



Hypothetical Prostate Biopsy and Production of Molecular Data in Diagnosis and Prognosis

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ABOUT THE STUDY

The study of Circulating Cell-Free DNA (ccfDNA) stands as one of the most promising methods for non-invasive disease monitoring. It has been successfully utilised to monitor tumour evolution and the development of resistance in cancer care, as well as for assisting in the therapy selection process for a variety of non-hematologic disorders. All cells are assumed to secrete ccfDNA into the circulation as a result of either active secretion or as a result of apoptosis or necrosis, in response to both physiological processes and malignant and non-malignant pathological circumstances, such as inflammation and tissue damage. Although problems in cancer patients were initially seen decades after the first description of ccfDNA in plasma in 1948, the precise mechanism of its release and its biological characteristics are still unknown.

Circulating tumour DNA (ctDNA) is released into the bloodstream during the course of cancer growth and mixes with a greater quantity of physiologically produced nonmalignant DNA, which is primarily sourced from hematopoietic cells. The percentage of ccfDNA produced by cancer is highly variable and primarily depends on two factors: the stage of the disease (more ctDNA indicates a higher disease burden) and the location of the tumour (colon, gastroduodenal tract, breast, pancreas, liver, and skin cancers release the most ctDNA, whereas glioma, thyroid, kidney, and prostate tumours are associated with the least amount of ctDNA in plasma). The blood-brain barrier's role in gliomas has been used to explain these tissue differences.

With a half-life of less than two hours, ccfDNA is rapidly degraded by nucleases after it is released into the bloodstream. As a result, the majority of ccfDNA exhibits a peak between 160 and 170 bp, which closely corresponds to the area of DNA that is protected by the interaction between histones and nucleosomes. There have also been reports of larger pieces, which are thought to be nucleosome multimers and range in size from 360 to 400 bp and beyond. It has been demonstrated that

tumours and dying cells emit longer fragments, up to 10 kb. The organ of origin of the ccfDNA has been successfully determined using nucleosome phasing analysis and tissue-specific methylation patterns. Recently, it was discovered that the length of ctDNA fragments from various malignancies varied more than non-cancer ccfDNA, perhaps reflecting chromatin structural modifications unique to the tumour.

Only a few studies have looked into ctDNA as a potential biomarker in localised disease to distinguish PCa from benign prostatic hyperplasia in patients with elevated PSA, to provide prognostic information in patients undergoing radical prostatectomy, or to follow-up patients with PCa. Hennigan and colleagues recently confirmed that in patients with localised PCa, allele-specific alterations in ctDNA are below the detection threshold, even in high-risk patients who will eventually develop disease recurrence. In contrast, more studies investigated the viability of using ctDNA in advanced metastatic and castration-resistant disease. These studies showed that ctDNA from liquid biopsies can be used to gain knowledge about the mutational burden of metastatic PCa without the need for direct tissue sampling and as a prognostic biomarker of therapeutic response.

It has been unequivocally established that specific circumstances, such as inflammation, physical activity, or tissue damage, significantly raise the ccfDNA level. It was demonstrated, in instance, that during surgery, ccfDNA levels may rise by more than an order of magnitude. Similar to this, we could speculate that prostate biopsy, which necessitates multiple punctures of the organ, would release a significant amount of prostate DNA representative of all analysed regions, creating a sort of temporary time window for ccfDNA analysis, which may allow getting genetic information for a precision-decision making, even in localised PCa. Therefore, we investigated the possibility that prostate biopsy could result in a brief rise in prostate-derived ccfDNA, which is also probably enriched in ctDNA and could provide molecular information on the tumour that is useful for diagnosis, prognosis, and theranostic purposes.

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