



CSHL Meetings & Courses
Abstract Submission



Dear Sebastien Leon,

Thank you for submitting your abstract for Yeast Cell Biology 2011 .

You have requested that your abstract be considered for:

Talk or Poster.

We will contact you as soon as the organizers have made their decisions. This usually takes about a month. A full list of talks and posters will also be available at our website:

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We look forward to your participation at the Yeast Cell Biology meeting.

Sincerely,
CSHL Meetings & Courses Program

A metabolic switch on a yeast arrestin connects glucose signaling to transporter endocytosis

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Endocytosis is a critical component of plasma membrane dynamics, by allowing the removal of proteins such as transporters or receptors in response to environmental cues.

In yeast, transporter endocytosis requires their ubiquitylation at the plasma membrane by the Nedd4-like E3 ubiquitin ligase, Rsp5. Since the ubiquitylation of a given transporter occurs only in response to specific signals, this raises the question of how substrate specificity is achieved, and how it is regulated dynamically. Various "adaptor" proteins were identified, which may promote the interaction between Rsp5 and its substrates, and may provide a basis for this regulation. However, how they modulate Rsp5 function in response to extracellular stimuli is unknown.

We addressed this question by studying a model transporter, Jen1, which is a lactate transporter induced in the presence of lactate and endocytosed in response to glucose (Paiva et al., JBC 2009). We identified an Rsp5 adaptor protein that belongs to the alpha-arrestin family, Art4 (also named Rod1), as essential for Jen1 ubiquitylation and endocytosis of in response to glucose.

Interestingly, when cells are grown in lactate medium to induce Jen1 expression, Art4 is strongly phosphorylated by the yeast AMPK homologue, Snf1. Addition of glucose, known to trigger Jen1 endocytosis, leads to a rapid dephosphorylation of Art4, a process that requires the PP1 phosphatase regulatory subunit Reg1. This dephosphorylation allows Art4 ubiquitylation by Rsp5, and we provide details on the molecular mechanism of this regulation. We also show that Art4 ubiquitylation is required for Jen1 endocytosis. Therefore, a switch in Art4 post-translational modifications occurs in response to glucose and is required to modulate its function as an adaptor of Rsp5. This establishes yeast arrestin-like proteins as key regulators of transporter endocytosis in response to extracellular signals.

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