

What Can Development Tell us about Evolution?

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Abstract

The neo-Darwinist theory of evolution is fundamentally about genes. Random mutations occur, those that produce advantageous phenotypic traits spread through the population, and that is how change happens. Any trait of an organism is to be explained by giving an account of the advantage it gives, and, if possible, by indicating a sequence of small changes by which it could have appeared. Development, the process that comes between the genome and the phenotype, is largely ignored as irrelevant to the outcome. It is seen as the study of construction rather than of architecture and design. Even the field of study known as “evo-devo”, that purports to bring evolution theory and developmental biology together, is about genes and their regulation.

Organisms are, however, constructed by physical and chemical processes. Nature can only select from among the forms that can appear, and the forms that are most readily made by developmental processes are the ones that are the most likely to be selected, not because they are necessarily the best conceivable but because they are available for selection.

Many years ago, CH Waddington pointed out that developing organisms possess what he called chreods, homeorhesis and canalization. These shared properties largely explain why there are discrete species instead of continuous variation and why major changes in evolution are likely to occur rapidly rather than by the gradual accumulation of small ones. These properties do not have to be explained as products of natural selection. They are typical of complex dynamical systems, which organisms certainly are.

Introduction

The waves of the sea, the little ripples on the shore, the sweeping curve of the sandy bay between the headlands, the outline of the hills, the shape of the clouds, all these are so many riddles of form, so many problems of morphology, and all of them the physicist can more or less easily read and adequately solve: solving them by reference to their antecedent phenomena, in the material system of mechanical forces to which they belong, and to which we interpret them as being due....

Nor is it otherwise with the material forms of living things. Cell and tissue, shell and bone, leaf and flower, are so many portions of matter, and it is in obedience to the laws of physics that their particles have been moved, moulded and conformed.

D'Arcy W. Thompson [1]

In this beautiful quotation, D'Arcy Thompson is expressing something that many others also find puzzling. Why is explanation in biology so different from explanation in physics? Why, to put it in Aristotelian terms, do biologists concentrate on the final cause, which has disappeared from the physical sciences many centuries ago? It has been a very long time since anyone thought that heavy bodies fall to earth to achieve their proper positions.

To see what D'Arcy Thompson was driving at, look at Figure 1, which consists of three shapes taken from his book *On Growth and Form* [1]. The one on the right is a medusoid, *Cordylophora*. Note especially the tentacles that get more and more ramified as they go out from the body. How are we to explain this complicated shape in what seems to be an otherwise simple organism?

Tentacles are useful for getting food, so they presumably confer a selective advantage. And it should not be too difficult to construct a story about how medusoids evolved from a more or less spherical ancestor by a long sequence of small changes leading to the complex shape they have today.

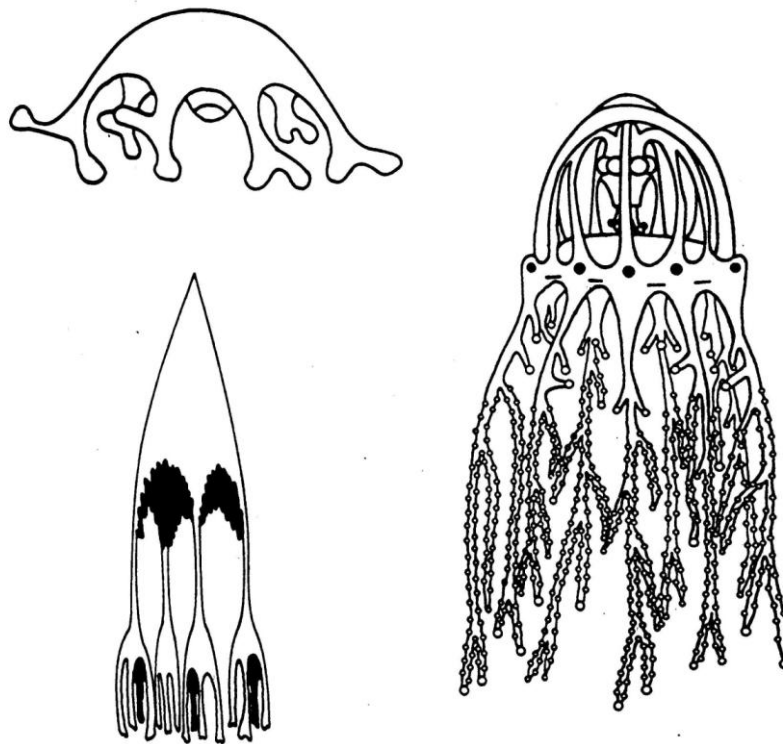


Fig 1. Cordylophora, Caldomena and a drop of fusel oil in paraffin. After D'Arcy Thompson (1917).

