UNIVERSITY OF WARSAW | CeNT CENTRE OF NEW TECHNOLOGIES

invites to a seminar by

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How mutant p53 and the proteasome machinery reshape transcriptomes and proteomes of cancer cells

Date: March 1st, 2019 at 12 p.m.

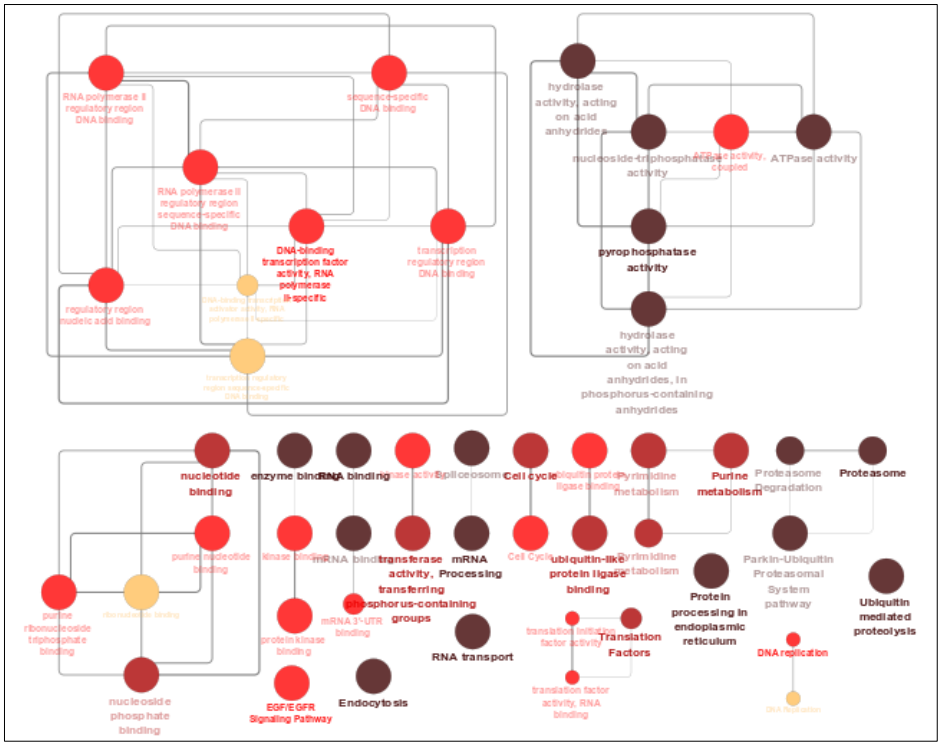
Venue: Centre of New Technologies, Banacha 2C,
Lecture Hall 0142 (Ground floor)

Host: Marta B. Wiśniewska

Abstract:

Mutated variants of the *TP53* tumor suppressor gene frequently present in human cancers are often supporting growth of cancer cells – as active oncogenes. My research in the recent years has been focused on how mutant p53 proteins reprogram cells to support growth and metastasis of breast cancer. One of the major components controlled by mutant p53 is the cellular proteasome machinery - via which p53 mutants reshape proteomes of cancer cells, affecting multiple pathways important for cancer progression.

The proteasome machinery is also a powerful oncogenic system by itself and is commonly activated in all human neoplasias. One of the main research streams of my new research group at Mossakowski Medical Research Centre PAS in Warsaw is to find out why some neoplastic cells rely more on the proteasome (such as multiple myeloma cells), while others (solid tumor-derived cells) are more resistant to proteasome inhibition. To understand this we are using a combination of large scale methods allowing for observation of global molecular landscape changes in cells. In particular, we combine proteomics and transcriptomics analyses to compare multiple myeloma and solid tumor cells upon proteasome inhibition to find common and specific proteasome targets, as well as mechanisms of proteasome inhibitor sensitivity or resistance.



Picture: Network of molecular functions common to proteasome targets identified by global proteomics across 8 human neoplastic cell lines (preliminary result; tools: MaxQuant, Perseus, Cytoscape, ClueGO, yFiles Layout Algorithms).