which a genome generates a phenotype would be interesting in their own right, evolutionary biologists could reasonably idealise away from these complications.

It is not likely, though, that this idealisation is appropriate: it is tied to Fisherian models which supposed that the development of a phenotype trait (and hence variation in that trait) depends on large number of small effect genes. But mutation is not restricted to point mutations: mutation which might substitute one amino acid for another. Mutation can result in movement, duplication, inversion and deletion of DNA sequences, and hence can result in changes to gene regulation and to shifts in reading frames as well as changes to the amino acids that are transcribed from the new DNA. Mutations which make a significant difference to continuously varying traits — “mesomutations” — are likely to be important in many evolutionary changes<sup>1</sup>. These may not be distributed normally around current phenotype values.

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<sup>1</sup> For a good discussion of these issues about mutation and their significance to our view of evolution, see Leroi, A. M. (2000). "The Scale Independence of Evolution." Evolution & Development 2(2): 67-77.

Such a bias in its supply is potentially relevant to evolutionary trajectories<sup>2</sup>. (Arthur 2004) drives this point home with exceptional clarity. As he notes, it is likely that the supply of variation is structured, and that structure matters. Consider a diagram plotting mammal species on a two-dimensional grid. The y-axis gives the length of front-limbs; the x axis the length of the back legs. Since most species have front and back legs of near-equal length, the result is a pattern where most mammals fall close to the diagonal from bottom left to top right ((Arthur 2004) p99). Why are mammal phenotypes clustered in this way? The supply of variation to selection is likely to be biased, for many developmental processes affect both sets of limbs. Selection might play some role: perhaps few life-ways reward elongated front limbs. Notice, though, that it is much harder to give a plausible selective explanation of why mammals mostly have the same number of fingers and toes. So while it is not impossible to alter front to back ratios — kangaroos have much longer back legs than front ones — that ratio does not change easily. Adaptive complexes like kangaroos are difficult to reach, and difficult to reach because of structure in the supply of variation. The natural supply of variation will not provide much variance in length between the two limb pairs, and hence adaptive peaks with unequal lengths will be too far away from current variation pools for them to be available. Likewise, while it is possible to lose toes from hind legs, it is difficult to adaptively optimise finger and toe numbers to their different roles, even when the functional demands on front and hind digits are quite different.

So bias in standing variation can influence evolutionary trajectory, by making apparently nearby optima inaccessible. Similar considerations apply to the supply of new variation to selection. Suppose a population finds itself on a flat fitness landscape

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<sup>2</sup> So too is the spread of variation. Imagine a rodent population on a flat fitness surface. Perhaps within a certain bound, mass makes no difference to fitness, and that the current mean mass of our rodents is 500 grams. In the vicinity, there are two higher local optima: 600 gram rats are fitter than 450 gram rats which in turn are fitter than those of 500 grams. The evolutionary trajectory of this population depends not just on bias in the generation of variation around the current mean, but also its spread. If the mechanisms of generation generate very little variation, the population will stay where it is. If it generates variants within 50 but not 100 grams of the current mean, the population will migrate to the lower local optima. If the spread is great enough for variants to intersect with the slopes of the higher peak, the population will find that peak.

near incompatible local optima. Such a population is “waiting for a mutation”. Different possible mutations will extend standing variation to the foothills of one of those local optima, after which selection-driven hill climbing will do the rest. The trajectory of the population will depend on which variation arrives first, and one might be more probable than another, even if the two potential variations are an equal distance in phenotype space from current phenotypes; phenotype distance may not correspond well with genotype distance. Moreover, some phenotypes are multiply realisable, as the phenomenon of phenocopies shows: different genomes can give rise to the same phenotypic outcome<sup>3</sup>. Other phenotypes have a single genetic profile; their development depends on a specific set of genes. Phenotypic variants that can be reached by a number of tracks through gene space are more likely to appear than those that depend on a specific track.

So the first crucial point in the argument of this paper is that if the supply of variation to selection is biased, that supply can play a pivotal role in determining evolutionary trajectories. It does so if variants at points in phenotype space that are equi-distant from current phenotypes are not equi-probable.

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<sup>3</sup> This phenomenon may be quite extensive. West-Eberhard mentions that upwards of 50% of experimentally induced gross chromosomal rearrangements in *Drosophila* have no phenotypic effect. While some of those no-effect results will be the result of changing junk DNA, many are inert because changes are buffered by canalising mechanisms (p19).

