Genetic revolutions
Evolution and repetition

Rissa:
Another revelation from the DNA record – and something we could only have detected by using it – is how evolution can repeat itself in exquisite detail. Similar adaptations arise in very different species when they're faced with similar challenges.

Sean:
One of the very common ways that animals blend with their surroundings is changes in their body colour. And there's all sorts of examples of species we know that have dark and lighter forms. When we look at the genes responsible for making those colour difference, we can see the same gene altered in a same way in different species, living on different parts of the planet, living in different sorts of habitat. Species as different as fish and birds and reptiles and different species of mammals, the same changes taking place in the same gene.

And what that tells us is given similar conditions, in this case maybe a selective advantage to being darker, that evolution finds a similar solution, the modification of the same gene, and we have many, many cases of this. So we know, for example, that while old world primates invented full colour vision, Howler monkeys which live in the New World independently invented full colour vision by a similar means but at a different time, in a different place, in a different part of the world

Rissa:
Even human evolution displays this theme of repetition. Take sickle cell anaemia, a blood condition that takes its name from the sickle-shaped appearance of the red blood cells in affected individuals. When a person inherits 2 copies of a gene with the sicke cell mutation, the oxygen-carrying molecule in the red blood cells is abnormal and a life-threatening anaemia results. Given this disadvantage, it's astonishing to discover that not only has the sickle cell mutation arisen several times in our evolutionary history but that it persists in human populations. It turns out that individuals who inherit one copy of the sickle cell mutation and one copy of what we might call the normal gene have, in some circumstances, an unusual advantage.

Sean:
What was discovered in the 1950s is that having one copy of that mutation and one call it normal copy of the haemoglobin gene, gave some protective benefit, particularly to young children, against malaria. So they had, for example, fewer parasites, less severe malaria when they were exposed to the organism. So this is a case of a trade-off and it turns out that if mutations in the globin genes will give some protective benefit to malaria, that outweighs the hazards, if you like, of that mutation being at high levels in a particular population.

Now, in places in the world where there is no malaria, we don't see that mutation at all. So that mutation is prevented from becoming common, again by natural selection in areas where there's no malaria, but it's driven to very high frequency in areas that have a high incidence of malaria.

That mutation, the same change in our DNA code, has arisen at least five separate times in different parts of southern Europe, southern India and three different places in Africa, that have led to resistance to malaria in different human populations.

So we can trace with 100% accuracy the origin of different mutations, different adaptations within our species, within different species, to understand that evolution in fact does repeat itself given the same sorts of selective challenges.