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Pharmacological activation of p53 protein family affects proliferation and migration of cancer cells with *TP*53 gene mutations

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In the majority of all human cancer cases, the cancer suppressor protein p53 is lost or mutated. p53 belongs to a family of proteins that includes p73 and p63. The latter two can be categorized into two main groups: TA isoforms, which, like p53, act as tumor suppressors; and ΔN isoforms, which act like oncogenes and affect the functions of p53, TAp63, and TAp73.

*TP*53 mutations lead to gain of function, which promotes more metastatic and fatal disease that are extremely difficult to cure. As reconstitution of p53 suppresses established tumors *in vivo*, current targeted therapies focus on the activation of p53 but this strategy is still difficult to implement therapeutically. A promising strategy to overcome p53 deficiency and restore p53 pathway functioning is to manipulate the p53 family members, TAp63 and TAp73.

In our on-going efforts we identified small moleculesthat restore the p53 pathway and inhibit proliferation of cancer cells in tumors with various *TP53*gene mutations. TAp73 has been identified as a factor that engages pro-apoptotic signaling and inhibits migration of metastatic cancer cells.

We propose a hypothesis that targeting interactions of TAp73 with its inhibitors with simultaneous inhibition of synthetic sick factors to those inhibitors can provide a therapeutic advantage over currently developed strategies to target tumors with *TP*53 loss or mutations.

Biography:

Dr Joanna Zawacka-Pankau is a Principle Investigatorat KarolinskaInstittuet. She has done her Ph.D. at University of Gdansk, Poland under supervision of Prof. Anna J. Podhajska. Her research focused on activation of p53 tumor suppressor by small molecules and photodynamic therapy in colon cancer. In 2005 she joined Prof. Galina Selivanova Lab from the Department of Microbiology, Tumor and Cell biology, KarolinskaInstitutet as a postdoctoral fellow to study the mechanism and outcome of activation of wild-type p53 by small molecules. Then, she was appointed Assistant Professor at the Department of Biotechnology, Intercollegiate Faculty of Biotechnology, Gdansk, Poland where she focused on exploring the mechanism of tumor suppression upon pharmacological activation of p53 family members in p53 deficient and mutant tumors.

In 2012 she took up a position of Assistant Professor at the Department of Microbiology, Tumor and Cell Biology and in 2016 she joined the Department of Clinical Science, Intervention and Technology.