### **3. Phenotypic Plasticity and Its Significance**

We have just seen that the supply of variation has the potential to play a driving role in evolution. This section has two aims. The first is to explain the connection between plasticity and evolvability. The second is to show that the mechanisms of phenotypic plasticity play a crucial and biasing role in supplying that variation: there are indeed biases in that supply. In part, that is because environments play a crucial role in inducing variation, not just selecting it. This idea and its evolutionary consequences has been explored by Mary-Jane West-Eberhard and, in collaboration, Marc Kirschner with John Gerhart (Gerhart and Kirschner 1997; West-Eberhard 2003; Kirschner and Gerhart 2005). West-Eberhard, Kirschner and Gerhart all argue that phenotypic plasticity has profound consequences for evolution. Within-generation plasticity is a pre-adaptation to evolutionary plasticity. Lineages are evolutionarily plastic because organisms are phenotypically plastic.

It is no accident that organisms are plastic; plasticity reflects the complexity of the causal pathway from genetic material to expressed phenotype, and the many points at which contextual factors can affect these pathways, and hence plasticity is, as Schlichting notes, the primitive condition<sup>4</sup>. This primitive condition can be selectively modified, sometimes to reduce sensitivity to environmental factors, sometimes to channel it. For organisms (especially multi-celled organisms) can be advantaged by plasticity. (i) They develop, and in many lineages development involves profound morphological change: thus core organ systems must continue to function and co-ordinate with other systems through changes in those other systems. (ii) Though tolerances vary, all organisms must be able to recover from damage. Organisms must be organised in ways that buffer rather transmit the effects of local failure and damage. (iii) Individual organisms do not live in constant environments: some organisms are mobile, and even immobile organisms experience environmental

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<sup>4</sup> Thus Schlichting remarks that even nucleic acids and enzymes have reaction norms: they will function only within a restricted band of temperature and pH, a band which can itself be selectively modified, as the production of so-called "heat shock" proteins by Antarctic, cold-adapted fish at 5C strikingly show Schlichting, C. (2003). "Origins of Differentiation via Phenotypic Plasticity." *Evolution and Development* 5(1): 98-105. p 99

variability as their local circumstances change daily and seasonally. Yet different environments require different physiologies. (iv) Organisms live in unpredictable environments: plasticity extends the range of environmental stresses to which organisms can accommodate. (v) Organisms develop, and development does not yield phenotypic uniformity because development resources both within and without the embryo are not uniform. Developing embryos will be exposed to differing environmental fluxes, and be supplied with differing nutrient packages. Nor is the genetic environment is predictable: elephant seal heart-building genes need to be able to initiate the development of a functioning heart in both females and in the enormously larger males. Moreover, once a certain level of complexity is reached, development cannot be precisely controlled. Thus a given component needs to be able to work in somewhat different internal environments. Its systems of signalling, coordination and linkage must be able to cope with somewhat varying organizations of internal components.

Plasticity is mediated by a combination of modularity and “exploratory behaviour” (as Kirschner and Gerhart label it). Developmental modularity is crucial: unless the development of organ systems are decoupled from one another, a disruption anywhere will ramify through the system. Even so, organ systems must be appropriately connected to one another if the organism is to function. For example, the vascular system of mammals is extraordinarily complex: cells are never more than a few cell diameters away from a capillary. Yet the precise plumbing cannot be pre-specified: it must be sensitive to bone and muscle growth. So its development is patterned, but responsive to developmental signals from elsewhere in the organism. Thus capillaries are provided by a process of over-supply and selective attrition. Wherever muscle development is dense and hence the need for oxygen flow is great, less of the over-supply will be deleted.

West-Eberhard has some wonderful examples of phenotypic adjustment of this kind. Her flagship example is a two-legged goat: a goat that was born with essentially dysfunctional front legs and which acquired a kangaroo-like gait. The important point about this example was the accommodation of the rest of the phenotype to this massive reorganisation of locomotion: there was an extraordinary suite of muscle,

nerve and bone reorganisation to accommodate the different stresses, loads and movement ((West-Eberhard 2003) pp 52-53). This is a very extreme example, and no doubt the goat would never have lived had it not been born into a domestic environment. But West-Eberhard gives a series of other examples. In human populations there are many pathologically developed hearts, with arteries, veins and valves in non-standard places. While these may not be optimal, these developmental pathologies are not instantly fatal. The rest of the phenotype accommodates to them, connecting the system functionally to the circulatory and respiratory system. In development, bone and skin accommodate to the loads to which these systems are subjected (see eg (West-Eberhard 2003) p35), and the friction they endure. Bone is hard at a time, but in its growth and form it is extraordinarily plastic.

