

## **Fungal Physiology**

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## **✓** Abstract

This subproject is one of many research subprojects utilizing the resources provided by a Center grant funded by NIH/NCRR. The subproject and investigator (PI) may have received primary funding from another NIH source, and thus could be represented in other CRISP entries. The institution listed is for the Center, which is not necessarily the institution for the investigator. Cellular expansion is an absolute necessity during the growth and development of plants and fungi. Expansion relies upon the accumulation of inorganic ions, the resulting water influx creates the hydrostatic pressure (turgor) that causes the cell to expand. Cellular expansion is normally asymmetric. Rather than expanding in all directions, there are localized regions of expansion that result in the well-defined final shape of the cell. The extreme example of anisomorphic cell expansion is tip growth. Here, the machinery of expansion is highly concentrated in a small region such that the cell exhibits tubular growth. How does the cell maintain the turgor that drives expansion? How is expansion controlled spatially? We are documenting the role of signal transduction pathways in turgor recovery in the model organism Neurospora crassa. We showed that an osmotic MAP kinase cascade activates ion transport after hyperosmotic treatment, causing rapid recovery of the normal high hydrostatic pressure (turgor) (Eukaryotic Cell 5:460-487, 2006). At the BioCurrents Research Center, I examined a novel signal transduction pathway, identified by osmosensitivity, that includes the CUT gene (a phosphatase). We demonstrated hyperosmotic-induced changes in the ion fluxes in the cut mutant: Ion uptake that was responsible for turgor recovery (glycerol accumulation, also mediated by the MAP kinase cascade, is absent in the cut mutant).

## **→** Funding Agency

**Agency** National Institute of Health (NIH)

**Institute** National Center for Research Resources (NCRR)

**Type** Biotechnology Resource Grants (P41)

Project # 5P41RR001395-27

Application # 7953830

**Project Start** 2008-12-01

Study Section Special Emphasis Panel (ZRG1-BPC-H (40))

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Project End 2009-11-30
Budget Start 2008-12-01
Budget End 2009-11-30
Support Year 27
Fiscal Year 2009
Total Cost $11,195
Indirect Cost

➤ Related projects

➤ Publications

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