Cancer cells do it the "quick-and-dirty way"

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(Medical Xpress)—The hallmark of cancer is uncontrolled cell growth directed by a cell cycle engine gone into overdrive. The centrepiece of this engine is the enzyme Cdc2 kinase. While Cdc2 kinase is tightly regulated in normal cells, this control is lost in cancer cells.

Cutting-edge research conducted at Bangor University in the North West Cancer Research Institute discovered now that hyperactive Cdc2 kinase not only forces cells into uncontrolled growth but also reprograms the repair of broken chromosomes.

The team leader Dr Thomas Caspari explains: "When a chromosome breaks, human cells have the option to repair it safely, which takes time, or quickly, which could result in mistakes. Our work has now revealed that cells with hyper-active Cdc2 kinase prefer the `quick and dirty' way over the safe option. This exciting discovery could explain why cancer cells often splice the wrong ends of broken chromosomes together, a process which fuels tumour growth."

The findings will be reported in the June issue of the high-impact journal *Nucleic Acid Research* which is published by Oxford University Press.

Dr Thomas Caspari comments:

"This exciting research would not have been possible without the generous support from the North West Cancer Research Fund, a charity based in Liverpool, and from the Government of Libya, which supports

the lead author Mr Salah Adam Mahyous Saeyd. It also shows how international research at Bangor University is as the two other students, who contributed to the work, are from Poland and China."

More information: Salah Adam Mahyous Saeyd, Katarzyna Ewert-Krzemieniewska, Boyin Liu, and Thomas Caspari. "Hyperactive Cdc2 kinase interferes with the response to broken replication forks by trapping S.pombe Crb2 in its mitotic T215 phosphorylated state." *Nucl. Acids Res*. first published online May 26, 2014 DOI: 10.1093/nar/gku452

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