Title: Evo-devo and Deep Homology

Narrator: Welcome to this presentation on evo-devo and deep homology. Evo-devo is the study of the role that embryonic development pathways have played in shaping evolution. This is a relatively young field, around 30 years old, that has already answered some very interesting questions. Let’s spend a little more time discussing the fundamental principles behind it.

Slide 2
Title: We Must First Understand Embryology

Narrator: You may not have thought about this before, but multicellular organisms begin life as a single cell. You began as a single cell that resulted from the fusion of an egg and a sperm. You share this inauspicious beginning with all animals.

Slide 3
Title: Human Development

Narrator: You are certainly bigger now than you were then, but how did that happen? The single cell that was the originally you goes through many cycles of growth and division to form a multicellular baby. Not all of your cells have the same properties; you would certainly agree that a cell in your eye has a different form and function compared to a cell in your liver. This is incredible considering that all cells in your large multicellular body contain the same genetic information. So how is it that your cells differentiated and adopted different identities?

Slide 4
Title: Multicellular Forms Undergo Similar Processes

Narrator: Biologists have asked this very question for a long time. Animal embryos are very similar to one another, and, regardless of the species, proceed through similar developmental stages. This suggests that regardless of whether we are following the growth of an embryonic mouse, turtle, chicken, or person, in each organism, similar mechanisms are responsible for determining different identities for cells.

Slide 5
Title: Developmental Control Genes
Narrator: An understanding of these mechanisms began to emerge when scientists studying fruit fly embryos found genes called Hox genes that gave the developing animals' body segments their identity (abdomen, thorax, head, etc.) In addition, these genes were responsible for some segments bearing appendages, while others did not. How? It seems that most of these genes code for transcription factors, that bind to DNA and either promote or inhibit (even halt) the activity of specific genes. So, what was found were genetic control switches; even though all of the cells contain the same genes, Hox genes and other developmental genes control which are expressed and which are not. By varying the timing and location of the switches activity, regions of embryos are given distinct identity including:

- Anterior/posterior
- Dorsal/ventral
- Growth/death
- Etc.

The control switches also effect cell identity by initiating

- Production of specific cellular products

As a simple illustration of the power of transcription factors, imagine a gene that produces black pigment in an animal body. If a transcription factor prohibits expression of the gene, then the animal will produce no black pigment and be pale. On the other hand, if a different transcription factor were present that promotes the expression of the pigment gene then the animal will be dark. The same pigment gene is present in both cases, but the phenotype can be very different depending upon the transcription factor present.

Narrator: Scientists soon discovered that the developmental genes were highly conserved in all animals. By conserved, I mean that much of their nucleotide sequences have been unaltered over time so that they are extremely similar in even distantly related animals. As an example, there is a gene, Pax-6, that tells a developing fly where to form its eyes. Mice (and you) also use Pax-6 to determine where to form an eye. If a functioning Pax-6 is removed from a fly egg and replaced with Pax-6 from a mouse, the developing fly embryo produces a fly eye where it should: a fly eye. The Pax-6 from a mouse and fly are functionally identical even though the genes they control, those to build fly eyes and mouse eyes are not the same. Pax-6 is one of many development control genes shared amongst animals.

This underlying and previously unknown similarity is referred to as “deep homology.” That distantly related animals share these embryonic development regulatory genes suggests that their last common ancestor from hundreds of millions years ago had these genes. The degree of homology is remarkable, particularly
for Hox genes; not only are many of the genes present in all animals, but their position along chromosomes is the same in most species.

Slide 7

Title: Is Animal Diversity a Result of This Mechanism?

Slide Content:
Text: None

Image: Chart illustrating a comparison between the shrimp *Periclimenes* and the copepod *Paranebalia*

Narrator: This developmental system can create novel structures and body designs without necessarily relying on the creation of many new genes. Consider arthropods, a group of animals that includes lobsters, spiders, flies, scorpions, and mites. These animals have noticeable body segmentation with some segments bearing appendages and others not. Appendages differ somewhat in their form depending on where they are located. Different types of arthropod are distinguished from one another on the basis of the segment identity and the appendages that they bear.

For example, both the shrimp *Periclimenes* and the copepod *Paranebalia* have appendages on each of their thoracic segments T1-T5. Not all crustaceans do. But, where in *Periclimenes*, the first three of these are small feeding appendages called maxillipeds, all are walking leg-like in *Paranebalia*. Why? There is not maxiliped gene and a separate walking leg gene. Instead, the activity of a Hox gene, Ubx (Ultrabithorax) varies in each segment. Where there is high activity of Ultrabithorax (indicated in red) a long walking leg-like appendage will form. With no or very low Ultrabithorax, the appendage takes on the form of a maxilliped. This same pattern was found in other crustaceans, prompting the conclusion that much of crustacean diversity (or even animal diversity) can be explained not through the advent of novel genes, but through the differences in the relative expression of these developmental genes.

Slide 8

Title: In Arthropods, Different Appendages Are Variations on the Same Structure

Slide Content:
Text: None

Image: Chart with numerous images illustrating the evolution of appendage specialization

Narrator: In fact, the full range of crustacean appendages is created in this way. Each is a variant on a theme of two branches off of a central stalk. Differential growth of the branches leads to appendage structures that vary in a continuous fashion. The differences in growth are dictated by the activity of the controlling development genes.

Slide 9

Title: Regulatory Genes Make All the Difference

Slide Content:
Text: None

Images: A butterfly and a large fly with halters

Narrator: This sort of activity is seen in insects too. Where flies have small balancing bulb-like appendages called halters, butterflies have a second pair of wings. Why? There is a difference in response to Ultrabithorax between the two species. Yes, Ultrabithorax, the same gene that played a role in appendage formation in crustacean is implicated in appendage, that is, wing formation in flies and butterflies.
Slide 10

Title: Mammals Are No Different!

Slide Content:
Text: None

Image: A bat with wings outspread

Narrator: We see the influence of developmental genes in shaping mammal forms too. Consider the bat. It is different than all other mammals in that it can fly. It is nonetheless similar to mice. Why can’t mice fly? Of course, they don’t have wings. Bats must have a wing gene right? One that is missing in mice. Actually, no.

Slide 11

Title: Regulatory Genes Make All the Difference

Slide Content:
Text: None

Image: Comparison illustrating the similarities between the limbs of a bat and those of a mouse

Narrator: Mice limbs and bat limbs begin their development looking quite similar to one another. What creates the differences over time? Well I am going to greatly simplify this in order to make a point: Bats have a greater expression than mice of a gene called BMP (Bone Morphogenetic Protein) that causes bones to elongate. This creates the long digits of the bat wing. But BMP also causes the webbing connecting digits to die. This activity results in separate fingers in humans (we use BMP also). In bats, another gene called Gremlin disables BMP in cells between the digits, so the webbing remains. Through differential control of genes found in other mammals, wings are formed in bats. This was not the result of a bat wing gene.

Slide 12

Title: Deep Homology Supports Darwin’s Concept

Slide Content:
Text: None

Image: Darwin’s first tree drawing

Narrator: So there is another layer of homology that we’ve discovered in the last 30 years; a common genetic tool kit governing the development of complex forms. I’ve not discussed it, but a similar toolkit exists in plants also. These toolkits are further evidence of common ancestry.

By understanding how these genes regulate development, we are gaining new insights into how these “endless forms most beautiful” can arise through a different type of descent with modification.

Slide 13

End of Presentation