

## Abstract 03-02

### **OLE1 and HSP30 affect yeast membrane fluidity modulation during growth and in response to glucose.**

**Robert Learmonth (1)**, Brett Butcher (2)

(1) Centre for Systems Biology, University of Southern Queensland, West Street, Toowoomba, QLD, 4350, Australia; (2) Contract Manufacturing, Progen Industries, Darra, 4076 Australia

Adaptation of yeasts to changing environments during growth, fermentation and bioprocessing requires rapid modulation of membrane fluidity. We studied fluidity modulation in *S. cerevisiae* FY1679-28c and its mutants lacking lipid desaturase *ole1* or membrane-associated *hsp30*. We assessed relationships between fluidity and physiological parameters during aerobic and anaerobic batch culture, as well as membrane responses to glucose availability, determining progressive fluidity changes with fluorescent membrane probes. During batch growth the parent strain evidenced balanced relationships between membrane fluidity, phospholipid and fatty acyl composition, growth rate, glucose or ethanol consumption and viability. However the *ole1* and *hsp30* deletants appeared out of balance with less efficient metabolism (higher nutrient consumption), lower budding rates and lower viability. The *ole* defect was rectified by supplementation with C18:1 or C18:2. The *hsp30* defect had particular impact over the transition at glucose exhaustion. The parent had negligible membrane fluidity response to glucose while the *hsp30* and *ole1* deletants both showed marked changes in fluidity on glucose addition above (0.5% w/v) or below (0.1%) the catabolite repression threshold. Thus membrane responses in the deletants were different to the parent strain, indicating that their fluidity regulation mechanisms were unbalanced by ablation of the activities of either of these membrane-associated proteins.