

New DNA research could help combat cancers that are resistant to current standard treatments

An [analysis](#) of more than 700 different cancer cell types has found thousands of Achilles' heels or cancer vulnerabilities that could lead to new ways to stop cancer cells in their tracks by using existing drugs, as well as proposing new targets for drug development.

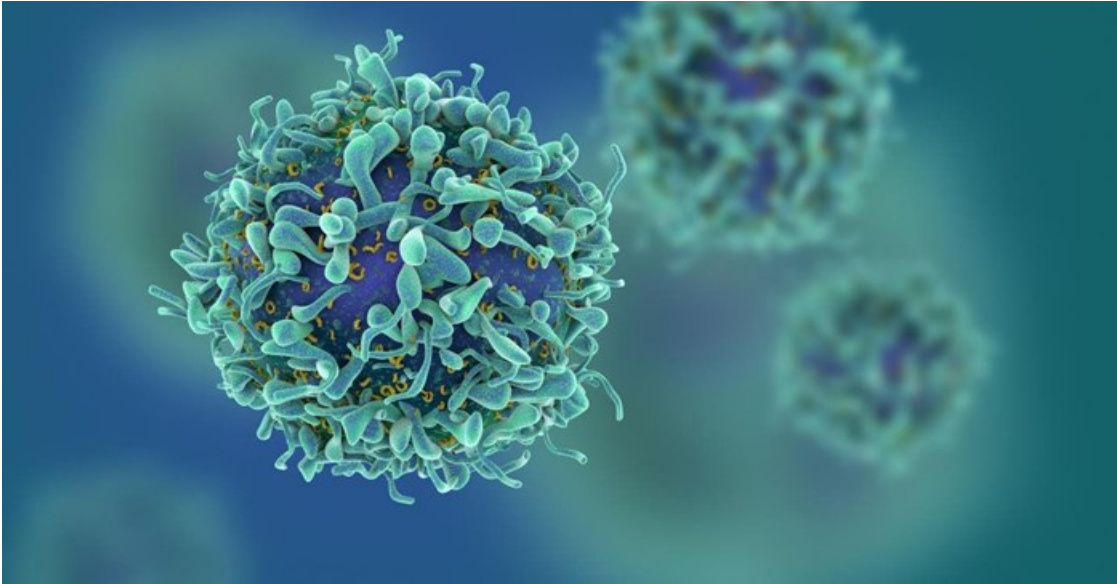


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These drugs could even be used to combat cancers that are resistant to the current standard treatments.

To conduct their study, the researchers created a computer programme called MultiSEp that analyses large and complex datasets.

“Understanding the molecular fingerprints of cancer can pinpoint ways to target drugs precisely to those patients where they will be most effective. Our work makes a step towards more effective and personalised cancer treatments, ultimately saving lives.

“We make our results available on the Synthetic Lethality with Genetics and Genomics web server, opening a window to share these rich resources with researchers across the scientific community – in order to accelerate progress in cancer research globally,” says Dr Ian Overton, senior lecturer from the Patrick G Johnston Centre for Cancer Research (PGJCCR) at Queen's University.

Cancers usually have many mutations which can cause genetic weak spots or Achilles' heels. For example, cancers frequently become more dangerous by mutating to stop some protective genes called tumour suppressors - leaving the tumour reliant upon a back-up gene.

Hitting the back-up gene with a chemical hammer, can therefore kill the cancer cells.

This new programme has identified thousands of back-up genes in more than 700 different kinds of cancer cell types, providing the intelligence to design more effective treatments in the war against cancer.

Resources

"This research represents an important resource for researchers worldwide to extract invaluable data from functional genomics screens, that has potential to translate to significant benefits for cancer patients in the long term, but also, for researchers around the world to easily and more efficiently analyse genetic data," Dr Simon McDade, senior lecturer from the PGJCCR at Queen's University, says.

"Our results provide the wider scientific community access to key datasets generated by cutting edge technologies, and a toolkit with which to analyse this data.

"Ultimately, we hope that, by increasing the reach of this data we can expedite more targeted and effective cancer treatments," says Mark Wappett, PGJCCR honorary lecturer at Queen's University and head of bioinformatics at Almac Discovery, who led the research.

The research was performed in collaboration between the Overton and McDade groups in the Patrick G Johnston Centre for Cancer Research at Queen's University Belfast, Almac Discovery and the Department of Biochemistry and Vanderbilt University, USA.

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