# Lack of Effect of RuvB-Like Proteins on DNA Damage Signaling Activation

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Ataxia telangiectasia mutated (ATM) kinase is a central player in cellular response to DNA damage. Phosphorylation of the histone H2AX by ATM is required for the accumulation of repair proteins at the sites of double-strand breaks. Recently, it was reported that the histone acetyltransferase Tat interactive protein-60 (TIP60) is required to acetylate ATM prior to its activation. The RuvB-like proteins TIP48 and TIP49 are known to be necessary for the assembly and functional activity of the TIP60 acetyltransferase complex. In the present communication, we investigated the requirements of TIP48 and TIP49 for ATM activation by monitoring the cell cycle distribution and H2AX phosphorylation after irradiation of TIP48- and TIP49-depleted cells. We found that neither the cell cycle nor ɔ-H2AX were affected in TIP48- and TIP49-silenced cells, suggesting that the TIP60 chromatin modification complex is not engaged in DNA damage signaling upstream of ATM.

*Key words:* RuvB-Like Proteins, ATM, DNA Damage Signaling, Ionizing Radiation