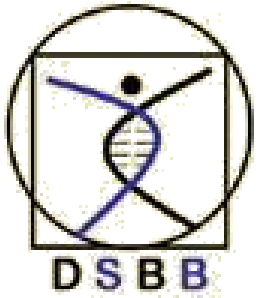
Ddc1 and Dpb11 recruit Rad9

independently of histone H3 methylation

Fabio Puddu, Gargnano 2007



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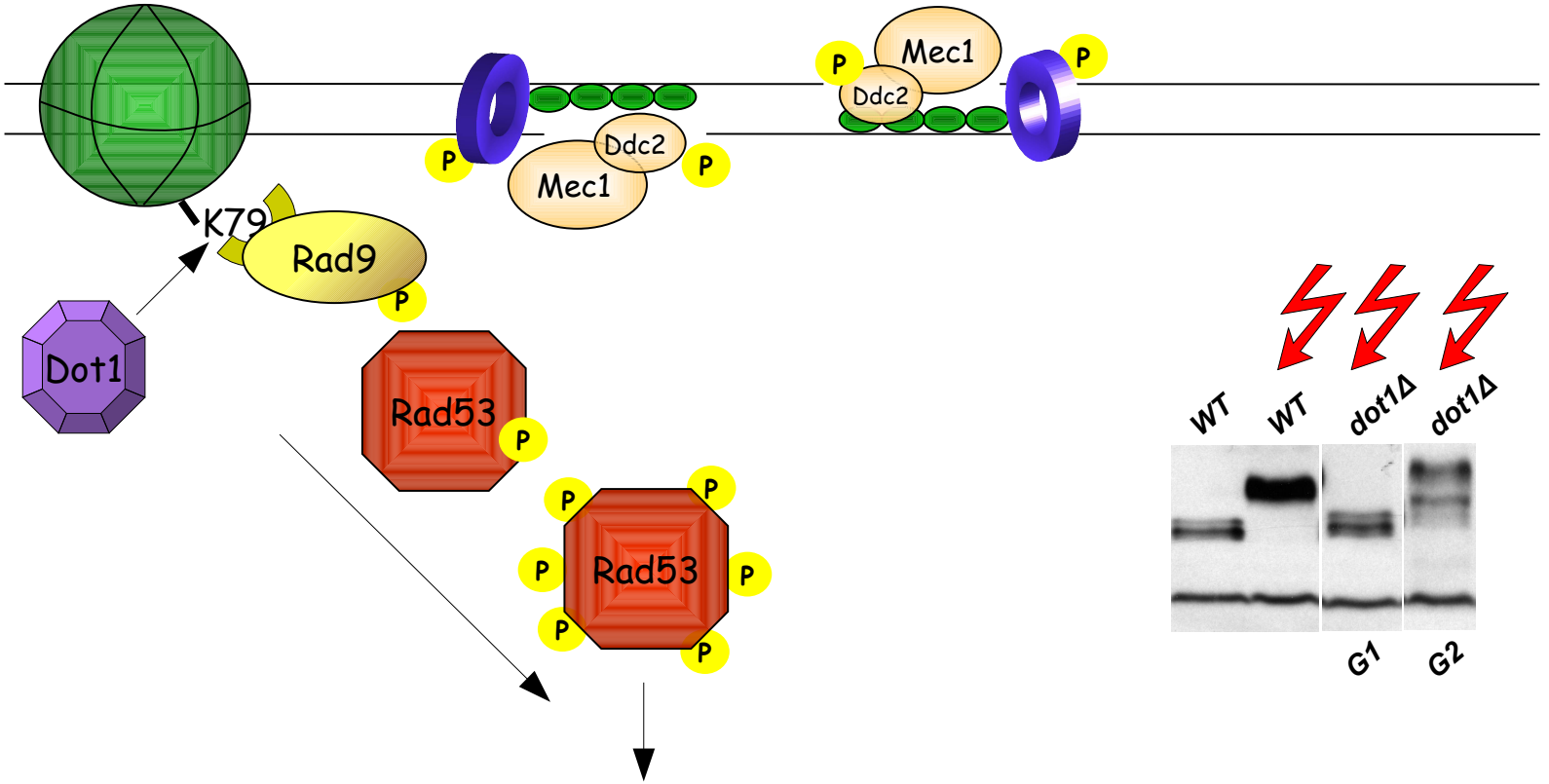
DNA damage checkpoint



Responses to DNA damage

Cell cycle

DNA repair

Transcription

...

