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'Genomic junk' of iron storage gene FTH1 critical for suppressing prostate cancer growth

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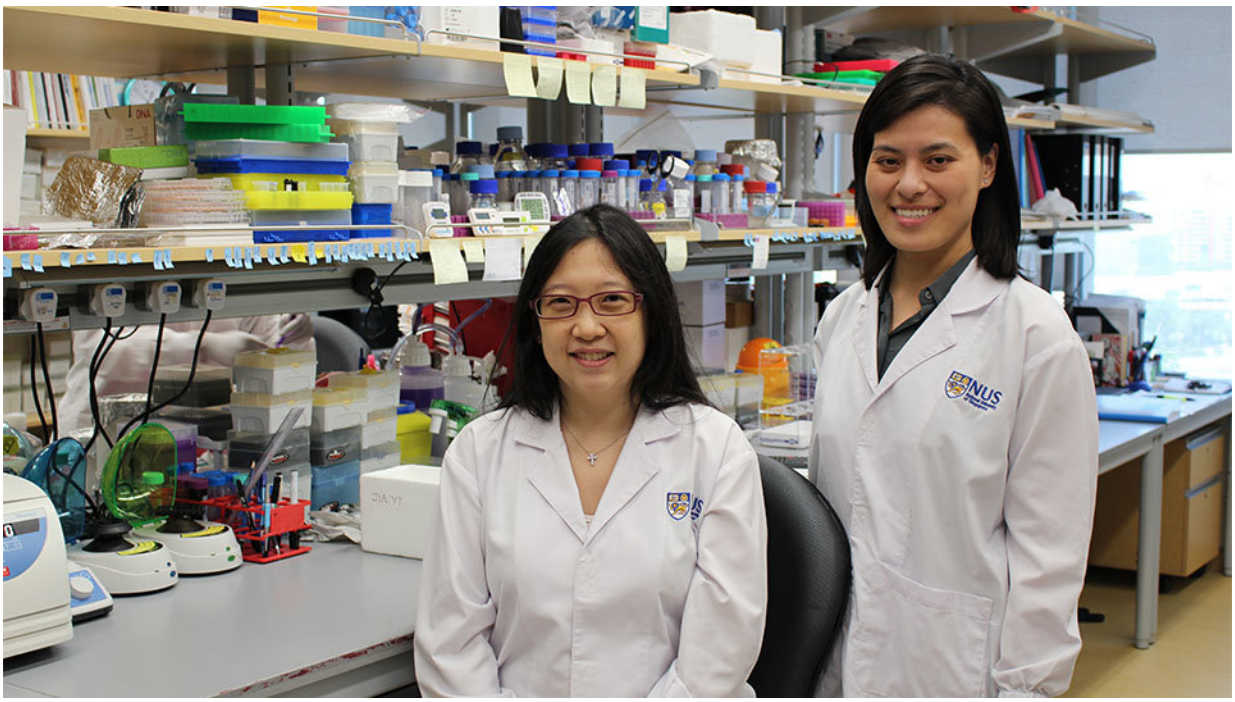

Assistant Professor Yvonne Tay (left), Principal Investigator at the Cancer Science Institute of Singapore at the National University of Singapore and Dr Chan Jia Jia (right), a Research Fellow in Asst Prof Tay's group uncovered new biomarkers and therapeutic targets for prostate cancer linked to the FTH1 iron storage gene. Credit: National University of Singapore

An abnormally high level of iron in the body is associated with prostate

cancer, and researchers from the Cancer Science Institute of Singapore (CSI Singapore) at the National University of Singapore may have uncovered the mechanism to explain this link. They have found the role of the iron storage gene, FTH1, and its pseudogenes in regulating iron levels in cells and slowing down prostate cancer growth. The new findings could pave the way for future developments in prostate cancer diagnostics and therapeutics.

Prostate cancer is the most common cancer and the second leading cause of cancer death in men worldwide. The survival rate for prostate cancer is high if diagnosed early but it falls significantly at advanced stages. Currently, there is no available drug to treat aggressive or late-stage prostate cancer, and the accuracy of current screening techniques remain relatively low at 20 to 30%. With global prostate cancer incidence rising rapidly, accounting for 9.6% of all newly diagnosed cancer cases in 2017, there is a need to attain better understanding of the underlying biological mechanisms leading to the disease in order to build more robust diagnostic tools and efficacious therapies against it.

In the study published in the scientific journal, *Nucleic Acids Research*, earlier this year, researchers from CSI Singapore focused their investigation on the iron storage gene, FTH1, noting the link between iron level in cells and the development of prostate cancer. It was found that the function of FTH1 is highly influenced by its pseudogenes, a set of genes originating from their parental gene, which were previously thought to be non-functional and were classified as 'genomic junk'. FTH1 and its pseudogenes are often suppressed in prostate cancer, and expression of both FTH1 and its pseudogenes are required to reduce iron levels in cells and slow down prostate cancer growth.

Further experiments showed that FTH1 and its pseudogenes are linked by a shared pool of microRNAs that are often highly expressed in prostate cancer, and these microRNAs inhibit the regulation of iron

levels by the genes. As such, isolating and manipulating the pool of microRNAs can therefore relieve the inhibition, restore the iron regulatory functions of FTH1 and its pseudogenes and their tumour suppressive effects.

"Our study presents an interesting finding in the form of the multicomponent, tumour suppressive FTH1 gene:pseudogene network which can be harnessed for future diagnostic and drug development. The highly expressed microRNAs that link FTH1 and the pseudogenes are attractive biomarkers, as well as therapeutic targets for prostate cancer. Targeting or restoring proper iron storage could also be a potential avenue for therapeutic development," said Assistant Professor Yvonne Tay, Principal Investigator at CSI Singapore who led the study.

High iron levels in cells is also linked to various types of cancer apart from prostate cancer. As such, the research team will further explore and delineate this regulatory network, not only in prostate cancer, but also other cancer types to uncover more potential biomarkers and druggable targets for more effective cancer treatment.

Provided by National University of Singapore

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