The shape on the lower left, however, is not a medusoid. In fact, it is not an organism at all. It is a drop of fusel oil that has been allowed to fall into paraffin. Now a drop of fusel oil doesn't have a genome and it doesn't have ancestors. It certainly is not subject to natural selection. It has that shape through the interaction of the physical forces as a drop of one liquid passes through another, depending on the speed and on the densities, viscosities, and surface tensions.

Are you still inclined to accept the story about the tentacles on medusoids? They do seem to be useful, and you'd expect them to be selected for once they appeared. All the same, wouldn't you agree that the chief reason the medusoids have their characteristic shape is that it happens to be one that is easily produced as they develop, even if that isn't obvious at first glance? The growth and development of a medusoid is, after all, very much like a drop falling through water, though with the membrane replacing surface tension.

And from the mathematics and the physics of the situation, we are led to doubt that the tentacles appeared one at a time. What is much more likely is that the proto-medusoid was more or less spherical in shape and then a number of tentacles appeared all at once, in the sort of sudden jump that isn't supposed to happen in evolution. It also suggests that the medusoids are not all necessarily closely related; the easier a shape is to produce, the less its appearance counts as evidence for common ancestry.

Random variation and gradual Evolution

Neo-Darwinists insist that evolution proceeds by the natural selection of *random* variations. And they mean random not just in the sense that they do not occur preferentially in the directions in which they are wanted. They mean random in the strong sense that there is really nothing we can say about them. And that means explanations like the one I just suggested play no part in their work.

It's also why evolutionists have always insisted that all change in evolution is gradual. That's certainly not because of the evidence: the gaps in the fossil record were one of the objections to the acceptance of the idea of evolution in the 19th century and they are still used by modern creationists to attack evolution. It would be much easier to counter the so-called creation scientists and intelligent design people if evolutionists didn't keep offering them the easy target of gradualism. The evidence for evolution is overwhelming; the evidence for gradualism is not.

But whatever the evidence says, if you insist that the individual variations must be random then they must also be small, for the chance that a large totally random variation will be anything but disastrous for the organism in which it occurs must be infinitesimal. Thus Darwin [2] wrote that change has to be gradual, quoting the maxim *Natura non facit saltum*. Dawkins [3] has written that "gradualism is of the essence". And in his paper for this meeting, Francisco Ayala [4] tells us that according to Darwin's theory of natural selection, "the design has arisen gradually and cumulatively, step by step, promoted by the reproductive success of individuals with incrementally more adaptive elaborations."

Yet you do not have to know much about development to know that large coordinated changes are possible. During development, the components, if I can call them that, are developing together, automatically adjusting to fit and to work properly together. The two bones that meet in the ball and socket joint in my shoulder fit together so well because they

developed and later grew together. They did not get their precise shapes separately under independent genetic control.

In a car, if you decide to use larger pistons, you have to design larger cylinders, larger cylinder heads, a heavier crankshaft, and so on. In an organism such as a human, if a mutation were to cause a bone to become longer, you wouldn't have to wait for separate mutations to lengthen the muscles, tendons, nerve fibres, blood vessels and so on. They would automatically adjust to fit. And that is why large coordinated changes are possible.

We can see this in the children who suffered because their mothers took thalidomide during pregnancy. Many were born with very stunted limbs. But the developmental process had coped with the changes caused by the thalidomide and produced a viable if not especially useful limb.

To be sure, this was the effect of a chemical, not a mutation. But what exactly do mutations do? Genes do not produce traits, they code for gene products (at least in their original, simple, molecular biology definition). In other words, they act through chemicals. And in fact there is a rare mutation that produces much the same effect as thalidomide. So what happened was an example of a *phenocopy*, a variant produced by some environmental perturbation – a chemical, a heat shock, an electromagnetic field – that closely resembles what can happen through a mutation. The phenomenon is quite common, but largely ignored by biologists.

