

**P039** How does RNA Polymerase I relay DNA damage-induced apoptosis in *C. elegans*?

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Eukaryotic organisms preserve their genome integrity by exhibiting a properly regulated response when DNA is damaged. Failure of DNA lesions to be restored can lead to mutations or large-scale genomic instability, which may implicate tumorigenic potential. We are using the germline tissue of the nematode *C. elegans* as a model to dissect the signalling network that responds to damage inflicted by chemical or physical agents. In screening experiments, various mutant worm strains have been selected which are defective for proper cell cycle arrest and induction of apoptosis upon treatment with UV-light or ionizing radiation. Damage response mechanisms are highly conserved among metazoans, which allows to extrapolate many of our observations and working models to higher organisms including humans.

Based on the findings in one mutant, RNA polymerase I seems to be involved in the regulation of DNA-damage induced apoptosis. It is the topic of our current research to elucidate whether the defective response we observe in this mutant is due to a specific role of the affected RNA Pol I subunit in apoptosis, or whether it results from a more general implication on transcription of ribosomal RNA and on synthesis of ribosomes.