There are crucial cellular processes which manifest similar adaptive plasticity: for example, the mechanism in mitosis that ensures that each daughter cell receives the right chromosome complement (“spindle formation”) is adaptively plastic. It is not and cannot be pre-programmed with the location in the dividing mother cell of these chromosomes. So the microtubules that usher to them to the daughter cells explore from the centriole, and are stabilised if they connect with a chromosome. Thus mutations which increase chromosome number need not be fatal: the mechanisms of mitosis can accommodate to this change without needing a further genetic change. The phenotype adjusts to the unpredictable at all organization levels of the organisms, through cells (cytoskeleton and microtubules); vascular system; to the behaviour of the organism as a whole (as in learning).

The existence of these mechanisms allow phenotypic adjustment to changes elsewhere in the phenotype without correlated genetic change. Thus if selection for size or strength results in genetic changes which increase the neck muscle mass of a male deer (via, say, sexual selection), there is no need for further genetic changes to ensure that those muscles are adequately serviced by the circulatory system of the animal. These mechanisms of phenotypic accommodation remove a crucial roadblock that would constrain the evolution of novelty. Limb evolution, for example, requires simultaneous changes in many tissues: bone and cartilage; muscle position and perhaps mass relative to bone; muscle attachment points; innervation of new muscle

tissue; vascular change. Without exploratory mechanisms, the evolutionary coordination problem would severely constrain adaptive change. This problem of correlated change was one reason for thinking evolutionary change must be gradual. A large-effect mutation increasing neck muscle mass on a male deer would decrease fitness unless there were correlated increases in bone mass and shape, and in the supply of blood to those tissues. A small increase in muscle mass might still attach successfully in an unchanged context, while selecting for “catch-up” changes in the associated systems. Thus the deer lineage might inch towards a new male phenotype. But if, within limits, a whole suite of systems are developmentally responsive to signals from their partners, a large effect mutation that (say) increased horn size might be favoured. For its development would trigger the appropriate adjustments of neck muscle, bone and circulatory systems. Phenotypic accommodation finesses the problem of correlated change: a genetically-caused modification of in one system need not wait for a genetically-caused change in associated systems, even when both must change for either to be adaptive.

So, first, mechanisms of phenotypic plasticity enhance evolvability by enabling phenotypic adjustment to genetically-caused changes in an organism. These mechanisms act as change amplifiers: small genetic changes (changes that directly affect only one component of an organism) can result in a suite of adaptively correlated changes. Thus a small change in G-space can map onto a large change in P-space. Consider, for example: the gracile human face: our teeth, face and jaw are less robust than those of earlier hominins and our closest great ape relatives. We do not know the genetic distance between contemporary humans and early hominins, but we do know that the genetic distance between the chimps and modern humans is surprisingly small, given the impressive phenotypic differences between us. These facts about the  $G \Rightarrow P$  map point to a possible explanation. The evolutionary restructuring of our jaw, face and teeth might depend on only a few genetic changes which affect directly only a few elements of the facial complex, together with correlated change mediated by the mechanisms of adaptive plasticity. This is no mere conceptual possibility: it is known that bone growth is very sensitive to the loads imposed on bone in development. It is phenotypically labile. Indeed, there is some suggestion that a common contemporary human dental problem may be due to an

insufficient load on our lower jaw in development, due to a shift in childhood diets to softer foods ((Gilbert 2001) p8).

Plasticity enhances the flow of variation to selection, but it also biases that flow. Some aspects of phenotypes are easily altered; others are not. The switches that control developmental subsystems — that turn them on, and connect them one to another — are easily alterable. In vertebrates, sex determination can depend on a variety of environmental or genetic switches. As lineages evolve, control can be passed from genes to environment or vice versa. But once initiated, the developmental mechanism is highly conserved. So while in some ways the mechanisms of sex regulation in vertebrates is very labile, in other respects it is conservative. The underlying molecular mechanism is conserved, depending on an estrogen circuit with two stable states and the SF-1 protein trigger. There is much lability in how that trigger is pulled, but once pulled, the cascade has a conserved structure across the vertebrates ((Kirschner and Gerhart 2005) pp 90-96). In general, while the regulation of developmental modules varies, the modules themselves are conserved. Each phylum seems to have a common and distinctive pattern of compartmentalisation in the early embryo; a pattern which sets up its stock of developmental modules ((Gerhart and Kirschner 1997) chapter 7). So lineages are at once more and less variable than the isotropic model predicts. Small genetic changes can generate a suite of correlated changes: more change than the isotropic model expects. But some crucial aspects of the phenotype are invariant, or almost so.