RPA

PCNA-like

Question

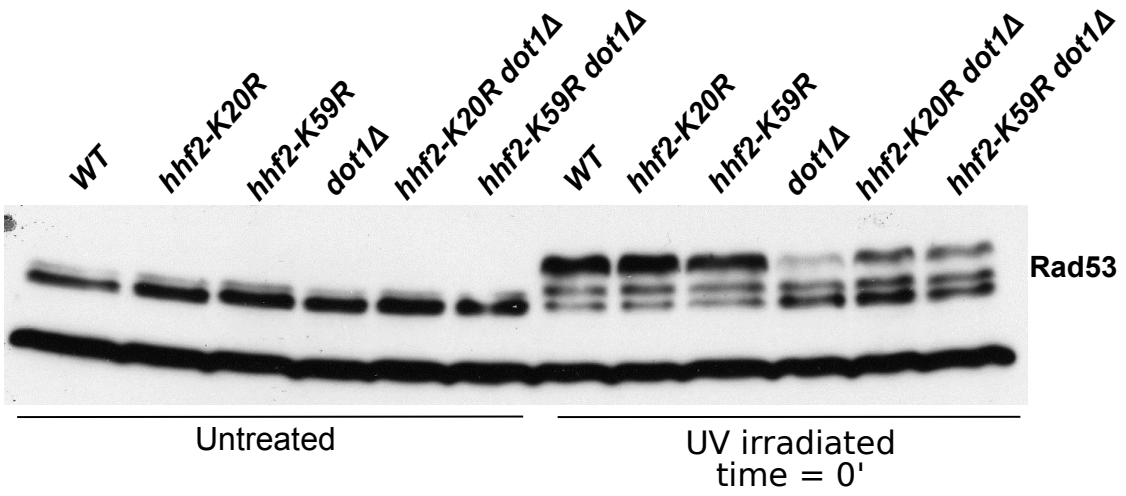
In G2 exists an alternative Rad9 recruitment pathway

What are the factors this alternative pathway depends on?

We tried with different histone residues...

How is Rad9 recruited in absence of histone H3 methylation?

