The next step will be to find out whether *Tlc* has functional homologues in other vertebrate species. Although it is similar in sequence to members of the mammalian sFRP family of Wnt antagonists, none of these has yet been shown to be expressed in the mammalian equivalent of the ANB. However, mutations in other Wnt antagonists, such as the mouse *dickkopf* gene, can cause loss of telencephalic structures. Taken together with the latest discovery, this indicates that studying the role of Wnt antagonism puts us on track for elucidating the molecular basis of telencephalic induction.

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DEVELOPMENT

Sox, flies and neurogenesis

The *Sox* genes encode a family of DNA-binding proteins, and they are divided into several subgroups on the basis of sequence homology. The vertebrate group B genes, *Sox1*, *Sox2* and *Sox3*, are all expressed in the neural tube from the early stages of its development, but their roles in early neural development remain unclear. Now, Buescher *et al.* and Overton *et al.* report in *Development* that the fruitfly *Drosophila melanogaster* could provide some clues.

In the *Drosophila* neuroectoderm, the neuronal precursor cells, or neuroblasts, arise from proneural clusters, which are arrayed along three longitudinal stripes on each side of the embryo. From each cluster, one cell is singled out to become a neuroblast, and lateral inhibition prevents the rest from adopting a neuronal fate. The clusters are specified by proneural genes, including *achaete* (*ac*). The dorsoventral patterning genes *ventral nerve cord defective* (*vnd*), *intermediate neuroblasts defective* (*ind*) and *muscle segment homeobox* (*msh*) confer spatial identity on the ventral, intermediate and lateral clusters, respectively.

In *Drosophila*, there are two group B *Sox* genes — *SoxNeuro* (*SoxN*) and *Dichaete*. *Dichaete* is expressed only in the ventral and intermediate neuroectodermal stripes, and *SoxN* is expressed throughout the neuroectoderm. Mutations in *Dichaete* cause a reduction in the numbers of ventral and intermediate neuroblasts, and in these new studies, both groups showed that mutations in *SoxN* cause specific neuroblasts to be lost from the lateral and intermediate regions. Mutating the *SoxN* gene in a *Dichaete*-negative background generated a more severe phenotype in the intermediate and ventral clusters, indicating that *Dichaete* and *SoxN* act in a partially redundant manner to control the development of neuroblasts in these domains.

To investigate the precise function of *SoxN* during neurogenesis, Buescher *et al.* asked whether it is required for the formation of proneural clusters. They found that although *ac* expression was reduced in *SoxN* mutant embryos, the initial establishment of proneural clusters was largely normal. However, in many clusters, no cells were singled out to become neuroblasts, so *SoxN* seems to be crucial for this step.

Buescher *et al.* also examined the interactions of *SoxN* with the patterning genes *vnd* or *ind*. In a mutant strain in which *SoxN* function was only partially lost, the additional loss of one copy of *vnd* or *ind* increased the severity of the *SoxN* mutant phenotype in the ventral and intermediate clusters, respectively. However, mutations in *SoxN* did not affect the expression of either gene. The authors concluded that *SoxN* might interact with *Vnd* and *Ind* to modulate their activities, but not by controlling their expression.

Much of the molecular machinery that regulates the processes of neural induction and neuronal specification has been conserved throughout evolution, so it is likely that at least some aspects of *Sox* gene function have been conserved too. Homologues of the proneural genes and *vnd* and *ind* have been found in vertebrates, and these genes should provide a good starting point for further investigations into the roles of the group B *Sox* genes in neural development.

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Coiling of microtubule bundles in a retracted axon. Courtesy of Peter Baas, Drexel University College of Medicine, Philadelphia, Pennsylvania, USA.