The mechanisms of phenotypic plasticity thus modify and in some ways accentuate the variability available to selection. But as Mary-Jane West-Eberhard argues at (great) length, they also make the environment itself a direct source of evolutionarily significant variation. The environment induces variation; it does not just select genetically-induced variation. For one thing, environmental change can make both new signals and new resources available to developing organisms. For example, many insects protect themselves with poisonous chemicals without manufacturing those chemicals: they are found in the plants they eat, and the insects have evolved the capacity to tolerate and sequester them ((West-Eberhard 2003) pp 501-5). It is likely that such novelties often have environmental causes. The environment changed as

plants evolved chemical defences. But some insects in the environment managed to accommodate ontogenetically to this change, perhaps by storing these chemical relatively harmless in their tissues, thus leading to a subpopulation with a novel phenotype. Subsequently, this capacity has been elaborated and fine-tuned by genetic accommodation, giving us the array of chemically protected butterflies and other insects that we now find<sup>5</sup>.

It is true, of course, that environmentally-induced variations of this kind do not thereby rewire the organism's genome, thereby transmitting the novelty to the next generation. Even so, environmentally-induced variation is often evolutionarily important. One important contrast between mutation-driven and environmentally-induced novelties concerns the size of the initial population of novelties. Initially a mutation-triggered novelty will be very rare; carried only by the organisms in which the mutation occurred and some of its direct descendants. An environmentally-induced novelty is likely to characterise a significant sub-population right from the beginning: since its development depends only on widely-shared mechanisms of phenotypic plasticity, it is likely to develop in many of the organisms exposed to the environmental trigger. The novelty is therefore much less likely to drift out of existence. Its effects on fitness are scrutinized much more effectively, not just because the novel phenotype is less likely to drift to extinction, but also because there is likely to be variation in the novel phenotype itself. For those with the novel phenotype are likely to differ both in the details of the new trait and in how well the rest of the phenotype accommodates to the novelty.

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<sup>5</sup> Of course, this is not the only possible trajectory. There is an alternative possibility: the plant develops a toxin which drives the ancestral caterpillars from it: they retreat to alternative food sources, to wait on a favourable mutation, conferring metabolic protection against the toxin. Once this mutation arrives, eventually, the caterpillars can reinvade, and once they have done so, natural selection on continuing genetic variation elaborates this adaptive complex. In this scenario, the novelty is selected by the environment but not induced it: the toxin-protected leaves are part of the selective environment, but no part of the explanation of the origin of the novelty. Generalists might evolve toxin resistance by this route but specialists are less likely to do so. For the mutation-lead event has to be combined with the fortuitous accident of host-switching for the mutation to be selectively favoured.

Moreover, some mechanisms by which phenotypes adjust to new environments are biased in the direction of adaptive response, and thus environmentally induced change is an especially important route to the evolution of adaptation. For in many cases the underlying mechanism is some version of trial and error, with the selective stabilisation of success. The proximate signal of successful learning — physiological reward — is not, of course, perfectly correlated with fitness-enhancing actions. But it is certainly to some extent correlated with fitness, and the same is true of other exploratory mechanisms. For while learning is an obvious example of adaptive accommodation, it is by no means the only one, as West-Eberhard's wonderful example of the two-legged goat illustrates. Finally and crucially, the environmental changes which induce a novel phenotype in generation N — say, the invasion of a plant species with new toxins in its leaves — continue to act. So the novel phenotype will be induced again in the next and subsequent generations. The novelty is induced as a multi-generational and varying subpopulation. The novel phenotype is generated in significant numbers, over several generations, and it has fitness consequences. So it will trigger processes of genetic accommodation: there will be selection on genes which modify the novel phenotype.

Genetic accommodation can take many forms. Suppose, for example, that the novel phenotype is deleterious: the caterpillars that eat the new plant with its new toxins survive and develop, but have reduced fertility as butterflies. Then genes which modify or suppress those effects will be selected. Suppose that the phenotypic effects are on balance positive but with negative side-effects; the toxins protect the caterpillars but at some cost to fertility. Then there will be selection on genes which suppress the side-effect. Suppose the new phenotype is superior: the caterpillars can store the toxins harmlessly, buying protection without cost. Then genes that make the development of this phenotype more likely will be selected: for example, genes which make the plant more perceptually salient to the caterpillar. The much-discussed Baldwin Effect is a special case of genetic accommodation to an environmentally induced phenotypic change: accommodation that makes development of the new phenotype less contingent on specific signals from the environment.