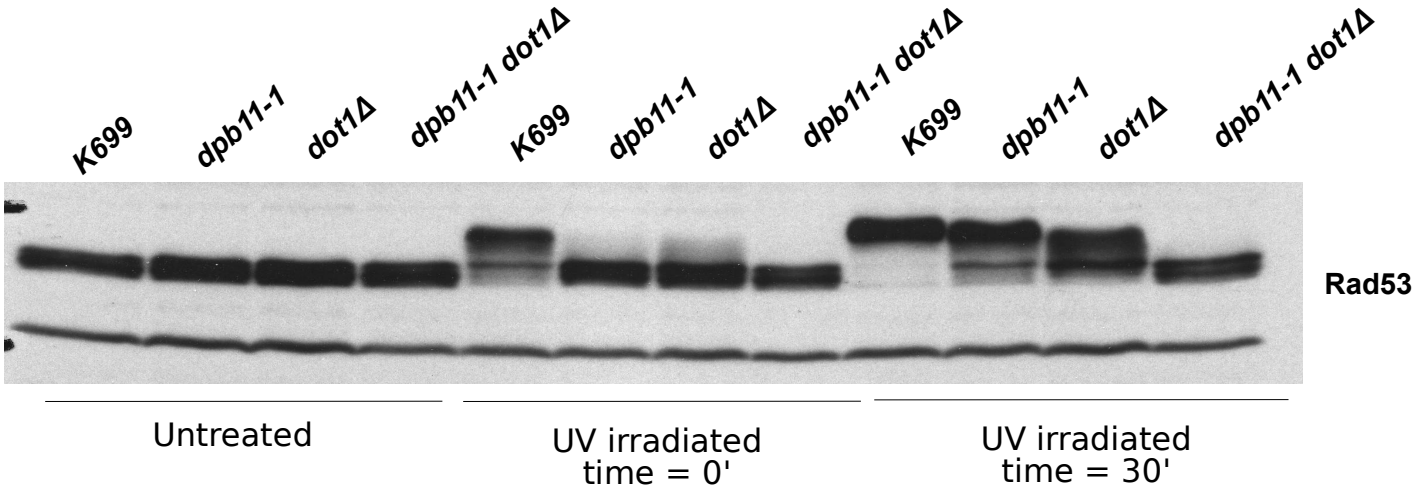
noco → noco → protein extracts



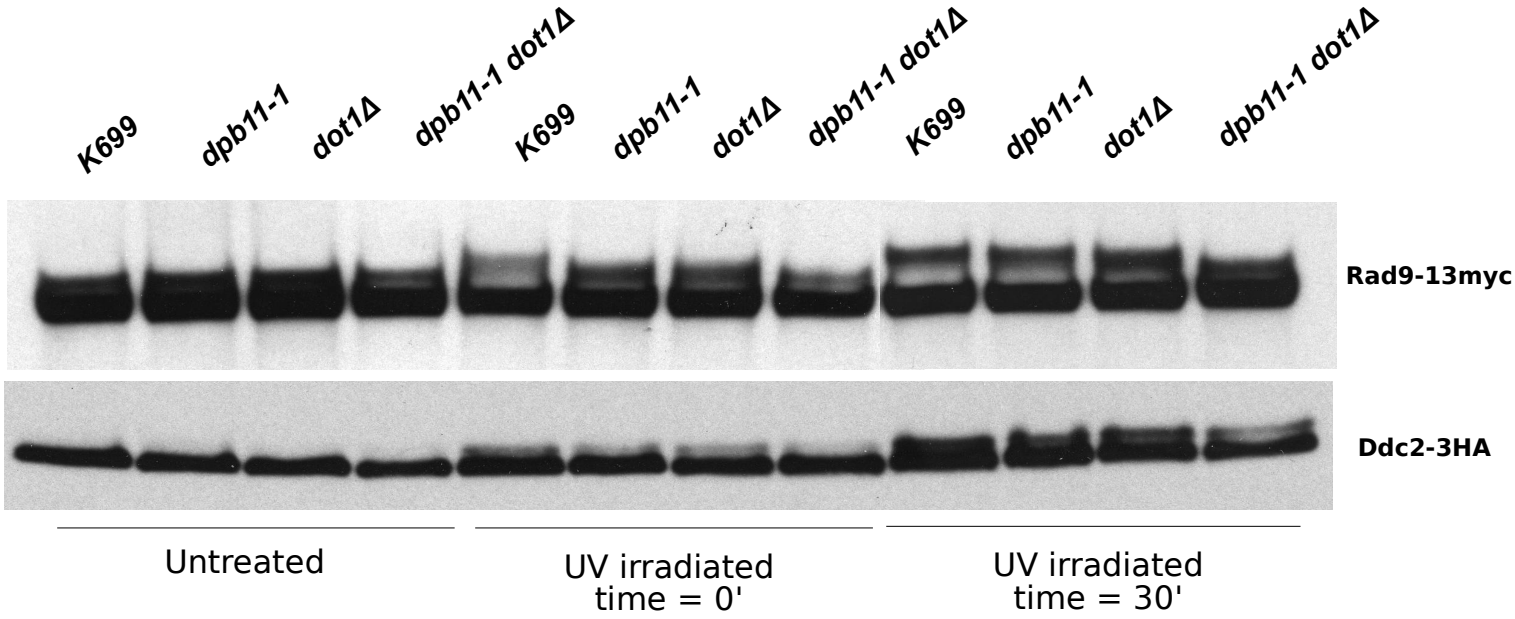
We looked for something else...

How is Rad9 recruited in absence of histone H3 methylation?

