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PHOSPHORYLATION OF p54^{NRB} DURING MITOSIS

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p54^{nrb} is a nuclear factor that regulates many processes including, transcription, transcription-splicing coupling or DNA repair. Using 2D-PAGE followed by EIS-QTOF-MS and "in vitro" dephosphorylation assays we found that p54^{nrb} is phosphorylated after treatment with mitotic damaging agents that cause M phase arrest. These agents include drugs such as vincristine or paclitaxel used for treatment of many tumor types and anticancer drugs such as kinesin spindle protein (KSP) inhibitors that are currently in clinical trials. These drugs also cause cell death and induce the processing of this nuclear factor by caspases. Furthermore, we use cell cycle regulators to determine that p54^{nrb} phosphorylation is dependent on the mitotic state induced by these antitumoral drugs. Finally, using double thymidine block synchronized cells; we demonstrate that p54^{nrb} is also phosphorylated during normal mitosis.