MedicalXpress

Targeted cancer treatment—new dual strategy halts cell division

September 29 2015

A team of researchers at the MedUni Vienna has confirmed in a recent study its new concept for the targeted treatment of ovarian cancer. The concept is intended to better control the development of resistance and improve treatment outcomes. The strategy focuses on halting tumour growth by inhibiting two signal networks instead of just one. The results are extremely promising and were presented at the ECC2015, which was held from the 25th to the 29th of September in Vienna. The next stage involves the verification of the concept in in vivo studies.

Targeted cancer treatment is designed to block the signalling networks used by tumour cells. This stops the malignant cells from receiving signals which lead, for example, to cell growth or cell death. Usually the structures involved are proteins, known as receptors, which are found in abundant numbers on the surface of the tumour cells or inside it and which pick up these signals and pass them on, ultimately causing degeneration of the cell.

Until now, the primary focus for treatment was the cell division signalling pathways, i.e. the mechanisms that prompt the cell to divide and grow. Thomas Grunt from the University Department of Internal Medicine I, Head of the CCC Research Cluster Cell Signaling and Metabolism and leader of the new study, says: "Unfortunately, malignant cells are very flexible and develop resistances to the new, targeted therapeutic agents we use. This is the biggest problem in oncology. Our idea was therefore to block a second signalling system in order to improve the impact of the substance being used."

One such network involves metabolic pathways, for example. They are also responsible for the establishment of the cell structure, energy gain and cell nutrition. Since malignant cells have a hyperactive fatty acid metabolism, the team of researchers took a closer look at this in their current study. Says Grunt: "We investigated how the two signalling pathways interact with each other at molecular level and we were able to identify an enzyme known as PI3K-mTORC1 kinase as the central interface for both systems. Cell tests have shown that inhibiting this enzyme leads to cell death and reduces the rate of cell division."

The next step is to organise further studies designed to test which of the substances that are already available for inhibiting PI3K-mTORC1 also work in humans. The study is being presented on Monday, 28th September, at the ECC2015 as part of the translational research poster session.

More information: "Molecular interplay between cancer cell fatty acid metabolism and oncogenic signaling as resource for novel treatment strategies against ovarian cancer." European Cancer Congress 2015 www.europeancancercongress.org … rch?abstractid=19933

Provided by Medical University of Vienna