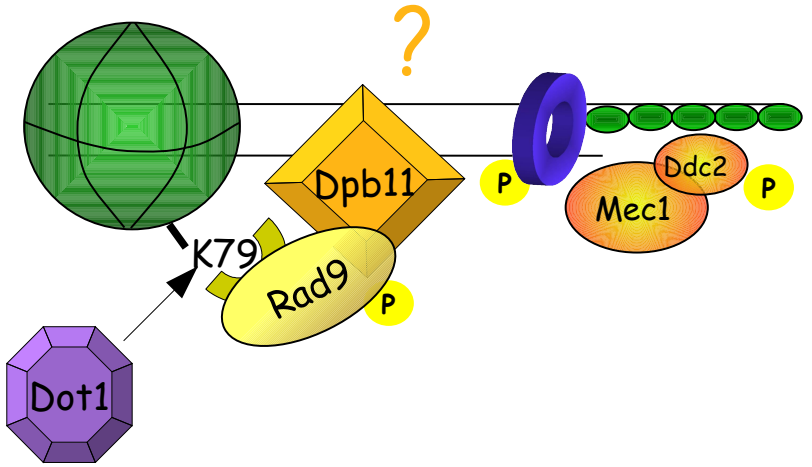
noco → noco → protein extracts



dpb11-1dot1Δ is defective in Rad9 phosphorylation



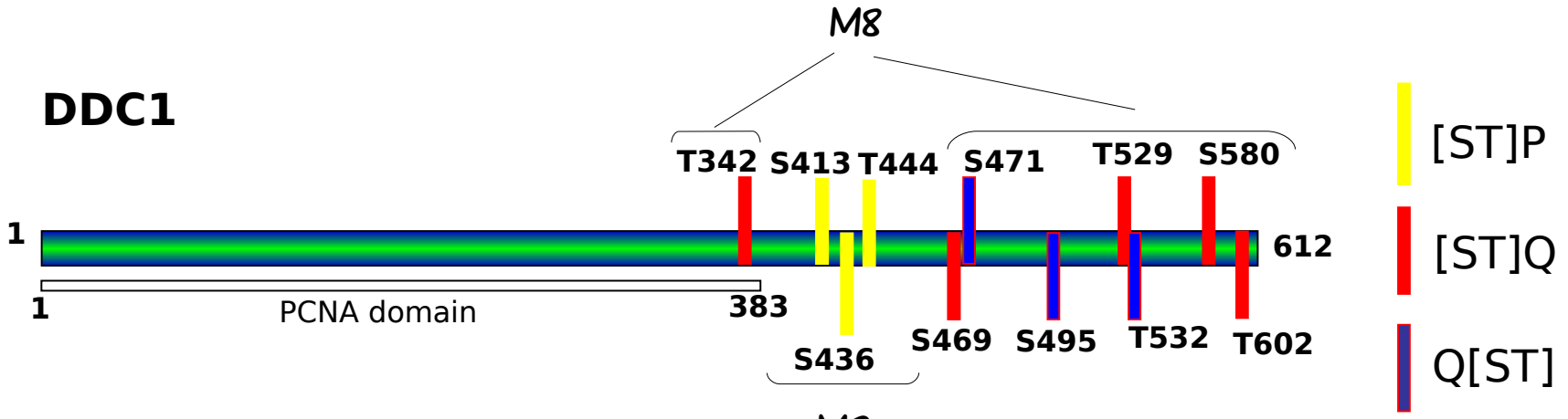
How is Dpb11 recruited to the damage site?



