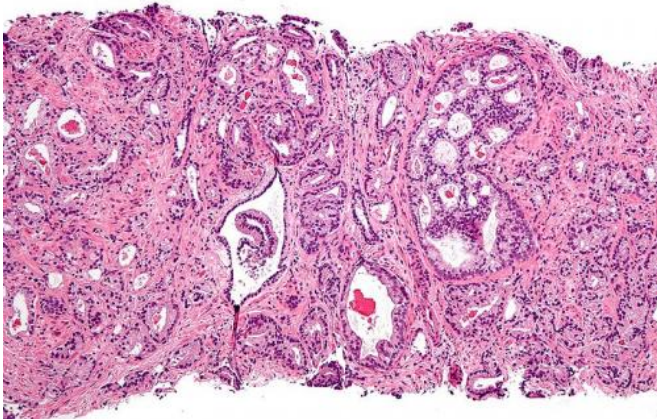


Researchers pinpoint key regulatory role of noncoding genes in prostate cancer development

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Micrograph showing prostatic acinar adenocarcinoma (the most common form of prostate cancer) Credit: Wikipedia

Prostate cancer researchers studying genetic variations have pinpointed 45 genes associated with disease development and progression.

Principal investigator Hansen He says the findings published online today in *Nature Genetics* show how these genes - known as "noncoding RNA" - function in activating the disease process. Dr. He, an epigeneticist, is a Scientist at the Princess Margaret Cancer Centre, University Health Network. He is also Assistant Professor in the Department of Medical Biophysics, University of Toronto.

"Our research looked at genetic variations associated with prostate cancer and found that about half of these variations may function through noncoding genes rather than the protein-coding genes. In other words, we have discovered that noncoding RNA has a very important function in driving prostate cancer development and disease

progression."

Dr. He says: "In prostate cancer there are more than 100 known risk regions associated with the development and progression of the disease but for most of them we don't know how. In our work, we found that half those risk regions may function through noncoding genes."

Dr. He says that integrating this new knowledge about genetic variations and the function of noncoding RNA moves the science closer to developing a clinical biomarker to advance personalized cancer medicine for patients by being able to predict who will develop prostate cancer and whether or not it will be aggressive.

The research team collaborated with the Princess Margaret Genome Centre and delved further into the genetic variations associated with noncoding RNA PCAT1, which is already known to be highly expressed in [prostate cancer](#) patients, to zero in on how the noncoding genes function.

"Noncoding RNA has many functions and in this study we found that PCAT1 functions as a kind of glue to attract different protein complexes together and guide them to specific genomic location to activate their target gene expression that starts the disease process.

"We are going to expand this knowledge as we research the other 44 genes associated with genetic variations," says Dr. He.

"Cancer is very smart to take every possible way to survive and use every piece of our genome. If research only focuses on the two percent of the genome that is the protein-coding genes, we will have limited understanding of how the cancer can survive. We cannot achieve personalized cancer

medicine without understanding the other 98 percent of our genome."

The other 98 percent - what used to be called 'junk DNA' because it was outside the protein-coding genes with no known function - is now providing a treasure trove to epigeneticists such as Dr. He.

"The major contribution of our work is to the link genetic variations outside of the gene to noncoding genes rather than protein-coding [genes](#), which have been the traditional research focus."

More information: Modulation of long noncoding RNAs by risk SNPs underlying genetic predispositions to prostate cancer, [nature.com/articles/doi:10.1038/ng.3637](https://www.nature.com/articles/doi:10.1038/ng.3637)

Provided by University Health Network

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