Thresholds and evolution

We know from observation that organisms have the capability to adapt to some quite large changes. But how can large changes happen in the first place? Well, we know that development proceeds by physical and chemical processes. And that means typical properties of physical and chemical processes can have important typical consequences for evolution. In particular, abrupt changes in development arise through abrupt changes in the processes by which it occurs. These appear as discontinuities in the equations we use to model the processes, and some powerful results of the French mathematician René Thom [5] allow us to make some general statements about them. (For a more accessible introduction to the mathematics, see *Introduction to Catastrophe Theory* [6]; and for its biological implications see [7].)

To make the discussion easier to follow, let us work in terms of a chemical model. This must, however, be understood as only an illustration. The same general behaviour should be observed in most cases where a sudden change occurs whether the process by which this happens is physical, chemical, or a combination of the two. Unless we are up against something quite unexpected, which here basically means something that cannot be modelled by the sorts of equations that scientists normally use, Thom's result tells us that the properties to be described are generic. (See [8, 9] for a complete mathematical analysis of the model.)

Suppose we have a gene product S that governs the concentration of a substance g that is important in development; we can call g a morphogen. The two concentrations are related by the following equation, originally proposed by Lewis, Slack and Wolpert [10]:

$$\frac{dg}{dt} = K_1S + \frac{K_2g^2}{K_3 + g^2} - K_4g$$

For a given value of S , the reaction proceeds until the system reaches equilibrium, i.e. until $dg/dt=0$. This determines the value of g , through the equation

$$K_4g^3 - (K_1S + K_2)g^2 + K_3K_4g - K_1K_3S = 0$$

If a mutation occurs that brings about a small increase in S , the result will almost always be a small increase in g . There is, however, a threshold value, S_c , and if S crosses that value, there will be a large change in g .

Thus if S is originally small and changes over time in a sequence of small steps, the equilibrium value of g will almost always change slowly as well, with the magnitude of each change in g depending on the magnitude of the change in S . An increase in S that takes it over the threshold value S_c , however, will result in a large change in g ; the magnitude of this change will be almost totally independent of the change in S that triggered it. Further small increases in S will cause only small changes in g and these will again be proportional to the changes in S . In this way, a mutation that has only a small effect on a gene product, can have a significant effect on the developing organism.

The chemical model has more to tell us, however. A small change in any of the parameters K_i will produce a small change in the curve shown in Fig. 3, *i.e.* in the relation between S and g . In particular, it will cause a small change in the critical value S_c . In most cases this will make very little difference. Suppose, however, that S is only slightly less than S_c and suppose the small change in S_c takes it to below the current value of S . Then g will move abruptly to the same higher value it would have reached if S had been increased. What matters is that S crosses the threshold, not whether this is done by moving S or moving the threshold.

Now the parameters of the equation are determined by the conditions under which the reaction is taking place. So mutations that changed these conditions, by affecting gene products other than S , could have the same effect as a mutation in the gene that codes for S . And an environmental perturbation could do the same, again whether it changed S or something else.

We can now understand both how a single mutation can have a large effect, and also how the same effect can occur by an environmental perturbation. There has been a great deal of work on phenocopies in *Drosophila* (see, for example [11-12] and references given there). Less familiar to scientists but well known among butterfly collectors is that chilling the pupae can produce phenocopies of rare mutants; this is not surprising if we bear in mind that a change in temperature is a simple way of changing how a chemical reaction proceeds.

