

**P018** p38 positively regulates growth and Tor signalling  
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To identify novel components of the Tor signalling network, an RNAi screen was performed in *Drosophila* cells. This screen identified the p38 pathway as a regulator of Tsc2-mediated changes in cell size. In both *Drosophila* and mammalian tissue culture models, inhibition of p38 signalling using RNAi prevents the phosphorylation of S6 and 4EBP1 in response to amino acids, and also decreases Tsc2 RNAi-mediated increases in cell size. Consistent with a model in which p38 activates Tor, the activation of p38 using H<sub>2</sub>O<sub>2</sub> or anisomycin increases the phosphorylation of the downstream Tor targets S6/S6K and 4EBP1. Furthermore, long-term activation of the p38 pathway increases cell size. To investigate the role of p38 in the activation of Tor *in vivo*, we generated *Drosophila* mutants null for *p38b* and the upstream kinase *mkk3/licorne*. Interestingly, mutants for either *licorne* or *p38b* are small. This decrease in organism size seems to be due at least in part to a decrease in cell size. Mutants for *licorne* or *p38b* are nutrition-sensitive; low-nutrient food accentuates the small organism phenotypes as well as the partial lethality of the *p38b* null allele.

Our data suggest that p38 can activate Tor. This may be important during processes such as the activation of the immune system or during angiogenesis, where increased translation in response to stress would be key to mounting an appropriate response.