

**P008** Sumoylation of the RNA helicase p68 and its role in transcriptional regulation

**A-MF Jacobs, SM Nicol, EG Jaffray<sup>1</sup>, RT Hay<sup>1</sup>  
and FV Fuller-Pace**

*Division of Pathology & Neuroscience, Ninewells Medical School; <sup>1</sup>Division of Gene Regulation and Expression, School of Life Sciences; University of Dundee, Dundee, UK*

The nuclear DEAD box RNA helicase p68 can act both as a transcriptional co-activator and, in some contexts, as a promoter-specific transcriptional repressor.

We have shown that p68 is preferentially modified by SUMO-2. We identified a single lysine (K53) as the sumoylation site on p68 and showed that the SUMO E3 ligase PIAS-1 interacts with and significantly enhances sumoylation of p68 in cells.

There is growing evidence that sumoylation plays an important role in transcriptional regulation and often leads to attenuation of transcriptional activation. Given that p68 can act both as a transcriptional co-activator and repressor, we examined whether sumoylation affects p68 transcriptional regulatory function. Our data show that SUMO modification enhances p68 transcriptional repression activity and inhibits the ability of p68 to function as a co-activator. These findings may be explained by the ability of wild type, but not a sumoylation-deficient mutant p68, to alter the modification state of chromatin through recruitment of HDAC1. We are currently investigating whether p68 sumoylation is regulated by different cellular stresses/growth conditions or by other post-translational modifications of p68.