ROS for your roots

Production of reactive oxygen species (ROS) by an NADPH oxidase induces a calcium channel to open and root hair elongation to occur, according to a report from Julia Foreman, Liam Dolan, and colleagues (John Innes Centre, Norwich, UK).

Starting with the rhd2 Arabidopsis mutant, which has stunted root growth and very short root hairs, the authors used transposon-tagging methods to identify the RHD2/AtrbohC gene, which encodes an NADPH oxidase that produces ROS. “When we cloned this gene, we were completely dumbfounded,” says Dolan. But at around the same time, other groups were finding that plants use ROS as second messengers in response to pathogen invasion and to control the movement of guard cells to open and close stomata.

What is unusual about the new work is that both the activity and the protein controlling it have been identified, whereas in the other systems, the proteins that produce the ROS are not yet known.

In rhd2 mutant plants, ROS production is decreased by 50%, there is little accumulation of intracellular calcium in the root hair tip, and little growth occurs. Upon addition of exogenous ROS to the system, however, the calcium concentration rises and root hair elongation occurs, indicating that an increase in ROS concentration causes the rise in calcium. What is not yet clear is how this occurs. Whether the effect is direct or indirect “is a complete black box as yet,” says Dolan. The group is already looking for genetic suppressors of the rhd2 mutant, which will identify other components in the pathway, including the calcium channel itself.

Plants use two types of cell growth: tip growth, which occurs in the root hair, and diffuse expansion, which occurs in the root itself (and in most other cells in the plant). Dolan thinks that one of the important implications of this study is that ROS appear to be required for both diffuse growth and tip growth, implying that the systems share some underlying mechanisms, a situation that was not previously apparent.

Reference: Foreman, J., et al. 2003. Nature. 422:442–446.

Conserving synaptic weight

Long-term potentiation (LTP), a phenomenon by which previously stimulated synapses become increasingly sensitive to stimulation such that the same level of presynaptic input induces a larger postsynaptic output, and long-term depression (LTD), which conversely reduces efficacy at such synapses, have been implicated in memory formation and storage. But computer models predict that without a balancing force of some kind, LTP and LTD will cause neural circuits to go haywire. Now, Sébastien Royer and Denis Paré have identified just such a force that can maintain balance in a network—and might imply that humans have a limited memory capacity.

“There have been lots of studies about what happens in LTP and LTD, but little attention has been paid to the synapses that are not stimulated when you induce LTP,” says Paré, despite the fact that each cell has thousands of synapses, only a few of which gain LTP or LTD.

To find out what was happening at the distant synapses, the authors induced LTP or LTD at a known location within a single neuron in brain slices from the amygdala of guinea pigs and then measured the response to stimuli at physically remote synapses in the same cell. They found that if LTP was induced in one locale, the distant synapses showed a slight depression in efficacy. Thus, there appeared to be an overall compensation for the synaptic activity in the cell, so that it remained constant.

When the group blocked calcium-induced calcium release in the neurons, the distant synapses failed to change their behavior in response to localized LTP or LTD induction. Therefore, a self-propagating wave of calcium released from internal stores appears to be the mechanism of communication between distant synapses.

These results imply that the formation of one memory affects others, since a single neural network is involved in many memories. “I guess one of the depressing implications of this is that there is a limit to how much you can accommodate,” says Paré.

Reference: Royer and Paré. 2003. Nature. 422:518–522.