

The skeleton: Size matters

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Vertebrates have in common a skeleton made of segments, the vertebrae. During development of the embryo, each segment is added in a time dependent manner, from the head-end to the tail-end: the first segments to be added become the vertebrae of the neck, later segments become the vertebrae with ribs and the last ones the vertebra located in the tail (in the case of a mouse, for example). In this process, it is crucial that, on the one hand, each segment, as it matures, becomes the correct type of vertebra and, on the other, that the number of vertebrae in the skeleton, and therefore the size of the spine, are minutely controlled.

It has long been known that the identity of each vertebra is due to the activation of a class of genes called Hox. Now, in the latest issue of *Developmental Cell* researchers from the Instituto Gulbenkian de Ciência, in Portugal, the Institute KNAW and University Medical Centre (The Netherlands) show that besides determining the identity of the [vertebrae](#), Hox genes also have a say in how many are going to be formed at all.

There is a huge diversity in number of vertebrae in animals: some have many vertebrae, and are thus longer, like a snake, and others have fewer vertebrae and are shorter, like mice. Vertebrae are made from precursors known as somites, formed in the embryos, sequentially from head to tail. This process is directly linked to growth of the embryo at its tail end: the more it grows, the more somites it makes and, as a result the more vertebrae the adult animal has. Of the many genes involved in this growth, a family of genes called Cdx are known to play a central role.

According to Moises Mallo, group leader at the IGC and one of the lead authors on the paper, 'We knew that some Hox genes are not activated when the Cdx genes are turned off, but this was always considered to be part of a mechanism to ensure that each new somite generates the appropriate type of vertebra. We now show that the activation of Hox genes is also part of how Cdx genes promote growth of the embryo at its tail end: when the relevant Hox genes were activated in the Cdx mouse mutants the embryos recovered and were born with a quite normal vertebral column, proving that the Hox genes were able to compensate for the lack of Cdx. This is a novel role for Hox genes'.

The researchers also show that some Hox genes are important to stop the addition of extra segments, at later stages in development. Indeed, if Hox genes that are usually active later on in development, in the last forming segments, are turned on before their time, in mouse embryos, they interrupt addition of new segments and lead to a tail truncation in the vertebral column.

As Mallo puts it, 'This paper provides an important addition to a long-standing view on the role of the Hox genes - one of the most-studied genes involved in embryonic development: that it controls not only identity, but also number of vertebrae. Although these observations were made in the tail-end region of the embryo, it is very likely that similar mechanisms might be acting to determine the number of segments closer to the head'.

More information: Young et al., Cdx and Hox Genes Differentially Regulate Posterior Axial Growth in Mammalian Embryos, *Developmental Cell* (2009), [doi:10.1016/j.devcel.2009.08.010](https://doi.org/10.1016/j.devcel.2009.08.010)

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