

P008 TOR signalling regulates mitotic commitment through the stress MAPK and Polo kinase in response to nutrient availability

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The coupling of growth to cell cycle progression allows eukaryotic cells to divide at particular sizes depending on nutrient availability. In fission yeast this coupling involves the Spc1/Sty1 MAP Kinase pathway working through Polo kinase recruitment to the Spindle Pole Bodies (SPBs) (Petersen and Hagen 2005). A change in nutrient quality or rapamycin induced inhibition of TOR signalling advanced mitotic onset, reducing cell size at division (Petersen and Nurse 2007). This inhibition of Tor1 stimulates Spc1/Sty1 activity through regulation of the Pyp2 MAPK phosphatase. The increase in Spc1/Sty1 activity promotes Polo kinase SPB recruitment to advance mitotic onset. This advanced mitotic onset is abolished in cells depleted of Pyp2, Spc1/Sty1 or upon blockage of Spc1/Sty1 dependent Polo SPB recruitment. Therefore mitosis is advanced to reduce cell size at division when cells experience a reduced nitrogen quality. However, following an acute removal of the amino acid leucine from wild type fission yeast *leu⁺* cells, mitosis is transiently blocked. Thus, changes in basal MAPK activity in response to changes in nutrient quality, advances mitotic commitment, but the acute stress following complete amino acid withdrawal transiently blocks mitosis.