Avital and Jablonka have sketched a second special case: their “assimilate and stretch” model of the evolution of behavioural complexity. On this picture, agents face cognitive bottlenecks, for they are under selection for the elaboration of cognitively demanding skills. Consider such traits as complex courtship displays, complex bird song, bowerbird bower building, language. Suppose that a species of bowerbird is under selection for bower-quality: for an individual male bird, the more elaborate the bower, the better. And suppose that learning to build a bower is cognitively demanding. A bird's cognitive resources constrain its capacity to build an elaborate and attractive bower. If the basic bower building skills no longer had to be learned, the cognitive bottleneck on such birds would be eased, and their cognitive resources would then stretch to a more complex bower. Bower-building as a whole would have both innate and learned elements, even as bowers became more elaborate. Further canalisation would then allow further stretching to more elaborate bowers yet, though still built through an ensemble of learned and innate elements. And so on. At each stage the baseline is raised. But female preference for elaboration selects for further canalisation, and hence we get runaway evolution of the trait in question. (Avital and Jablonka 2000; Jablonka and Lamb forthcoming).

Avital and Jablonka took their mechanism to describe one possible, though important, dynamic between canalisation and learning. But our hypothetical (though not very hypothetical) butterflies suggest that it might have more general application. It applies to any circumstances in which (a) there is enduring selection on a trait along a dimension. For our caterpillars, the more toxin-resistant they are, the better. They are better protected and have more access to food. (b) Though there are mechanisms of phenotypic plasticity that shift trait value in the right direction, there are resource limits on these mechanisms. (c) There are genetic changes which would shift the norm of reaction in the right direction. For example, there might be genetic changes which increase the number of cells in which the toxin can be stored; which up-regulate the production of antidotes, or which increase the extent to which vulnerable eggs are buffered against the toxins effects. Toxin-resistance would still depend on mechanisms of phenotypic accommodation, and the toxin would still present as an environmental stress, but the overall effect of genetic and evolutionary change would be to ramp up the quantities of toxin the caterpillars could safely ingest and store. It is

possible that the “assimilate and stretch” model might have general application in explaining runaway selection.

Avital and Jablonka’s work on learning and behavioural traditions leads naturally to the next theme of this paper: the role of the organism itself in causing the environmental changes which in turn cause changes in the supply of variation to selection. For they point out that the upstream generation profoundly affects the learning environment of the downstream generation, and hence profoundly affects behavioural competence, and variation in behavioural competence, of that downstream variation. The role of parents in shaping the behaviour of their offspring is obvious in species like ours, where both parents and children have psychological capacities that have been selected to enrich and to stabilise the flow of information across generations. But those capacities are rare: they characterise few species. It is important that stabilised behavioural traditions are not restricted to those species that are highly adapted to cultural learning. The ordinary ecological activities of parents structure the learning environment of juveniles. When offspring live with their parents, as they do in many species, adult activity structures the learning environment of the juveniles. Even in those species without special adaptations for high-fidelity cultural transmission, the result can be the reliable flow of information across generations. Once chimpanzees (for example) begin to termite fish regularly — once the harvest of that resource becomes a regular aspect of their foraging — juveniles will have many opportunities to acquire termite fishing skills by undirected trial and error learning (Avital and Jablonka 2000). The initial innovation might have been a low probability event, but once its made, it changes adult lifeways, enhancing the learning opportunities for the next generation. Innovations can thus be entrenched in local populations. Behavioural traditions of this kind are important expansions of evolvability, because they prepare generation N+1 phenotypes for local environmental conditions: conditions too local in space and time to be tracked by specific, genetically mediated adaptations.

The learning environments of mixed generation species are not found; to a very considerable extent they are made. The experience of the juveniles depends profoundly on adult activity: their learning world is a made world. As we shall see,

this is true not just of juveniles learning in populations with a mixed age structure. The world organisms experience is in part their own product.

#### **4. The Organism-Environment Developmental Loop**

Genetic accommodation in its various forms initiates cascades of gene changes which in turn result in further phenotype changes, both directly and through further phenotypic adjustment. It thus makes environmentally-induced phenotype novelties evolutionarily significant. Sometimes the environment changes for reasons independent of the local biota, and that biota responds. The climate or the sea level changes; new species migrate in; others become locally extinct. But organisms often change their own environment. Termites, earthworms, parasites, trees do not live in the world as they find it. These organisms and others in part remake their world. As the theorists of niche construction have shown in such compelling detail, in acting on their environments, these organisms profoundly change the selective forces acting on them (Odling-Smee, Laland et al. 1996; Odling-Smee, Laland et al. 2003).

