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Identification of gene defects helps the treatment of prostate cancer

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The current method of treating prostate cancer involves identifying gene defects, which could help with the diagnosis of cancer and the development of individualised cancer treatments for patients. Professor Tapio Visakorpi at the University of Tampere is studying the molecular biology of prostate cancer, with funding by the Academy of Finland. The goal is to obtain a holistic picture of the disease's mechanisms and use those mechanisms as a basis for developing new treatments.

Prostate cancer is the most common cancer among Finnish men and the second most common cancerous cause of death in males. The disease's underlying mechanisms vary significantly from one individual to the next. Therefore, in Visakorpi's opinion, prostate cancer treatments should be designed individually for each patient according to their personal clinical picture.

"Most of the prostate cancers diagnosed are relatively benign and don't necessarily require active treatment. On the other hand, some prostate cancers are highly aggressive. A major problem from the treatment perspective is that once the disease has been diagnosed, the clinical course is difficult to predict reliably," says Visakorpi.

Several new treatments have been developed for prostate cancer in the past ten years. The same problem remains: the inability to predict which treatment will be most effective for each patient.

"Recent genome studies have shown that even though prostate cancer

initiates in a single cell of origin, several cancer cell subpopulations with different genome types emerge as the disease progresses. This is not a single disease; several mechanisms lead to the emergence of the disease. Therefore, it's important to identify those genome defects in each patient that occur in all cancer cells, that is, the so-called truncal mutations, and target the treatment to them," says Visakorpi. "This requires taking multiple samples from the patient. The processing of samples also needs to be improved to make them more suitable for molecular analysis than the current methods. We've developed a new processing method for cancerous tissue."

Some current treatments can become harmful to the patient

For half a century now, it has been known that prostate cancer growth is stimulated by male hormones, androgens. Hormonal therapy, which prevents the production or effects of androgens, has been the so-called gold standard in treating the advanced form of the disease. However, prostate cancer can reactivate the androgen receptor signalling pathway during treatment.

"It was already proven back in the 1990s that one mechanism stimulating this reactivation is the amplification of the androgen receptor (AR) gene. New research has shown that glucocorticoid medication, which is often used to treat end-stage prostate cancer, can turn against the patient. This causes a mutation in the AR gene, which leads to the activation of the signalling pathway by glucocorticoids," Visakorpi explains.

"All this indicates that we should be able to monitor the genome changes in each patient's cancer cells, not just at the time of diagnosis but also during treatment as the disease progresses. This would allow for the tailoring of the cancer therapy at any given time throughout the

progression of the disease."

Some types of prostate cancer eventually become independent of androgens. Visakorpi's research team has found a new mechanism related to the activation of the transcription cofactor HES6 as the result of gene fusion, which leads to this type of cancer cell development. These types of prostate cancer need non-hormonal therapy.

Provided by Academy of Finland

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