DPB11 and DDC1 interacts by TH

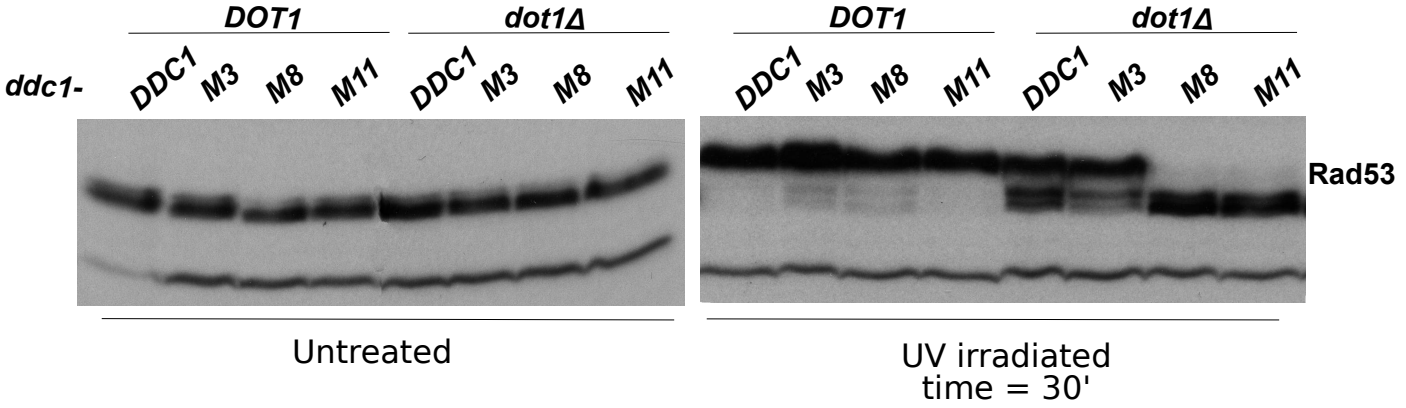
dpb11-1 mutant abolish this interaction

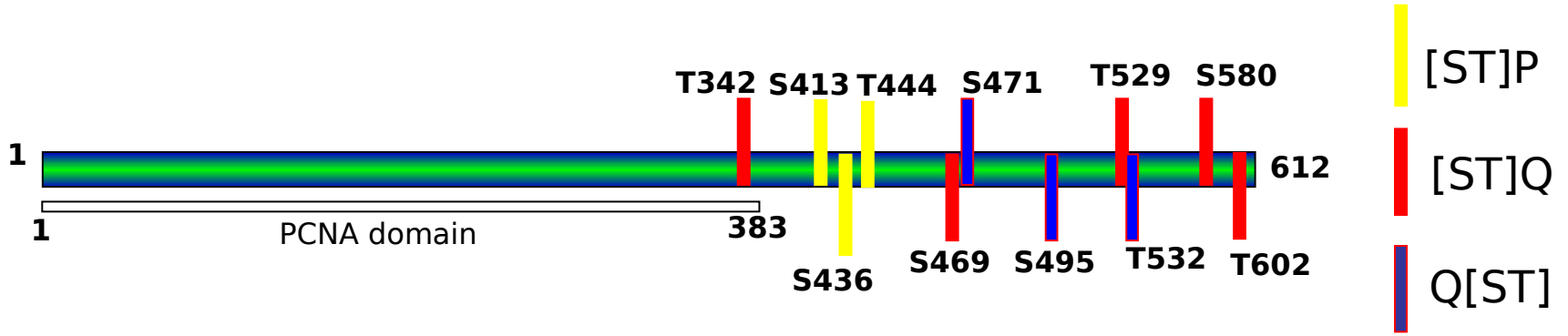
(Wang & Elledge, 2002)



