

Cell growth and cell cycle control

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Our understanding of the control of cell division has recently advanced through the application of new technologies (e.g., sequencing, cryo-electron microscopy [cryoEM], microfluidics) and cross-pollination from different fields (e.g., signal processing and applied mathematics). The success of this approach is demonstrated by the fact that our session hosted not one, but two, ASCB award winners:

1. **Sophie Martin** (Université de Lausanne) received the Junior Women in Cell Biology Award for her work on cell size control in fission yeast. In our session, Sophie presented further work on the geometric model of cell size control, whereby a gradient in membrane-associated Pom1 kinase determines the critical length for fission yeast division. In this model, as cells grow, the tip-localized Pom1 is moved away from its targets at the middle of the cell to allow entry into mitosis. This intriguing and somewhat controversial model was also the subject of posters from the Mosely (Dartmouth University) and Chang (Columbia University) groups.

2. **Gabriel Lander** (Lawrence Berkeley National Laboratory) received the Merton Bernfield Award for best work by a graduate student or postdoctoral scholar. In Eva Nogales's lab, Gabriel used cryoEM to elucidate the mechanism through which the proteasome recognizes substrates targeted for degradation by ubiquitin chains. Gabriel's work suggests a model in which the proteasome lid repositions its ATPases to deliver substrates to the proteolytic chamber.

The decision whether to divide is of obvious importance. **Andreas Donic** has been investigating this cellular decision in budding yeast in my Stanford lab. In this session, Andreas reported that the coherent feed-forward regulation of the cyclin-dependent kinase inhibitor Far1 is crucial to allow pheromone arrest to be both robust and reversible without compromising the ability to measure the extracellular environment. This suggests a feed-forward alternative to positive feedback for the regulation of reversible cellular transitions.

The duplication and segregation of cellular components, such as the spindle pole body, is tightly controlled in the mitotic cell cycle. **Sue Jaspersen** (Stowers Institute) presented work suggesting that spindle pole bodies compete with the nuclear pore complex for a factor (Ndc1) required for insertion into the nuclear envelope. Also along these lines, **Manjunatha Shivaraju** from the Gerton lab (Stowers Institute) presented work showing that the yeast centromeric nucleosomes, which contain the histone H3 variant Cse4, are duplicated at anaphase.

As the number of genome sequences continues to increase exponentially, their impact on our understanding of the cell division cycle might be considered inevitable. Nevertheless, work in this area has been sparse. **Nick Buchler** (Duke University) ventured into this gap in our knowledge to report that the yeast G1 cyclins most likely derive from the metazoan cyclin B family, rather than from the cyclin D family, and that the primary metazoan cell cycle transcription factor E2F appears to have been replaced by heterodimeric transcription factor SBF in yeast. Taken together, Nick's work suggests the conservation of systems-level architecture of the G1 control network rather than the individual molecular components.

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