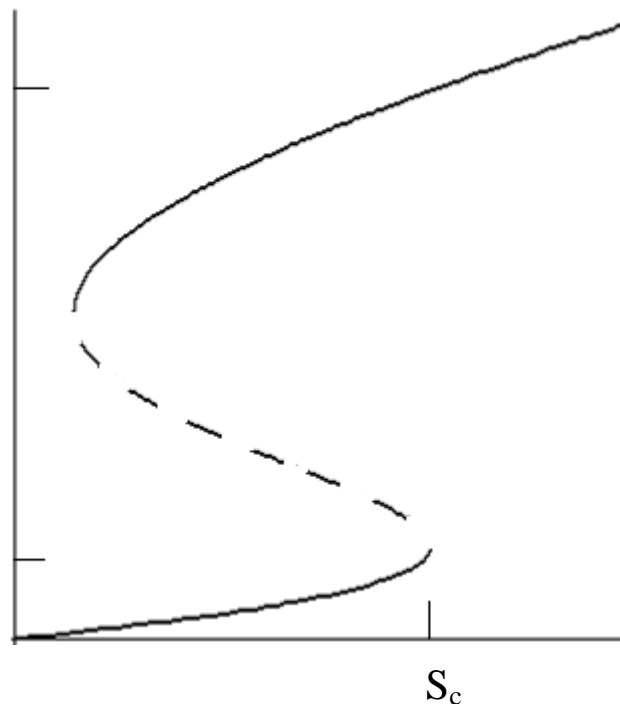


Fig 2. Plot of the equilibrium concentration of the morphogen g as a function of the concentration of the gene product S for the above equations. The part of the curve indicated by a dashed line corresponds to equilibria that are unstable. See [6]. (The values of the parameters used in the computation were $K1=3$, $K2=2.3$, $K3=0.67$, $K4=2.1$.)

This also helps us solve a problem in evolution theory that is usually ignored. If a favourable mutation occurs in a single individual, and if the selective advantage is small, as neo-Darwinists assume it must be (Ayala [4] writes of “incrementally more adaptive elaborations”) then it is very likely to be eliminated by genetic drift (*i.e.* by chance) before it can take hold in the population. That very much increases the time required for a trait to evolve by natural selection.

We can now see two possible solutions to this puzzle. First, if large changes in form are possible, so obviously are large changes in fitness. A medusoid with many tentacles could well have a large advantage over a medusoid with none. Second, if a population is very close to some threshold, then a number of different mutations – some changing S (not all by the same amount) some changing other things -- will all have the same effect. That makes it more likely to occur in more than one individual and the variation more likely to become established. What matters is that the variation is latent in the organism, not a property of a particular mutation.

It also helps us understand how something can evolve when it would take more than one mutation to get to the advantageous variant. Recall, for example, that in the sixth edition of the *Origin of Species*, Darwin was trying to explain how flat fish came to have both eyes on the same side of the head. It is obviously an advantage to have them both on top, but what was the advantage in the first step, when the right eye, then facing downwards, moved ever so slightly forward?

Suppose, to keep things simple, that it takes two separate mutations acting together to bring about some advantageous trait. On the face of it, an individual with only one of the mutations would have no selective advantage over those with none. But this mutation could make phenocopying more likely by bringing the system closer to the threshold. Even if half an advantageous variation is not useful, an increased probability of an advantageous phenocopy is. This adds to the mechanism known as genetic assimilation, in which mutations act to lock in advantageous variations that were already occurring.

Another point is that the way chemical and similar thresholds work [6], we expect phenocopying in at most one direction. There are genetically normal bithorax phenocopies, but this does not lead us to expect to be able to create apparently normal *Drosophila* from individuals with the bithorax mutation. If the phenocopy process plays a role in evolution, and if it occurs in one direction only, then that contributes to the irreversibility of evolution.

Pattern Formation

Sixty years ago, Alan Turing [13] showed that the combination of diffusion and chemical reaction can cause a pattern to form spontaneously in an originally homogeneous region. It has since been shown that other mechanisms can do the same: the motion of cells on a membrane together with the force they exert on the membrane as they move, for example. This is an example of the genericity discussed in the previous section, though there is as yet no mathematical theory to account for it.

Turing was one of the greatest mathematicians of the last century, and he gave his attention only to problems that he considered important. He did this work specifically to do what D'Arcy Thompson had advocated: to show that a feature of an organism, in this case a pattern, could be explained by physics and chemistry rather than by stories about adaptation.

Turing himself said that his aim was to refute the “argument from design”. That term usually refers to the claim of the natural theologians like William Paley that the apparent design of organisms is proof there must be a God, just as if you find a watch you know there must be a watchmaker. Darwin refuted this, of course, but what Turing was referring to was the Darwinists’ claim that the apparent design of organisms is proof of the marvellous power of natural selection.

(Dennett [14] argues that what he calls “Darwin’s Dangerous Idea” is not only the best way to account for all the “occasions for wonder in the world of nature” (p59), it also applies beyond biology. He writes that Darwin’s idea “eats through just about every traditional concept, and leaves in its wake a revolutionized world view.” (p63). Not only does Dennett disagree with Turing’s approach to biology, he now also claims [15] that Turing’s work on computability contributes to the Darwin project because it implies that comprehension too can arise through adaptation. He provides no real justification for this, other than to ask rhetorically why comprehension should be different in this regard from all the other “necessary competences of life”. But comprehension is not a competence, which is why Turing insisted that “Can machines think?” is the wrong question and devised instead what is now called the Turing test [16].)

