A gene that protects against colorectal cancers

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The research team in France has developed an animal model carrying a mutation of the DCC gene. Mice carrying the mutation develop tumours, because this gene can no longer induce the death of the cancer cells. This discovery could lead to the development of a new targeted cancer treatment that aims to reactivate the dying of cancer cells.

The results of this study have been published as a Letter in the 11th December 2011 issue of the journal *Nature*.

The team led by Patrick Mehlen, Director of the DEVweCAN 'Laboratory of Excellence' at the Lyon Cancer Research Centre, studies the cell death process (apoptosis) and, in particular, the mechanism that makes the cells understand that they should initiate a self-destruction process when they become abnormal. Patrick Mehlen's team suggested that this mechanism could operate via sentinels located on the surface of cells, which examine their environment. The scientists named these sentinels 'dependence receptors'.

The research team focused on this concept of 'dependence receptors'. When a cell receptor is associated with its ligand, the classic message indicates 'all is well', and leads to cell survival. On the other hand, when the receptor is deprived of its ligand, it can send a message leading to cell death. This mechanism is also called 'apoptosis.' When this is applied to cancer research, the absence of ligands could cause the death of cancer cells that proliferate in an anarchic manner.

In this study, Patrick Mehlen's team shows that the DCC gene (Deleted Colorectal Cancer), which codes for a 'dependence receptor', protects the organism from the onset of cancer by causing the death of cells that become cancerous. The researchers used a mouse model where the DCC gene has been genetically modified. The mutation of this dependence receptor prevents the induction of apoptosis. When the DCC gene is eliminated by mutation, the mouse spontaneously develops colon cancer.

'The organism is naturally protected from the development of cancers thanks to the presence of this tumour-suppressing gene. Unfortunately, certain cancer cells escape from this control by blocking this 'dependence receptor' mechanism. That is how we know that the DCC gene is extinguished in most human cancers,' explains Patrick Mehlen.

In the near future, this research work could lead to a new targeted treatment that aims to reactivate the death of the cancer cells to destroy breast cancer, lung cancer, etc. 'Our group has developed several candidate drugs that reactivate the cell death induced by the DCC receptor in animal models, and we hope to be able to carry out human clinical testing of these candidate drugs in three years' time,' concludes Patrick Mehlen.

More information: DCC constrains tumour progression via its dependence receptor activity, *Nature*, Dec 11, 2011. doi:10.1038/nature10708

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