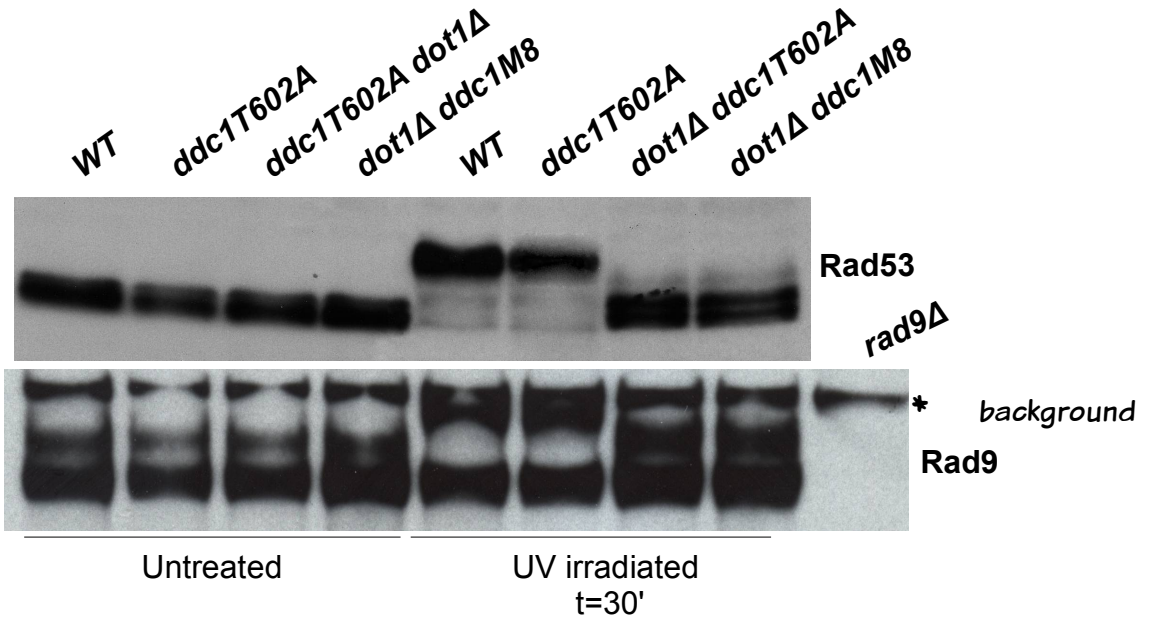
M3+M8 = M11

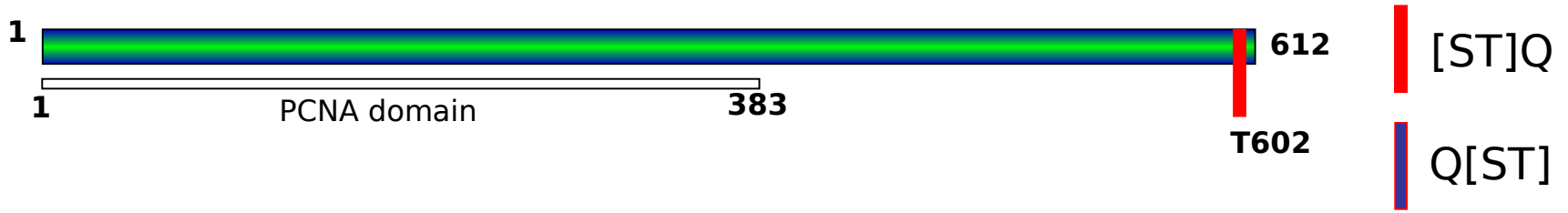
dot1Δddc1-M8 mutant mimics the *dot1Δdpb11-1* mutant



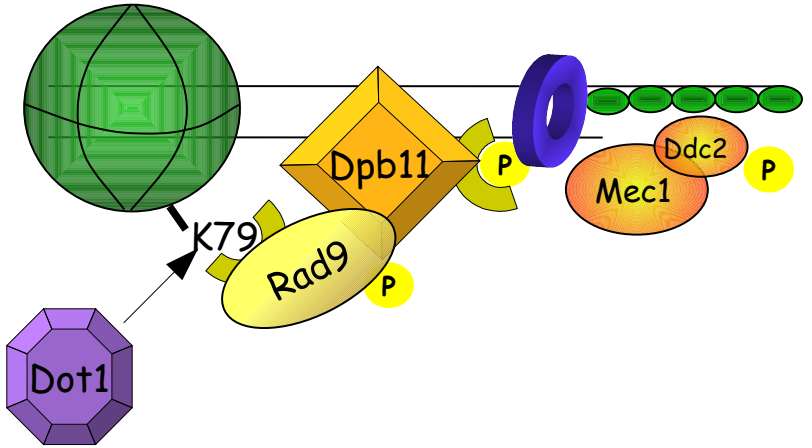


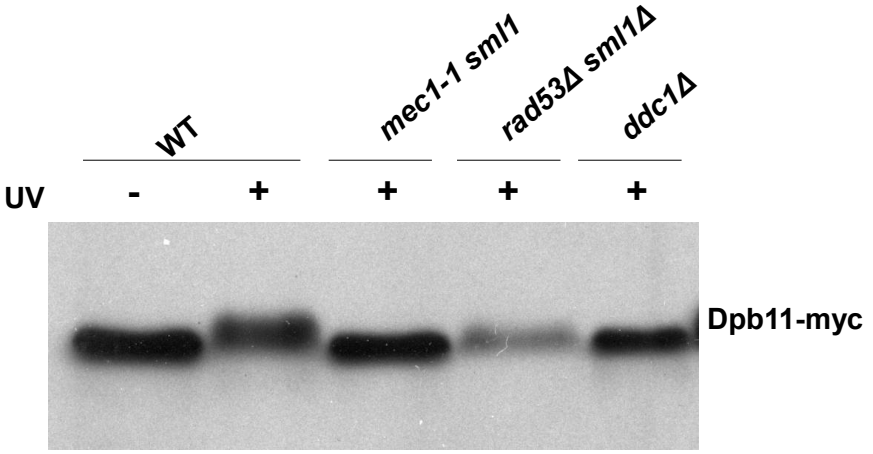
Threonine 602 is required for Rad9 recruitment in absence of Dot1