Termite mound and tunnel systems take most insectivores out of the termite danger equation, and moderate the ranges of temperature and humidity to which they are subject. The environmental changes termite engineering initiated have selected for modification in termite phenotypes until virtually nothing is unaffected by life in the mound (Turner 2000). There is, then, a selection-mediated feedback loop from the organisms in a lineage to further evolutionary change in that lineage. The current analysis shows that there is an additional feedback loop. The agents that compose a lineage can influence the supply of variation to selection and hence (as we have seen) its evolutionary trajectory.

Consider first niche choice. Organisms often choose their environments: they recognise and respond to environmental stimuli, and those responses result in sampling their physical surrounds selectively. One consequence is that they sometimes find themselves colonising new adaptive zones, both ecological and geographic. When organisms disperse to new habitats — for example, birds, insects or seeds being blown to islands — they clearly experience new selective environments. But such dispersing organisms also experience new physiological environments and (if the initial colonisation

survives) their offspring will experience new developmental environments. Often the same will be true when organisms colonise new ecospace: for example, when phytophagous insects switch hosts. Exploring ecospace has been very important. Such exploratory behaviour has been the leading edge of world-transforming change: for example, the invasion of sea-floor substrates at the beginning of the Cambrian and the colonisation of terrestrial habitats. Major shifts of adaptive zone result in major morphological and physiological re-organization, but they are almost certainly initiated by dispersal and exploration, and first made possible by mechanisms of phenotypic adjustment to altered environmental conditions<sup>6</sup>. The exploration of new ecospace, and especially involuntary exploration due to forced dispersal through storms and the like, taking organisms to quite unfamiliar environments, is likely to generate in the survivors new variation. Genotypes which are equivalent in one environment need not be equivalent outside the normal range. Stress can result in novel phenotypes, making previously unexplored ranges of norms of reaction relevant to selection and exposing once-hidden genetic variation to selection.

West-Eberhard argues that the importance of these phenomena have been understated by lab-based genetics. By narrowing the class of rearing environments, genetic control looks simpler than it really is. As the class of rearing environments becomes broader as organisms spread through a variable habitat, multiple influences on phenotype become more common. This facilitates genetic accommodation to environmentally-induced novelties, for there will typically be many genes influencing the form, extent, timing and phenotypic adjustment to a novelty ((West-Eberhard 2003) pp 505-515). So niche construction promotes variability by exposing organisms to stressful environments, and, more generally, environments outside the range of previously evolved canalisation mechanisms. Those are also populations subject to strong selection pressures. As selection intensifies, so to variability in the population is likely to increase.

Next, consider the norm of reaction concertina. The effects on phenotypes of novel environments are a special, though extreme, case of a general pattern. The idea of a

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<sup>6</sup> For suppose the lucky mutation (or mutations) that increased tolerance to (say) temporary exposure on mud surfaces and the like came first. There would be no selection for these mutations unless they were accompanied by a behavioural change to take advantage of these increased tolerances.

$G \Rightarrow P$  map is misleading. For the phenotypic effects of genes are context-sensitive. As we vary the environment, the same set of genes map onto different phenotypes. For example, amongst the insects there are many seasonal polyphenisms: the same butterfly can look very different, according to its hatching season. (For a brief overview, see (Gilbert 2001)). The sensitivity of gene effects on phenotype is often captured through the concept of a reaction norm (see, especially, (Schlichting and Pigliucci 1998; Lewontin 2000) ). We represent a reaction norm when we graph the phenotypic effect of gene/environment combinations. Occasionally reaction norms are flat (showing the phenotypic effects of a gene are insensitive to environmental variation) but that is the exception rather than the rule. As a consequence of the environmental sensitivity of gene effects, the environmental heterogeneity that a population experiences is relevant to the supply of variation it presents to selection. In turn, that heterogeneity depends in part on both niche choice and niche engineering by the agents that compose that population.

Niche construction helps determine the relevant range of the reaction norm: which areas of E intersect with G to produce P. Niche choice tends to extend the relevant range of norms of reaction: a given gene experiences more environments as a consequence of active exploration and choice. Phenotypic plasticity accentuates this exposure of the reaction norm; it expands the concertina. Plasticity in generation N tends to expose more of the norm of reaction to selection at generation N+1, because plastic organisms have a broader range of environmental tolerance. They reproduce in this broader class of environments and that widens the class of Es in which different Gs act.

Ecological engineering — the most dramatic and obvious form of niche construction — tends to narrow the relevant range, though at the same time that narrowed range is shifted by comparison to the ancestral state. Burrows, warrens, beaver lodges and similar structures buffer the effects of environmental variation: they make the experienced environment of those living within more homogeneous than they would otherwise be. Think again of termites. One consequence of the evolution of eusociality is that the social world of the nest and the physical environment of the nest have become crucial and stable features of the termite world. That world is very

different from the world of termite ancestors: so there has been a shift in the range of E in which termite eggs and larva develop. But for a given termite species, the nest environment is stable: there is some variation in size and construction from nest to nest. But many species have distinctively shaped nests with a characteristic architecture, so termite genes are expressed in a narrowed range of environments.

