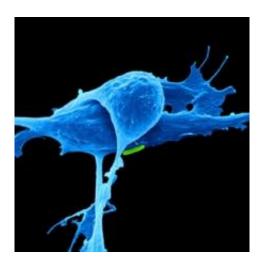


Molecule fights cancer on two fronts

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Researchers at the University of Leeds have made a new synthetic anticancer molecule that targets two key mechanisms in the spread of malignant tumours through the body.

A study published in the journal *PLOS ONE* today reports that the <u>synthetic molecule</u> JK-31 blocks the signalling of a "growth factor" chemical that promotes the creation of networks of blood vessels to feed tumours.

But the researchers also found that the new molecule intervened directly in the growth of the cancer itself, inhibiting a protein that controls the division and proliferation of <u>malignant cells</u>.



Dr Vas Ponnambalam, Reader in Human Disease Biology in the University of Leeds' Faculty of Biological Sciences, said: "The ability to mount this two-pronged attack on cancerous growths is exciting. There is a great need for better drugs against cancer than what we currently have and JK31 may represent an important addition to the toolkit for drug makers developing the next generation of drugs."

The researchers observed the effect of the synthetically produced molecule, JK-31, on the growth and proliferation of a model human breast cancer cell line and found that it effectively blocked the protein cyclin-dependent kinase 1 (CDK1), which plays a key part in the process of the division of <u>cancer cells</u>, and therefore inhibited the proliferation of the cells.

In a separate laboratory experiment, they found the same JK-31 molecule also blocked a specific growth factor (VEGF-A) produced by the cancer to attract the growth of <u>blood vessels</u>.

Other molecules exhibiting similar dual effects are known but JK-31 is the only compound so far shown to successfully target CDK1 and block VEGF-A.

More information: Antony M. Latham, Jayakanth Kankanala, Gareth W Fearnley, Matthew C Gage, Mark T Kearney, Shervanthi Homer-Vanniasinkam, Stephen B Wheatcroft, Colin W G Fishwick, and Sreenivasan Ponnambalam, "In Silico Design and Biological Evaluation of a Dual Specificity Kinase Inhibitor Targeting Cell Cycle Progression and Angiogenesis," *PLoS One* (2014).

Provided by University of Leeds



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