Protein kinases are the enzymes that catalyze adding phosphate groups to other proteins. Phosphorylation causes conformational changes in targeted protein what results in activity, localization and protein-protein interaction changes. The reversal reaction, dephosphorylation, is driven by proteins called phosphatases. Therefore, both types of enzymes regulate other proteins „on"/"off" state what enables a cell to adjust the internal molecular processes without a production of additional proteins. The on/off balance is often disturbed under disease conditions including cancer. For example, in cancer the kinases are frequently hyper-activated due to mutations it their genes or have elevated abundance what ultimately sustains uncontrollable cancerous cell growth. Modern oncological therapies are often based on kinase specific chemical inhibitors or antibodies to impede undesirable kinases' activity in cancer cells. CDK8 is a kinase that was initially found to drive oncological transformation in colorectal cancer what gave the rationale to develop chemical inhibitors targeting this kinase. However, recently published data indicate rather moderate anti- colorectal cancer activity of CDK8 inhibitors. The Selvita company who developed a CDK8 specific inhibitor, SEL120, together with scientists from the Cancer Center Institute in Warsaw have found that CDK8 inhibitors (CDK8i) potently inhibited growth of certain acute myeloid leukemia (AMLs) cell lines grown as grafts in immunodeficient mice what pointed that the drug could have anti-AML properties. However, to further ascertain on SEL120 mode of action in AMLs there is an urgent need to explore cancerous cells models on molecular level. To this end we will perform deep molecular characterization with modern technologies of SEL120 responsive and non-responsive AML cell cultures and patient derived xenografts (PDX) models that are created by injecting patient's cancerous cells into immunodeficient mice. Such a model has already been tested by the others and proved to mimic standard chemotherapy and efficiently monitor AML therapy response. The discovery CDK8 inhibitors mode of action and relevant biomarkers of response will advance drug's transition from the bench-to-bedside.