Sometimes both the extension and the channelling of reaction norms can be combined in a single process, as in Schlichting's model of the origins of cellular differentiation in multicellularity. Imagine a clone of cells aggregating after mitosis rather than drifting apart, perhaps initially to avoid being engulfed by other protists. The central cells in the cell ball will have a uniform and narrowed environment, consisting just of others like themselves. But edge cells will experience the world differently: with an internal interface as well as one with the less predictable and more hostile external environment<sup>7</sup>. The two cell populations will, therefore, explore different regions of their common reaction norm and may not be phenotypically identical. Differentiation may have begun as a by-product of differential exploration of a common norm of reaction, before its selective elaboration (Schlichting 2003).

Novelty and Squeezing The Concertina. This suppression of environmental variation may well be important to the evolution of developmental control over complex phenotypes. Maynard Smith and Szathmari emphasize the fact that evolutionary transitions are transitions in the kind and quantity of information transmitted across the generations. For phenotype complexity depends on a cross-generation flow of information and phenotypic complexity. If the complex phenotype is heritable, the cross-generational signal has to be both rich and of high fidelity to control a complex developmental process (Ridley 2000). But since genetic signals can do nothing more than initiate developmental cascades, genetic signals must be inserted into a sufficiently structured and predictable developmental environment. So in the typical case, an organism of generation N influences the phenotype of its offspring in N+1 in two ways. It contributes to the genetic complement of the N+1 individual. And it

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<sup>7</sup> The same is true, though to a lesser extent, if we think of differentiation as evolving in a cell film, so that each cell has feeding access to the external environment: central cells in the film will have a more protected, more cell-neighbour environment than those on the edges of the film.

engineers the developmental environment of those genes; it structures the way those genes are read. The egg (and its equivalent with plants and fungi) is a structured system: adapted both to function in an environment and to provide an initial set of triggers for gene expression. Eggs vary remarkably from one another; indeed, perhaps they vary more than the early embryos into which they develop (Gerhart and Kirschner 1997) chapter 8, (Raff 1996) chapter 6). But for all their differences, animal eggs must provide positional information that controls the initial differentiation of the early embryo. So eggs vary, but all are maternally engineered (for a review, see (Gilbert 2003) chapters 7, 8). Some parental engineering begins and ends with the egg, but many organisms engineer developmental environments more extensively. This structuring takes many different forms. Some parents provide key developmental resources: for example, many invertebrates have elaborate systems for the transmission of symbiotic micro-organisms from offspring to descendant. Development may be buffered by specifically adapted internal or external structures: cocoons, wombs, nests, burrows are engineered developmental environments. Even when the N-organism does not engineer the developmental environment of the N+1 organism, they quite often select them: many female Lepidoptera, for example, choose their egg locations very carefully.

Developmental engineering plays a crucial role in the evolution of novelty. If the defenders of the “developmental revolution” hypothesis are right, the adapted egg played a central role in the Cambrian explosion and thus in the invention of complex bilaterian animal life. On this view of the Cambrian, the earliest bilaterian animals had a simple developmental system in which the cells of the developing embryo differentiated immediately, and divided and moved to their adult positions under local control. But while developmental programs of this power sufficed for the reliable development of a very small, not very complex worm-like animal (somewhat akin, the idea goes, to the planktonic larval forms of some living animals) it does not suffice for larger and more complex animals: animals with appendages and complex sensory systems. The evolution of more complex animal life depended on the evolution of populations of “set-aside cells” — cells whose cell fate is not determined at the point at which development begins. And it required the evolution of compartmentalized development; a developmental organization in which the

developing embryo divides into a series of regions, which then develop semi-autonomously (Peterson and Davidson 2000; Davidson 2001; Erwin and Davidson 2002). This developmental recipe depends on positional information in the egg — information from which the initial embryo geography of autonomous compartments can be derived. Bilaterian complexity, on this view, requires downstream developmental engineering of the egg.

