

# Researchers reveal PAX gene's role in cancer

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(Medical Xpress) -- University of Otago researchers have uncovered further evidence that PAX genes – members of a small family of genes that play important roles in embryonic development – also allow cancer cells to grow and divide in adult tissue.

In 2003, research led by Otago's Professor Michael Eccles was the first in the world to show that proteins from one or more of the nine PAX [genes](#) were present in a wide variety of common cancers. Additionally, the researchers found that “silencing” the gene expression of PAX2 in ovarian and bladder [cancer cells](#), and of PAX3 in melanoma, led to the rapid death of the cells.

Now Professor Eccles and colleagues' investigations of PAX8, which have just been published in the leading UK journal *Oncogene*, reveal that silencing this gene also has a dramatic effect on cells in several kinds of tumours, but through a different mechanism.

After detecting high levels of PAX8 protein in the vast majority of kidney cancers, ovarian cancers and thyroid cancers they studied, the researchers used molecular techniques to silence PAX8 in several cancer cell lines.

“We found that these PAX8-depleted cancer cells ceased growing and dividing. The cells were essentially stopped in their tracks through the failure of multiple mechanisms and pathways crucial to their cell division cycle. They then entered into a state called senescence in which they no longer divided, and after that they ultimately died,” Professor

Eccles says.

The findings suggest that PAX8 could be a good target for the development of new [cancer](#) therapies, he says.

“Any resulting drugs would be a long way down the road, but in the meantime this research helps confirm that a focus on PAX genes may prove to be a fruitful line of attack against a number of cancers,” he says.

**More information:** The research article can be accessed at:  
[www.nature.com/onc/journal/vao ... /pdf/onc2011190a.pdf](http://www.nature.com/onc/journal/vao.../pdf/onc2011190a.pdf)

Provided by University of Otago

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