

Scientists discover how daughter cells receive the same number of chromosomes

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Scientists at Warwick Medical School have uncovered the molecular process of how cells are by-passing the body's inbuilt 'health checkpoint' with cells that carry unequal numbers of chromosomes that have a higher risk of developing cancer.

Studying simple <u>yeast cells</u>, scientists now understand the mechanism by which cells ensure their daughter cells receive the correct number of chromosomes.

Most cells in our bodies contain 23 pairs of chromosomes that encode our individual genetic identities. In healthy, dividing human cells, each of these chromosomes is duplicated and one copy passed to each of the two daughter cells. However, if this process is disturbed, <u>daughter cells</u> receive an unequal number of chromosomes, a state that is known to drive normal cells to become cancerous. In fact, aggressive human tumours are frequently composed of cells with an abnormal complement of chromosomes.

Professor Jonathan Millar explained: "This cell division process is monitored by the body's <u>surveillance system</u> known as the 'spindle checkpoint', and that is only switched off once everything within the cell is set up correctly. Amazingly, all of the elements of this process are conserved from yeast to human cells. Therefore it is extremely likely that what we have found in yeast also happens in <u>human cells</u>. So by preventing this process happening with drugs, you could restrict the cell's ability to develop into full blown cancer," explained Professor Millar.



Currently, one of the most frequently used classes of anti-cancer drugs are taxanes, which target the mitotic apparatus in part by preventing proper silencing of the spindle checkpoint. However, this class of drug affects healthy and <u>cancerous cells</u> alike and can have debilitating side effects including permanent <u>neurological damage</u> and hair loss.

Professor Millar explained: "Now that we have pinpointed the central elements of cell division, we are in a great position to design drugs that can be more selective and targeted about which cells they treat. But this is just the start – much more research has to be done before we can convert this into a commercial treatment for patients, but we are greatly encouraged that our research here at Warwick is leading the way in the search for more effective cancer treatments with fewer side effects."

More information: Professor Millar's research paper was published in *Developmental Cell*, <u>doi:10.1016/j.devcel.2011.05.008</u>

Provided by University of Warwick

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