Paradigm Shift in Cancer Research? Focusing on Tumour Stem Cells and Their Formation

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A research project is currently in progress that focuses on tumour stem cells and their division. Until recently, no one had any idea of the existence of these types of cells, even though they can play a crucial role in the formation of tumours. A project backed by the Austrian Science Fund FWF is now investigating the critical role of an individual protein, whose absence can be of vital importance for tumour stem cells. The project results will create the basis for optimizing certain cancer treatments.

Cancer cells split very quickly, thus giving rise to tumours. It is this fast division that distinguishes tumour cells from many other cells, but it is also their Achilles heel, as many cancer treatments eliminate only rapidly-dividing cells and preserve others. This form of treatment works well and has proved very successful. However, it does not always have a lasting effect, and tumours that seemed to have been eliminated long ago are able to regenerate themselves. The question as to how these tumours can regenerate when the rapidly-dividing cells have been removed is something that puzzled doctors and researchers for a long time. They found the - seemingly simple - answer a few years ago: not ALL tumour cells divide rapidly. Tumour stem cells divide slowly but can form new tumour cells and, therefore, new tumours.

Power of a Protein

The molecular mechanisms that lead to the formation of tumour stem cells are still largely unknown today. Prof. Jürgen Knoblich from the Institute for Molecular Biotechnology of the IMBA - Austrian Academy of Sciences is helping to turn this situation around. Prof. Knoblich and his team have already made a discovery that is making waves in the medical world. Prof. Knoblich explains: "We discovered that, in normal stem cells in the nervous system, a specific protein regulates the propagation of these stem cells. If this protein, which is known as Brat, is missing, it leads to the uncontrolled and rapid propagation of stem cells. Put simply, it leads to the formation of a tumour." In this way, the scientists have shown that cancer can originate from a single stem cell as a result of a single protein.

The Fate of Cells

In stem cells of the nervous system of fruit flies, the scientists found that, after the cell division, the Brat protein determines the fate of the two daughter cells that are created. The Brat protein is present in one of the daughter cells, and this one develops into a somatic cell, while the other does not contain the protein and remains a stem cell. At the same time, Brat prevents further cell division in the somatic cell. Prof. Knoblich continues: "We then wanted to find out what happens when Brat is missing from both daughter cells instead of just one. We assumed that, no longer protected by Brat, both cells would divide, as would their daughter cells and their daughter cells in turn. In other words, it would lead to unchecked cell growth and a rapid increase in the number of cells. And our assumption was confirmed." Once the IMBA team had succeeded in eliminating the Brat protein, they observed a quick rise in the number of neural stem cells. These eventually went on to form a tumour, which spread throughout the fly's body.

To get a better understanding of how Brat works, Prof. Knoblich and his team are now trying to identify proteins that are controlled by Brat. The importance of having a detailed understanding of the effect of Brat extends far beyond the boundaries of fundamental research. The Brat protein is not only present in flies; it also exists in a similar form in humans. If this human form of Brat has - as expected - comparable functions for the division of stem cells, it could also play a part in the formation of tumours in people. This FWF-supported project is making a contribution to the understanding of stem cells - and thus laying the foundation for future cancer treatments that could have a long-term impact in the future.

Image and text will be available online from Monday, 27th July 2009, 3.00 p.m. CET onwards:
www.fwf.ac.at/en/public_relations/press/pv200907-2en.html

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