Mechanism affecting risk of prostate cancer is found

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A research group at Biocenter Oulu in Finland has identified a mechanism related to a transcription factor that binds much more strongly onto a particular SNP variant, thereby initiating a genetic programme which enhances prostate cancer proliferation and metastasis. The study opens up an important new direction in investigating the mechanisms related to the way in which SNP variations cause an elevated risk of prostate cancer and other human diseases. Published in *Nature Genetics*, the study was partly funded by the Academy of Finland.

Prostate cancer is the most commonly diagnosed malignancy and the second most common cause of cancer-related deaths in men worldwide. In Finland, more than 4,000 new cases are diagnosed every year.

The human genome is mainly identical throughout the human population worldwide. However, millions of small variations or polymorphisms, often located in a single nucleotide, can be found between individuals. These variations are known as single nucleotide polymorphisms (SNP). Using DNA samples from tens of thousands of prostate cancer patients and healthy men, comparative genetic studies, known as genome-wide association studies, have identified dozens of SNPs associated with the risk of prostate cancer. However, because most of these SNPs are not found in the protein-coding regions of the genome, finding the genes that contribute to the risk of prostate cancer is difficult. For this reason, the question of how these single nucleotide genetic variations or SNPs lead to a risk of prostate cancer has not yet been answered.

More information: Qilai Huang, Thomas Whitington, Ping Gao, Johan F Lindberg, Yuehong Yang, Jielin Sun, Marja-Riitta Väisänen, Robert Szulkin, Matti Annala, Jian Yan, Lars A Egevad, Kai Zhang, Ruizhu Lin, Arttu Jolma, Matti Nykter, Aki Manninen, Fredrik Wiklund, Markku H Vaarala, Tapio Visakorpi, Jianfeng Xu, Jussi Taipale & Gong-Hong Wei, "A prostate cancer susceptibility allele at 6q22 increases RFX6 expression by modulating HOXB13 chromatin binding", *Nature Genetics* (2014), DOI: 10.1038/ng.2862

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