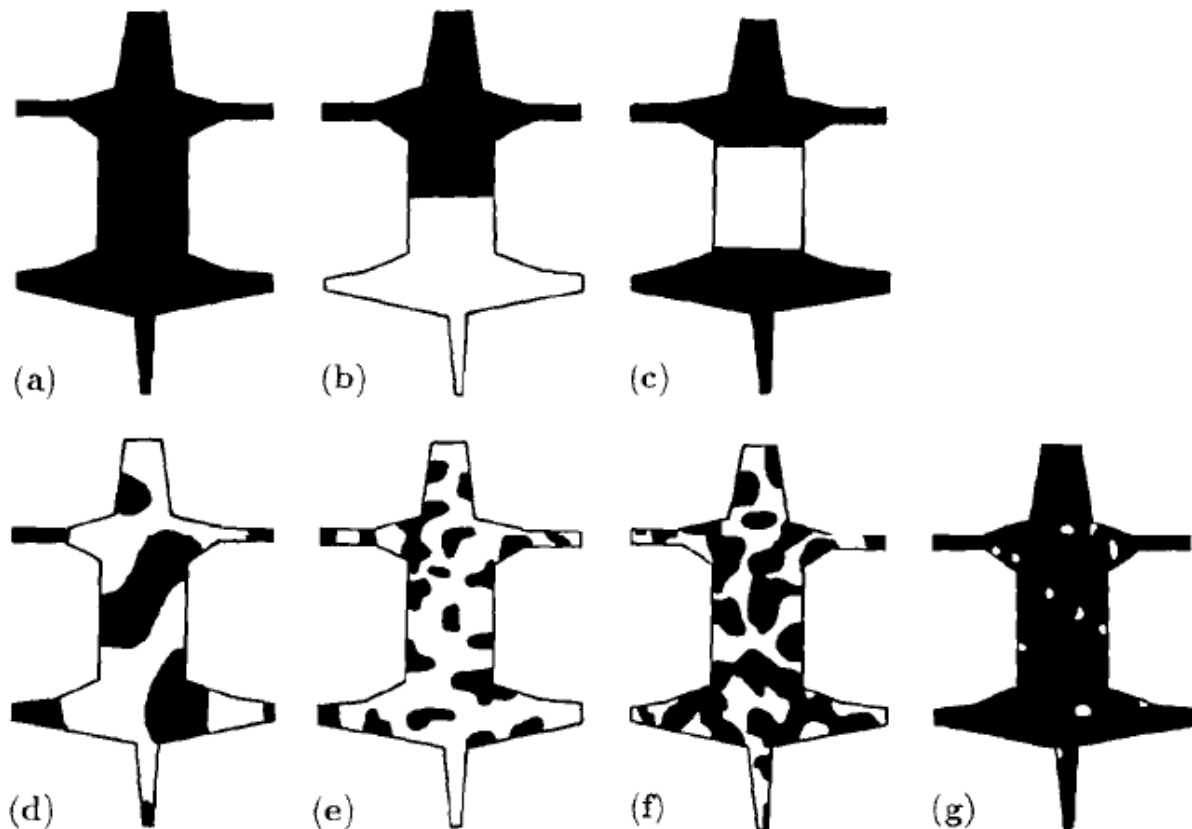


Fig. 2: Effect of body surface size on the spatial patterns formed by the reaction diffusion mechanism. Here they are all drawn the same size for convenience, but (a) is the smallest and (g) the largest. After Murray [16].

Of course we can, if we prefer, construct an adaptationist story to explain this property. Neo-Darwinists consider kin selection to be very important because it helps them explain something that is otherwise very difficult for them to understand. Just as Christians have to cope with the Problem of Evil – how there can be evil in a world that was created by a God who is both omnipotent and good – neo-Darwinists find it hard to understand how there can be good in a living world created by the action of a very powerful and inherently selfish force, natural selection.

