

New genetic discovery advances understanding of prostate cancer

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A new and important genetic discovery, which sheds light on how prostate cancers develop and spread, has been made by an international research team led by scientists at The University of Nottingham.

Prostate cancer is one of the most common cancers affecting [men](#). In the UK about one in eight men will develop it at some point in their lives, with older men and those with a family history of prostate cancer most at risk.

It is not yet possible to accurately distinguish between 'indolent' [prostate cancers](#), which need little, if any treatment, and 'aggressive' cancers, which require intensive interventions. Now in new research published in *Oncotarget*, a multi-disciplinary team at Nottingham, Weill Cornell Medical School, Lund University in Sweden and Copenhagen University in Denmark, have identified a significant gene called miR137 that is

switched off in prostate cancer cells.

Lead researcher at Nottingham, Dr Nigel Mongan said: "With many men continuing to die from [metastatic prostate cancer](#), there is an urgent need to develop new ways to enable the early identification of aggressive cancers when such tumours remain localised within the [prostate gland](#) when surgery is most effective. We also need to make sure that men with indolent disease do not receive unnecessary treatment which can lead to urinary continence and sexual dysfunction."

The researchers studied the role of androgens in prostate cancer. Androgens are important signaling molecules, which play an essential role in men's health by driving the development, repair and regeneration of the prostate and other tissues. However defective and amplified androgen signaling can trigger prostate cancer and its spread.

For this reason, many available prostate cancer treatments are aimed at blocking androgen signaling. However, resistance to such therapies is a major clinical challenge. The gene identified by the team, called miR137, is switched off in [prostate cancer cells](#). It functions like a 'dimmer switch' in normal cells to reduce androgen signaling.

In prostate cancer, where miR137 is switched off, the effect of androgen signaling is increased. Therefore the loss of miR137 leads to enhanced androgen signaling which contributes to prostate cancer initiation and progression.

The study has also identified many new potential targets for the next generation of drugs to treat prostate cancer. New research is now underway in the Mongan's laboratory at Nottingham to test the effect of various pharmacological treatments in pre-clinical prostate cancer studies.

Provided by University of Nottingham

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