

## Rogue gene hijacks stem cells to jumpstart human cancer

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A gene thought to be responsible for initiating human cancer has been identified by researchers at Barts and The London School of Medicine and Dentistry. The study - published online today (9 November) in the journal *Cancer Research* - paves the way for developing early cancer diagnostic tests, and finding new treatments that prevent or stop the spread of cancer cells at an early stage.

Led by Dr Muy-Tek Teh of the Institute of Dentistry at Barts and The London School of Medicine and Dentistry researchers have shown that a gene called FOXM1 exploits the inherent self-renewal property of stem cells causing excessive <u>cell proliferation</u>. Using adult human stem cells isolated from mouth tissues the team demonstrated that normal stem cells engineered in the lab to express abnormal levels of FOXM1 gene, triggered excessive cell growth within a 3D tissue culture model system set up to mimic human <u>tissue regeneration</u> in the laboratory. The 3D tissue culture system allows scientists to perform research on manipulated human cells without provoking ethical issues associated with human or animal subjects.

<u>Stem cells</u> expressing normal levels of the FOXM1 gene did not cause excessive cell growth. The abnormal growth triggered by FOXM1 resulted in a condition called hyperplasia - an early hallmark of precancer. This is thought to represent the very first step of a series of abnormal molecular events leading to cancer formation.

Dr Teh said: "Now we know that FOXM1 plays a key role in cancer



initiation we aim to translate our basic findings into clinically useful molecular diagnostic tests to detect cancer growth at early stages. Furthermore, understanding the origin of cancer initiation may unveil new research opportunities for finding effective anti-tumour drugs that stop or prevent cancer at its earliest incipient stage."

**More information:** 'Induction of Human Epithelial Stem/Progenitor Expansion by FOXM1' is published advanced online on 9 November 2010 in *Cancer Research*.

Provided by Queen Mary, University of London

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