One of the explanations that have been suggested is kin selection: we act altruistically towards our relatives because they carry some of the same genes we do [19]. For this to work, of course, we have to know who our relatives are, and that is difficult if everyone looks the same. We can therefore argue that the variability of the markings on many animals has evolved to make kin selection more effective [20].

An alternative explanation is simply that the way the pattern is laid down makes it very unlikely that any two individuals will be identical. Animals may indeed recognize each other by their markings, just as humans can recognise each other by the clothes they are wearing, but in neither case does it follow that the variation has arisen by natural selection.

The Epigenetic Landscape

About 70 years ago, the British embryologist CH Waddington [21] pointed out that developing organisms share some important properties. Organisms of the same species have characteristic developmental pathways – “chreods” – that they follow as they develop. These are stable in the sense that if the embryo is perturbed (though not, of course, too much) during development, it generally returns not to the state it was in when perturbed, but to the same pathway further down. Waddington called this property homeorhesis, to stress that it is the flow down the pathway that is stable, not any particular point along it. Development can typically proceed only to restricted number of alternative end states rather than a whole spectrum. The effect of this “canalisation” is that we observe distinguishable species rather than a continuum of forms.

Waddington illustrated this by what he called the epigenetic landscape. He imagined the developmental system as a looking like mountainous terrain. The valleys represent possible developmental pathways. The precise shape of the landscape depends on a network of guy ropes beneath it; the ropes stand for the effects of the genes and the complexity of the network is to remind us that while genes certainly affect development, they do so in a very complicated way.

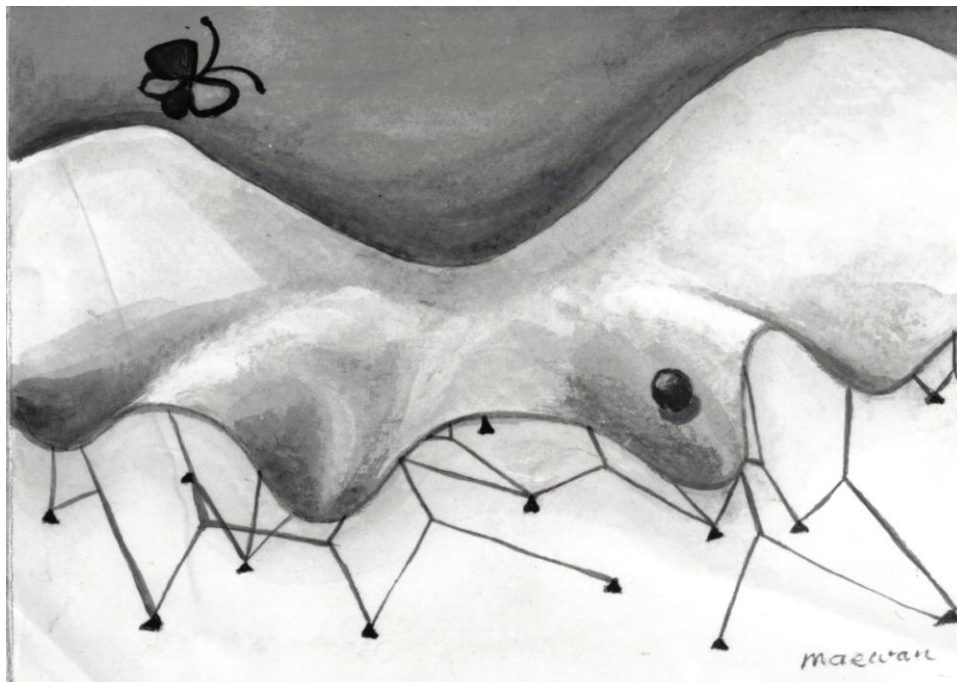


Fig 3. The epigenetic landscape (drawing by MW Ho)

The ball rolling down a valley represents the developing organism, and you can see the properties clearly illustrated: discrete end points rather than a continuum, alternate pathways, stability against perturbation (being pushed up the side of a valley like a bobsled) or most mutations, represented by changes in the ropes that determine the shape.

If the ball is disturbed so much that it is forced out of the valley, it will probably reach a dead end, but it is possible that it will pass over a watershed and then continue down a different valley, just as stable as the original one.

Mutations are most likely to have significant effects only if they disturb the landscape near where one valley divides into two. At such a point even a small alteration in the topography can be enough to send the ball down a different path, i.e. a small genetic change can bring about a large change in the phenotype, and without necessarily affecting the action of any other genes.

