synapses do not become depressed. The authors suggest that this lack of depression might be observed because the cortical neurons respond to only the first few stimuli in a train (owing to thalamocortical depression), so their synapses have time to recover during the rest of the train.

Further recordings ruled out other potential mechanisms of adaptation. The cortical cells showed no changes in membrane potential, input resistance or membrane excitability following adaptation, indicating that such postsynaptic effects are unlikely to account for rapid adaptation in this system. This contrasts with the previous finding that slower adaptation in the visual system results from membrane hyperpolarization in the cortex, and may represent a general difference between fast and slow adaptation.

Rachel Jones

References and links

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DEVELOPMENT

Sorted!

An important process in the organization of the developing nervous system is the clustering of neurons with similar properties to form nuclei. In a new study reported in *Cell*, Price *et al.* have examined how motor neurons in the spinal cord sort themselves into 'motor pools' — collections of neurons that innervate a common muscle target. It was already known that each motor pool expresses a distinct combination of transcription factors, but here the authors turned their attention to cell-surface molecules that might enable neurons of the same motor pool to recognize one another. Their results indicate an important role for adhesion molecules of the type II cadherin family.

Price *et al.* cloned 15 different cadherins from the chick embryo spinal cord, and they examined their expression patterns at the lumbar level, where the motor pools have been best characterized. They found that most motor pools express more than one cadherin gene, and each expresses a different combination of type II cadherins. This pattern is achieved through two mechanisms: some genes, such as *MN-cad*, *cad-12* and *cad-8*, are initially expressed in most or all motor neurons and are subsequently downregulated in certain pools, whereas others, such as *T-cad*, *cad-6b* and *cad-7*, are activated in a fraction of motor neurons after they have left the cell cycle. The emergence of these expression patterns coincides with the time when the neurons are beginning to segregate, making the cadherins good candidates for driving this segregation.

The authors looked at the development of two motor pools, eF and A, which differ in the expression of a single type II cadherin (A expresses *MN-cad*, but eF does not). They generated mosaic embryos in which *MN-cad* was either expressed ectopically or inactivated in a random selection of cells. Both manipulations led to increased intermingling of eF and A neurons, indicating that MN-cad is important for the segregation of neurons between these two motor pools.

They also showed that misexpression of the transcription factor Er81 causes ectopic expression of *MN-cad*, raising the possibility that the transcriptional profile of motor neurons translates into a cadherin 'code' on the cell surface. So, although this research is still at an early stage, the type II cadherins are already emerging as a possible link between transcription-factor expression and neuronal surface properties in motor pools.

Heather Wood

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