



Tomasz Śliwiński

PROFESSOR – UNIVERSITY OF LODZ · HEAD OF BIOCHEMISTRY INSTITUTE – UNIVERSITY OF LODZ

✉ tomasz.sliwinski@biol.uni.lodz.pl | 🏠 www.uni.lodz.pl/pracownicy/tomasz-sliwinski | 🆔 0000-0001-8385-7744 |

Scopus bibliometric data: citations 3502 · documents 152 · h-index 32

Currently held positions

Department of Molecular Genetics, Faculty of Biology and Environmental Protection

University of Lodz

PROFESSOR

Faculty of Biology and Environmental Protection

University of Lodz

HEAD OF BIOCHEMISTRY INSTITUTE

Scientific profile and collaborations

My scientific research focuses on molecular genetics. The most important scientific of my achievement is using the phenomenon of "synthetic lethality" to eliminate cancer cells based on DNA double crack repair systems. During my stay on a scholarship in the USA conducted research initiated and conducted by the team of prof. Tomasz Skorski from Temple University, using chronic myeloid leukemia cells. We used inactivation of the RAD52 protein, either by introducing a mutation or by using the F79 aptamer to block the domain of this DNA binding protein. Importantly, DNA repair utilizing the homologous recombination mechanism (HRR) is usually dependent on the pathway associated with the interaction of BRCA1 / BRCA2 and RAD51 proteins or its paralogs promoting single stranded DNA invasion and strand pairing. The mechanism based on the interaction of RAD51-RAD52 proteins is a replacement mechanism. Based on the disruption of the HRR system, a phenomenon called "synthetic lethality" (SL) was induced in cancer cells, which consists in the dependence of cancer cells on the single-stranded DNA repair pathway, whereas normal cells have 2 or more repair mechanisms. It was very important in my work to block the DNA-binding RAD52 domain, which induced SL in BRCA1- or BRCA2-deficient leukemia cells without affecting normal cells. In addition, RAD52-F79 interaction has been shown to disrupt RAD52-DNA binding, which leads to the accumulation of toxic double-stranded DNA breaks only in cancer cells, not in normal counterparts. Then, based on the knowledge gained in the USA, me, together with a my team, performed extensive research in Poland using cells derived from solid tumors from people with skin cancer, where the PARP1 protein inhibitor was used to induce SL. In these studies, for the first time in the world, my team demonstrated the effectiveness of SL induction in malignant melanoma cells deficient in ligase 4 by the use of the Olaparib inhibitor. The results of this unique study show the possibility of inducing SL via the pathway (D-NHEJ) based on the PK-cs DNA protein, which ligase 4 plays one of the key roles. The obtained results are not only important from the cognitive point of view, but also create the opportunity to develop new strategies for cancer therapy. The similar mechanism was then confirmed by our team in another type of solid tumor, glioma.

Selected publications

- 2023 **Simultaneous Targeting of DNA Polymerase Theta and PARP1 or RAD52 Triggers Dual Synthetic Lethality in Homologous Recombination-Deficient Leukemia Cells** [\[link\]](#)
- 2023 **DNA polymerase theta protects leukemia cells from metabolic-induced DNA damage** [\[link\]](#)
- 2013 **Personalized synthetic lethality induced by targeting RAD52 in leukemias identified by gene mutation and expression profile** [\[link\]](#)

Research grants

Principal Investigator: 6 grants: NCN

Co-Investigator: 20 grants: KBN, NCN, MNiSW, NCBiR, The European Commission's HORIZON 2020 project

International research stays

USA Philadelphia, Temple University, Department of Cancer and Cellular Biology, professor Tomasz Skorski