These properties are certainly important, so we may ask how organisms acquired them. For a neo-Darwinist, that must have happened, as for every trait, by natural selection acting on small selective advantages over many generations.

There is, however, a more fundamental explanation. Whatever else you can say about organisms, they are highly complex nonlinear dynamical systems. What is more, they are in some sense stable – they have to be pretty much by definition. For if they were not, we would not consider them as organisms at all. And almost any non-linear dynamical system, if it is stable at all, will have these properties. There didn't necessarily have to be such things as organisms, but once we know there are, we expect them to have these properties.

There isn't space to go into this in detail (see [9]) but let me make a couple of points. First of all, nonlinear essentially means that the whole is more than the sum of the parts. Almost all real systems are actually nonlinear, but mathematicians have tended to work with linear systems because they are much easier to analyse. This works well if the system can be approximated by a linear system, but that's only true in a limited number of cases. Unfortunately, it also means that our intuition about what systems typically do is based on linear systems – and I'd say that's even true for people who never write down equations, linear or otherwise. So our intuition is often wrong. In evolution, that means, among other things, that we often confuse what is easy for biologists to describe with what is easy for nature to produce, as with the medusoids.

Second, to get a bit of insight into what nonlinearity does, let me remind you of something you learned at school. The linear equation $ax+b=0$ has in general one solution. But the simplest nonlinear equation, $ax^2+bx+c=0$ has two. So it's not so surprising that nonlinear systems have multiple steady states or rather, because of the sort of dynamical system we are talking about, alternative trajectories. If these are stable at all, they will have the homeorhesis that Waddington described. And because there are distinct alternative trajectories, rather than a continuum of possible pathways, the system will also exhibit canalisation.

Punctuated Equilibria

Ever since the idea of evolution was first put forward, the gaps in the fossil record have been a problem for evolution theory. Darwinists, who insist that evolutionary change is gradual, have always argued that gaps are artefacts. Few organisms leave fossils, and few of those that are formed survive and are discovered. So it's not surprising that we don't have long sequences leading from one form to a quite different one.

Many palaeontologists disagree. They insist that if all those intermediates really existed, they'd have found lots of them. So about 40 years ago, Eldredge and Gould [22] put forward the theory of what they called punctuated equilibria. They pointed out that not only

are there gaps, there is also stasis. Nothing much seems to happen for a long time, then there's a gap and afterwards we find something significantly different. Apparently all the interesting evolution happened just when no one was looking.

Neo-Darwinists can find an explanation for just about anything, and the widely accepted one for punctuated equilibria is based on allopatric speciation. A small group gets separated from the main population, is able to evolve rapidly because of the small numbers, and then moves back and quickly displaces the original form on account of its selective advantage. We don't find the fossils of the intermediates partly because there weren't very many and partly because they wouldn't be in the location where the main population was. It is an ingenious idea, but there is little or no evidence for it, more or less by definition.

But think what the epigenetic landscape suggests. Or, if you prefer not to rely on a picture, it's what the combination of homeorhesis and alternative developmental pathways suggests. Most mutations, like most perturbations, have little or no effect. If however, a mutation, or an environmental change, or a combination of the two, takes a reaction over a threshold and so an organism onto a new developmental pathway, then there can be a substantial change. Macroevolution does not generally occur by a long sequence of microevolutionary steps; it is something essentially different.

And on that picture, punctuated equilibria is precisely what we would expect, rather than something that has to be explained away by a more or less plausible but inherently unverifiable story.

Conclusion

For years those who call themselves evolutionists have steadfastly avoided using physics and chemistry, and, in particular, developmental biology in their work. They have insisted that natural selection can explain everything of interest in the living world. This idea has even been taken up in other fields, giving rise to evolutionary psychology [23] (see also [24]), evolutionary economics [25], evolutionary epistemology [26], ... all the way up (or down) to evolutionary cosmology [27].

Even evolutionists are slowly beginning to realise that this is ultimately a blind alley. We are beginning to hear about "developmental constraints", though only as explanations for things even they can't find plausible selective advantages for: Maynard Smith [28] for example, could only suggest as a possible example that no monocot has a typical flower with more than three petals. And the field known as evo-devo, which is advertised as linking evolution and development, is still all about genes.

It's time to bring evolutionary biology back into the mainstream of science.

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