The lncRNA PANDAR is transcribed from the CDKN1A promoter in a p53-dependent manner [1]. Overexpression of lncRNA PANDAR has been observed in some tumor species, while the overexpression was correlated with an invasive phenotype and a reduced patient survival rate [2,3]. In this thesis it has been shown that endogenous and DNA damage-induced PANDAR RNA expression varies depending on the cell line. Furthermore, the knockdown of PANDAR RNA within U2OS osteosarcoma cells revealed a reduced cell viability, indicating that PANDAR plays an important role for cell sustainability.

One mechanism for this regulation process is the decoy function of lncRNA PANDAR, sequestering the transcription factor subunit NF-YA of its pro-apoptotic target genes [1]. The analysis of direct binding of lncRNA PANDAR with purified NF-Y fragments revealed an interaction of PANDAR RNA with the NF-YA subunit and the NF-Y trimeric complex, pointing towards a binding of PANDAR to NF-YA in region that is not involved in subunits association. The analysis of RNA binding motif of NF-YA by PAR-CLIP identified the genomic annotated sequence C(T/A)G(A/T) as a possible RNA-binding motif. However, the following mutational studies revealed that NF-YA binds to wildtype and mutated PANDAR RNA fragments in a sequence-independent manner. It is possible that the sequence specificity of the NF-YA PAR-CLIP analyzed RNA binding motive could be mediated by other regulatory mechanisms like RNA secondary structure, posttranslational protein modifications or additional involved NF-YA domains of the full length protein.

For identifying novel PANDAR binding proteins, PNAs directed against PANDAR RNA were synthesized, tested and used for a PNA-based SILAC experiment. Further validation steps of mass spectrometrically analyzed data revealed an interaction of PANDAR RNA with the splicing-involved proteins SAFA, hnRNPUL1, U2AF65 and PTBP1. Furthermore, the effect of lncRNA PANDAR concerning the transcript variant BCL-XS was analyzed, with PTBP1 being involved in the initiation of BCL-XS alternative splicing [4]. The overexpression of lncRNA PANDAR reduced the mRNA level of pro-apoptotic BCL-XS, while the simultaneous overexpression of PTBP1 was able to rescue this effect by normalizing BCL-XS mRNA to the initial level.

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