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Researchers discover potential cancer therapy target

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One of the most important genes in the human genome is called p53 and its function is to suppress tumours, according to Roger Leng, a researcher in the Faculty of Medicine & Dentistry. Leng has discovered the mechanism by which p53 is inactivated in cancerous cells, allowing tumours to grow.

"Successful completion of the proposed experiments could lead to novel anti-cancer therapies that could potentially improve the prognosis for cancer patients and reduce the public health burden from cancer," said Leng.

It has long been known by scientists that another protein, MDM2, lowers p53 in the body, but in cancerous cells p53 is inactivated in more than 50 per cent of all human tumours. MDM2 does not have the ability to functionally silence the tumour suppressing protein on its own, leaving scientists wondering what molecule in the body is helping MDM2 to nearly eliminate p53 in cancerous cells.

Leng's lab has answered that question and the culprit is called UBE4B. Leng made the discovery because he found that UBE4B binds with both p53 and MDM2. From there his lab was able to discover the relationship between the proteins.

Paired with MDM2, also known as HDM2 in humans, the two proteins completely degrade p53 in a laboratory model. This is a process known as poly-ubiquitination, which means a specific protein completely

disappears in a cell.

They also did experiments on cancerous human brain tissue and found the same results.

"They have the same function," said Leng. "The idea now is you can target UBE4B and MDM2 won't function."

This discovery landed Leng in the pages of one of the highest-impact scientific journals, *Nature Medicine*. His work was published online on Sunday, Feb. 13.

Now, the Alberta Heritage Foundation for Medical Research Scholar and Canadian Institutes of Health Research funded scientist wants to further understand the mechanisms by which UBE4B functions.

"We want to understand how it regulates MDM2," said Leng. "We also want to see, if you get DNA damage, what happens in UBE4B and p53."

All of which could provide answers which eventually lead to a pharmacological target for cancer therapy.

Provided by University of Alberta

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