Match your treatment to your cancer

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Lina Happo

(Medical Xpress) -- New research has uncovered why certain cancers don't respond to conventional chemotherapy, highlighting the need to match treatments to cancers better.

Cancer researcher Lina Happo and colleagues at the Walter and Eliza Hall Institute have identified three 'cell death' genes that are crucial for making anti-cancer drugs more effective at killing cancer cells. The discovery could be the first step in developing new cancer treatments that target only cancer cells.

Most currently available chemotherapy drugs do not distinguish between normal and cancerous cells, Lina says. This means when using them that collateral damage to healthy cells—the origin of side effects—is unavoidable.

“By understanding which of the three genes we identified are required for successful drug responses, medical researchers should be able to work out how conventional cancer therapies work, and why they sometimes fail,” Lina says.

Programmed cell death, or apoptosis, removes unwanted or dangerous cells from our bodies, protecting us against cancer and autoimmune diseases. The process is regulated by a family of genes called Bcl-2.

“Many anti-cancer drugs act by damaging the DNA in tumour cells, causing the cells themselves to commit suicide. Until now we didn’t know which genes were essential for this process,” Lina says.

Working with colleagues from the institute’s Molecular Genetics of Cancer division, she was able to identify that three Bcl-2 genes – puma, noxa and bim – tell cancer cells to commit suicide following treatment with conventional chemotherapy drugs.

“In our studies we found that puma, noxa and bim work together to instruct the cancer cell to die, once its DNA has been damaged by chemotherapy drugs.”

“But if certain combinations of these genes are missing or not functioning, the anti-cancer therapies are unable to work effectively, so the cancer cells continue to survive and the tumour continues to grow,” she said.

Abnormalities within the Bcl-2 gene family are common in many human cancers, Lina says, and can often be responsible for resistance to chemotherapy treatments.

Her discovery has the potential to improve treatment through the development of more efficient, targeted therapies for blood, breast and

ovarian cancers.

“We hope to be able to reduce unwarranted toxicity, ultimately improving the quality of life for patients.”

Lina Happo is one of 16 early-career scientists unveiling their research to the public for the first time thanks to Fresh Science, a national program sponsored by the Australian Government. Her challenges included presenting her discoveries in verse at a Melbourne pub.

Provided by Fresh Science