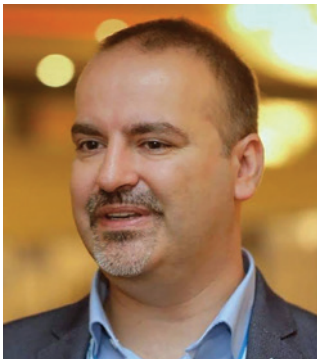
TIBOR PANKOTAI



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RESEARCH AREA

Faithful repair of DNA double-strand breaks (DSBs) is indispensable since improper repair can lead to genome instability and subsequently to tumorigenesis. DSBs can be repaired through different pathways, and the balance between the choice of them must be tightly regulated to preserve genome integrity. DNA damage can be considered as a harmful stressor in which various biochemical pathways are activated, ensuring the proper DNA repair and cell survival. The main focus of the project is to map the signalling circuit induced by DNA damage and to understand how the malfunctional DNA repair can initiate tumorigenesis. In our experimental setup, we will use state-of-the-art biochemical technologies and genomic mapping in a human cell culture model system, and we combine these with single-cell data using super-resolution STORM microscopy. Additionally, with our experimental data, we can verify the existence and the means of DNA damage-induced cell signalization circuits and reveal the potential mechanisms of cellular communication. Although as a primary goal, the project aimed to unveil a basic research clue, identifying key steps in the repair process can help us to recognize new anti-cancer therapeutic targets, thereby also contributing to the development of novel drugs in tumor therapy.

TECHNIQUES AVAILABLE IN THE LAB

Nucleic acid-based techniques: nucleic acid isolation, mutations and gene expression measurements by PCR and quantitative PCR technics. Genomic applications: short read-based Illumina sequencing and bioinformatics data processing. Protein detection: Western blot, immunoprecipitation, chromatin immunoprecipitation. Microscopy: Immunohistochemistry, confocal and super-resolution STORM microscopy. Test models: Frozen and paraffin-embedded sections of mammalian in vitro cell cultures, tumor-derived primary cell cultures.

SELECTED PUBLICATIONS

Khanam T, Muñoz I, Weiland F, Carroll T, Morgan M, Borsos BN, Pantazi V, Slean M, Novak M, Toth R, Appleton P, **Pankotai T**, Zhou H, Rouse J. (2021) CDKL5 kinase controls transcription-coupled responses to DNA damage. **EMBO J 4:** e108271.

Majoros H, Borsos BN, Ujfaludi Z, Páhi ZG, Mórocz M, Haracska L, Boros IM, **Pankotai T.** (2021) SerpinB10, a Serine Protease Inhibitor, Is Implicated in UV-Induced Cellular Response. Int **J Mol Sci 22:** 8500. DOI: 10.3390/ijms22168500.

Varga D, Majoros H, Ujfaludi Z, Erdélyi M, **Pankotai T.** (2019) Quantification of DNA damage induced repair focus formation via super-resolution dSTORM localization microscopy. **Nanoscale 11:** 14226-14236.

Caron P, **Pankotai T,** Wiegant WW, Tollenaere MAX, Furst A, Bonhomme C, Helfricht A, de Groot A, Pastink A, Vertegaal ACO, Luijsterburg MS, Soutoglou E, van Attikum H. (2019) WWP2 ubiquitylates RNA polymerase II for DNA-PK-dependent transcription arrest and repair at DNA breaks. **Genes Dev 33:** 684-704.

Caron P, **Pankotai T,** Wiegant WW, Tollenaere MAX, Furst A, Bonhomme C, Helfricht A, de Groot A, Pastink A, Vertegaal ACO, Luijsterburg MS, Soutoglou E, van Attikum H. (2019) WWP2 ubiquitylates RNA polymerase II for DNA-PK-dependent transcription arrest and repair at DNA breaks. **Genes Dev 33:** 684-704.

Borsos BN, Huliak I, Majoros H, Ujfaludi Z, Gyenis A, Pukler P, Boros IM, **Pankotai T** (2017) Human p53 interacts with the elongating RNAPII complex and is required for the release of actinomycin D induced transcription blockage. **SCIENTIFIC REPORTS 7:** 40960