The developmental revolution hypothesis remains controversial. But Brett Calcott has pointed to more general considerations with a similar theme. Complexity depends on reconciling two competing demands of differentiation and shared evolutionary identity. The generation of benefit requires differentiation; the division of benefit requires identity. It pays cells to stick together because of collective synergies in survival and in gathering resources. But this phenotypic power of complex animal and plant life depends on specialisation and the division of labour, and hence on cellular differentiation. August Weismann, the great German evolutionary theorist, supposed that cellular differentiation depended on the differential distribution of heritable factors from the germ-line: nerve cells are different from skin cells because they contain different genes, not because they have different genes active (Maynard-Smith 1989). In retrospect, perhaps it is no accident that Weismann's hunch was wrong, for a genetically chimerical body might well be an unstable alliance. Differentiation with identity, though, depends on signals from the previous generation. We see this in eusocial insects: the difference between queen and worker is a difference in upstream intervention. It is true of differentiation within multi-celled animals, too. Once development with differentiation begins, differentiation can be amplified through internal feedback loops. The compartment geography of the embryo is not already present but cryptic in the egg. But the initial differentiation — telling some cells in the early embryo where they are and what to do — depends on traces generation N insert into or attach to the adapted egg ((Gerhart and Kirschner 1997) chapter 8).

If these arguments are right, even roughly, then crucial evolutionary transitions depend on the impact of organisms on the spread of phenotype variation, an impact routed through their contribution to developmental environments. To the extent that phenotype similarity across generations depends on signals sent across generations,

those signals depend on channels and contexts engineered by the parental generation. If there is something to this perspective on novelty, what might it tell us about phylogenetic macroevolution? West-Eberhard has suggested that there is a critical connection between plasticity and phylogenetic macroevolution; in particular, between plasticity and adaptive radiation. For she points out that in the very same regions in which some lineages radiate, others do not. To go with the thirteen species of Galapagos finches, there is just one warbler and two flycatchers. Of these, only the Galapagos flycatcher is endemic. West-Eberhard suggests that such differences are explained by differences in phenotypic plasticity in the stem lineage. A difference in evolvability derives from differences of adaptive plasticity. Plasticity allows a lineage to simultaneously retain the capacity to change while enabling populations to specialise in the particular resources a given habitat makes available. Such populations use their pre-existing capacity to generate novelty in partnership with environmental inputs. Radiation is the response of a plastic stem species to a heterogeneous environment.

This hypothesis really does seem to fit the African lake cichlids. There is evidence that their tooth and muscle development is diet-dependent ((West-Eberhard 2003) pp575-577). Moreover, different morphs tend to specialise in different foods when food is scarce: competition seems to increase specialisation. So the spectacular radiation of the cichlids may depend on developmental plasticity in learned feeding specialisation and tooth, jaw and muscle development acting in conjunction with modularity and redundancy in their double jaw system. Likewise, the radiation of Darwin's finches may well be mediated by learning. There is evidence that these birds do learn feeding specialisations, and that those specialisations lead to the formation of subpopulations with different feeding zones, different feeding methods, and different foraging targets ((West-Eberhard 2003) pp 345-349; pp 582-583). However, this conception of the connection between plasticity and adaptive radiation is incomplete. How do subpopulations become species?

West-Eberhard downplays the role of isolation and speciation in the evolution of novelty. She points out that significant novelties arise as variants within populations: for example, the transitions amongst insects from phytophagous life styles to

carnivorous ones can begin as a within-population variation. Likewise, crucial transitions in the evolution of eusociality exist as facultative variants within populations ((West-Eberhard 2003) p606). However, the crucial issue is not with the origin of novelties but with the genetic accommodation that makes them a more permanent, less contingent aspect of a phenotype. The second pulse on the double-pulse model of the evolution of novelty depends on isolation. For accommodation will involve the evolution of a distinctive set of gene combinations, and these would be broken up by gene flow between novel and original phenotypes. Thus the plastic stem must act in conjunction with isolation mechanisms in order to generate an adaptive radiation. One possibility is that the novel phenotype generated by environmental induction themselves establish extrinsic barriers to gene flow. For example, some pairs of the cactus finch breed in the dry season, because they have both access to and can digest pollen. Most birds feed in the wet, relying on the insect flush for the protein they need. So exploiting a different resource can shift reproductive schedules, which would in turn limit gene flow ((West-Eberhard 2003)(p584)).

It is not clear how general this mechanism is. But while a piece of the puzzle may be missing, some crucial pieces seem to have come into focus. It has been clear for some time that if the mechanisms that generate variation are biased, then those mechanisms play a central role in explaining evolutionary trajectories. Phenotypic plasticity connects phenotypic variation to environmental change, and thus shows that the mechanisms that generate variation are indeed biased. Amongst those mechanisms are the activities of organisms themselves, as they explore their world, choose and engineer their environments, and structure the context in which inherited resources are activated.

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