Large scale genomic-wide studies revealed that only about 1–2% of human genome codes for proteins, and approximately 90% of genome is transcribed into RNA, including two main regulatory types of non-coding RNAs: microRNAs (miRNAs) and long non-coding RNAs (lncRNAs). miRNAs are small RNA molecules of approximately 21-25 nucleotides that possess the ability to downregulate gene expression by binding to the 3' untranslated region (3'UTR) of target mRNAs and hence degrading or blocking the translation of these transcripts. miRNAs can modulate a substantial fraction of human transcriptome. miRNAs are linked to the regulation of many cellular processes, such as proliferation, differentiation, senescence, survival, autophagy and migration.

lncRNAs are more heterogenic RNA molecules and are usually 200–1000 nucleotides long. Their genes are characterized by the same histone modifications as protein-coding genes and are transcribed by RNA polymerase II from independent promoters. lncRNA transcripts are usually 5'-capped and 3′-polyadenylated, often spliced with similar exon/intron lengths as mRNAs. Despite low overall sequence similarity, lncRNAs possess evolutionarily-conserved roles and secondary structure important for their functions. Depending on cellular localization, lncRNAs may control and modulate crucial signaling pathways in human cells: nuclear lncRNAs are mostly involved in chromatin modifications, transcriptional regulation and RNA processing, while cytoplasmic lncRNAs modulate mRNA stability and translation, and control various cellular signaling cascades. Moreover, lncRNAs can directly interact with proteins influencing their stability, activity and localization, act as sponges for miRNAs reducing their regulatory effect on targeted mRNAs, or even serve as precursors of miRNAs or circular RNAs.

Aberrant expression or activity of both types of non-coding RNA might impact the development of many diseases including cancer. Moreover, miRNAs as well as lncRNAs are also secreted from normal and cancer cells in exosomes, spherical membrane nanovesicles, which are a key element of intercellular communication in the body. The high stability of miRNAs and lncRNAs circulating in the bloodstream allows their use in non-invasive laboratory diagnostics as cancer biomarkers.

My scientific interest are mostly focused on the involvement of specific non-coding RNA in melanoma progression and the development of drug resistance. More specific information regarding miRNAs in melanoma can be found in my following publications:

Wozniak M., Peczek L., Czernek L., Düchler M. Analysis of the miRNA Profiles of Melanoma Exosomes Derived Under Normoxic and Hypoxic Culture Conditions. *Anticancer Res.*, **2017**, 37, 6779-6789.

Wozniak M., Mielczarek A., Czyz M. miRNAs in Melanoma: Tumor Suppressors and Oncogenes with Prognostic Potential. *Curr Med Chem.*, **2016**, 23, 3136-3153.

Wozniak M., Sztiller-Sikorska M., Czyz M. Diminution of miR-340-5p levels is responsible for increased expression of ABCB5 in melanoma cells under oxygen-deprived conditions. *Exp Mol Pathol.*, **2015**, 99, 707-716.

Wozniak M., Sztiller-Sikorska M., Czyz M. Expression of miRNAs as Important Element of Melanoma Cell Plasticity in Response to Microenvironmental Stimuli. *Anticancer Res.*, **2015**, 35, 2747-2758.

The involvement of lncRNAs in melanoma progression and drug resistance can be found in my recent review papers:

Wozniak M., Czyz M. lncRNAs-EZH2 interaction as promising therapeutic target in cutaneous melanoma. *Front Mol Biosci.*, **2023** 10:1170026.

Wozniak M., Czyz M. The Functional Role of Long Non-Coding RNAs in Melanoma. *Cancers (Basel)*, **2021**, 13,4848.

Between 2016 and 2019 I was a postdoctoral fellow at the Wright State University, Boonshoft School of Medicine, Dayton, Ohio, USA, where I worked in NIH-founded project regarding DNA damage repair and chromosomal instability using fission yeast *Schizosaccharomyces pombe* as a model eukaryotic organism. This scientific cooperation resulted in two published papers:

Zhang L., Geng, X.R., Wang F.F., Tang J.S., Ichida Y., Arishya S., Jin S., Chen M.Y., Tang M.L., Pozo F.M., Wang W.X., Wang J., Wozniak M., Guo X.X., Miyagi M., Jin F.L., Xu Y.J., Yao X.S., Zhang Y.W. 53BP1 regulates heterochromatin through liquid phase separation. *Nat Commun.*, **2022**, 13, 360, 1-16.

Xu Y.J, Khan S., Didier A.C., Wozniak M., Liu Y., Singh A., Nakamura T.M. A tel2 Mutation That Destabilizes the Tel2-Tti1-Tti2 Complex Eliminates Rad3/ATR Kinase Signaling in the DNA Replication Checkpoint and Leads to Telomere Shortening in Fission Yeast. *Mol Cell Biol.*, **2019**, 39, e00175-19.