

Brain tumour cells killed by anti-nausea drug

March 18 2013

(Medical Xpress)—New research from the University of Adelaide has shown for the first time that the growth of brain tumours can be halted by a drug currently being used to help patients recover from the side effects of chemotherapy.

The discovery has been made during a study looking at the relationship between brain tumours and a peptide associated with inflammation in the brain, called "substance P".

Substance P is commonly released throughout the body by the nervous system, and contributes to tissue swelling following injury. In the brain, levels of substance P greatly increase after [traumatic brain injury](#) and stroke.

"Researchers have known for some time that levels of substance P are also greatly increased in different tumour types around the body," says Dr Elizabeth Harford-Wright, a postdoctoral fellow in the University's Adelaide Centre for Neuroscience Research.

"We wanted to know if these elevated levels of the peptide were also present in [brain tumour](#) cells, and if so, whether or not they were affecting tumour growth. Importantly, we wanted to see if we could stop tumour growth by blocking substance P."

Dr Harford-Wright found that levels of substance P were greatly increased in brain tumour tissue.

Knowing that substance P binds to a receptor called NK1, Dr Harford-Wright used an antagonist drug called Emend to stop substance P binding to the receptor. Emend is already used in [cancer clinics](#) to help patients with chemotherapy-induced nausea.

The results were startling.

"We were successful in blocking substance P from binding to the NK1 receptor, which resulted in a reduction in brain tumour growth - and it also caused [cell death](#) in the tumour cells," Dr Harford-Wright says.

"So preventing the actions of [substance P](#) from carrying out its role in brain tumours actually halted the growth of [brain cancer](#).

"This is a very exciting result, and it offers further opportunities to study possible brain tumour treatments over the coming years."

Provided by University of Adelaide

Citation: Brain tumour cells killed by anti-nausea drug (2013, March 18) retrieved 23 April 2024 from <https://medicalxpress.com/news/2013-03-brain-tumour-cells-anti-nausea-drug.html>

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