

**PATTERNING OF THE EARLY NEURAL PRIMORDIUM IN THE AVIAN EMBRYO**

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In Amniotes the development of the neural primordium proceeds according to two different morphogenetic mechanisms. One, called primary neurulation, involves the formation of the neural plate which subsequently folds to give rise to the neural tube. Posteriorly to the level of the last thoracic vertebrae the neural tube is formed from the tail bud, a mass of mesenchymal cells in which transition from mesenchymal to epithelial cells generates the so-called medullary cord which gives rise by cavitation to the lombosacral spinal cord. This process has been designated as secondary neurulation. By using the quail-chick chimera system *in ovo*, the morphogenetic movements responsible for neurogenesis were analyzed from the 6-somite stage onwards. It was found that like the neural plate, the cells forming the medullary cord in the tail bud are initially located in the ectoderm before being incorporated in the tail bud. Moreover, in both primary and secondary neurulation, the floor plate and the notochord share the same origin in Hensen's node. Thus, the isotopic graft of Hensen's node of quail into chick embryo at the 6-somite stage generates a floor plate and a notochord made up of quail cells, from the level of the graft down to the extremity of the tail. This backward movement of Hensen's node bisects the presumptive neural ectoderm in its midventral line resulting in the insertion of the floor plate material. These results lead to the revision of previous notions according to which the floor plate differentiates in the neurectoderm under an inductive stimulus arising from the notochord.

The second part of this lecture will be devoted to the role of positional information in *Hox* gene expression in the rhombencephalon. Experiments involving transposition of rhombomeres at different levels of the rhombencephalon in the embryo at day 2 of incubation will demonstrate that *Hox* gene expression is controlled by external cues unevenly distributed along the anteroposterior axis and responsible for the "*Hox*-code" expressed in each pair of rhombomeres. Moreover, it will be showed that changing the *Hox*-code results in the homeotic transformation of neural structures differentiated from the transposed rhombomeres. The spatial and temporal limits of this neural plasticity have been determined and the nature and source of the factors responsible for the above described phenomenon will be discussed.