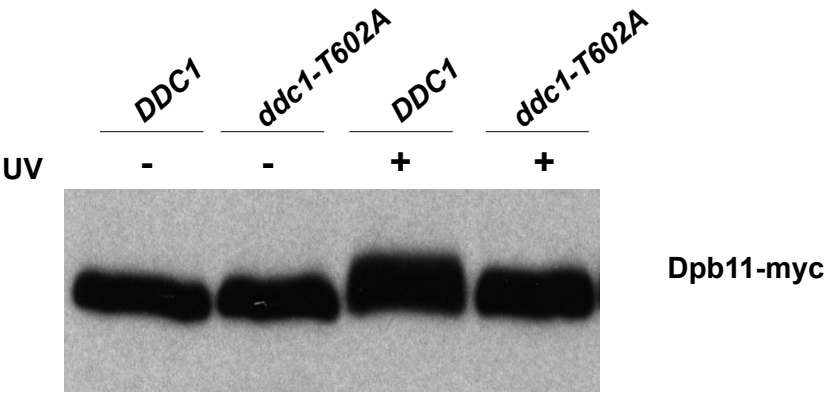




Dpb11 role in checkpoint signaling







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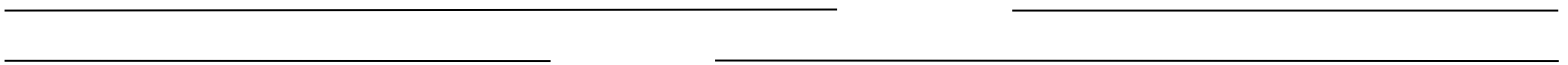
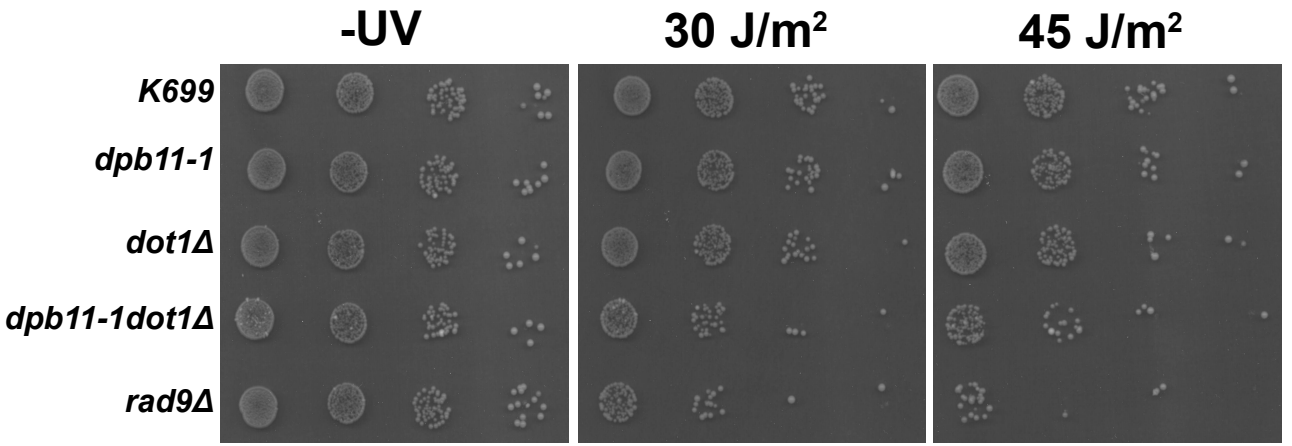
Dna damage checkpoint



Responses to DNA damage

dpb11-1dot1Δ is defective in Rad9 recruitment



Dpb11 is phosphorylated after UV treatment