Abstract 11-19

The sensitivity of *Saccharomyces* mutants to palmitoleic acid may provide a means to study the control of membrane fluidity in eukaryotes.

Daniel Lockshon (1), Emily O. Kerr (1), Robert Learmonth (2), Brian K. Kennedy (1)
(1) Department of Biochemistry, University of Washington, Box 357350, Seattle, Washington, 98195, USA; (2) Centre for Systems Biology, University of Southern Queensland, Toowoomba, 4350, Australia

The mechanisms which control the fluidity of eukaryotic membranes are unknown. We have identified *S. cerevisiae* deletion strains whose growth is impaired by palmitoleic (PO; C16:1) but not oleic (C18:1) acid. PO-sensitivity is suppressed by oleate thus perhaps identifying a signaling pathway that controls the ratio of these fatty acids in membrane phospholipid. Growth of these mutants is also inhibited by a known fluidizer, benzyl alcohol, thus indicating that PO has a fluidizing effect. Removal of Pkc1, known to play a key role in cell wall integrity control, leads to acute PO-sensitivity. Removal of Bck1, Mkk1, Mkk2, Slt2, or Swi6 downstream components of the cell wall integrity pathway, cause modest PO-sensitivity. Suppression by 1M sorbitol of the PO-sensitivity of these four mutants implies that PO/oleate ratio influences the cell wall. Acute PO-sensitivity of the *pkc1*Δ strain, even in the presence of 1M sorbitol, suggests the cell wall to be more severely compromised by PO addition to this strain. Alternatively, the failure to control the PO/oleate ratio could have an additional effect on the *pkc1* strain, perhaps by disabling a 2nd pathway downstream of Pkc1 thus allowing PO addition to cause excess membrane fluidity. We are attempting to distinguish these two models by a variety of genetic, biochemical, and physical methods. Most notably, the effect of PO on the fluidity of the plasma membrane is being examined by measuring the depolarization of laurdan fluorescence.