Technion Ubiquitin Studies Section

Sunday Jan 3rd 2016 @ 16:00
Emerson #417 Conference Room

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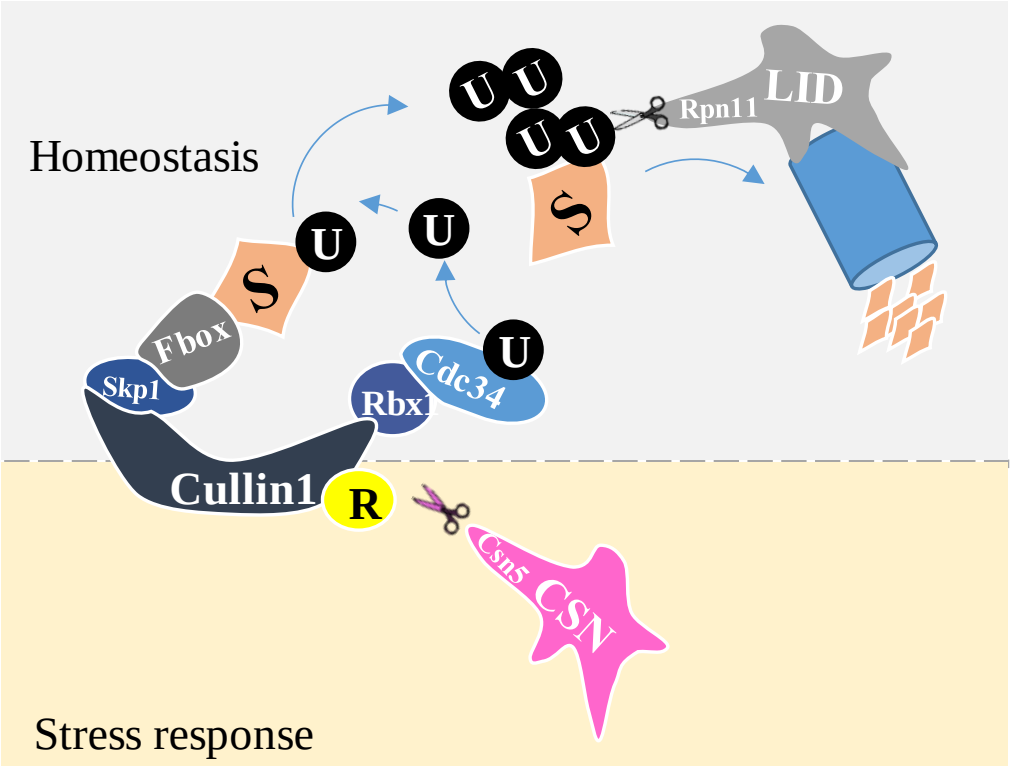
The proteasome subunit Rpn11 is required for a proper deRubylation of cullins



The proteasome lid and the COP9 signalosome (CSN) diverged from a common ancestor yet fulfill distinctive roles within the ubiquitin-proteasome sphere. Each complex harbors a metalloprotease catalytic subunit, Rpn11 in the lid and Csn5 in the CSN, responsible for the lid-mediated hydrolysis of ubiquitin from protein substrates and for the removal by the CSN of the ubiquitin-like protein NEDD8/RUB1 from the Cullin scaffold subunit of CRLs. Loss of function of either Csn5 or Rpn11 results in phenotypes attributed to the respective complex. Surprisingly, Sus discovered that Rpn11 mutants exhibit a CSN-related phenotype of accumulation of Rubylated Cul1. In her talk, Susu will discuss the reason(s) for this phenotype.