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## Role and applications of artificial intelligence (AI) and biomedical vibrational spectroscopy in cancer diagnostics

Alireza Heidari, Elena Locci and Silvia Raymond

**Abstract**

The CARTITUDE-1 study is a stage 1B / II clinical trial. The trial targeted B-cell maturation antigen by targeting CAR-T cell therapy in patients with multiple myeloma who had received at least three previous lines of treatment with standard drugs, including proteasome inhibitors, immunosuppressive drugs, and CD38 antibodies. Cilta-cel is made from the patient's own T cells, which is genetically engineered and is given as a single injection. The overall response rate to treatment was 97%, while the complete response rate and progression-free survival rate were 67% and 77%, respectively. The overall survival rate was 89%. Updates to this study were recently presented at the annual meeting of the American Clinical Oncology Association after our paper was accepted for publication in The Lancet. Our ASCO presentation showed a deeper response for patients receiving this treatment. These results are very impressive for patients with myeloma who have already undergone many treatment lines for their disease. It will be important to better understand the clinical features of patients who have experienced long-term recovery from this treatment and the mechanisms by which patients' relapse. While it is not possible to formally conduct two separate single-arm studies on the idea of cells and cilia, the rate of dramatic response and progression-free survival of eyelash-treated patients is very interesting.

**Keywords:** Cancer, cells, tissues, tumors, prevention, prognosis, diagnosis, imaging, screening, treatment, management

**1. Introduction**

According to the American Cancer Society, about 80% of breast cancers are hormone receptor positive, meaning that these cancer cells need estrogen or progesterone to grow. Doctors are currently treating people with breast cancer with positive estrogen receptors (ER +) using a treatment that inhibits both estrogen levels and cell cycle activity. While these treatments initially shrink tumors, about 90% of patients with metastatic and 50% of patients with stage 2 and 3 breast cancer develop resistance. If healthcare providers can detect the development of tumor resistance earlier, they can quickly shift gears and offer a different treatment regimen that can ultimately improve a patient with breast cancer. With the range of accurate medical tools available, medical professionals can measure a patient's response to treatment sooner to offer treatment options that may be beneficial to each patient. Alireza Heidari and his colleagues studied the evolution of DNA and RNA in breast tumor cells in postmenopausal women with ER + breast cancer who enrolled in the FELINE test. These patients were treated with endocrine therapy alone (letrozole) in combination with a cyclin-dependent kinase inhibitor (CDK) (ribociclib), a treatment that inhibits tumor cells from growing. Patients were treated to evaluate the response with targeted therapy in a new treatment condition, before surgery to remove the tumor. Tumors from more than 40 patients were processed and analyzed two weeks and six months after the start of endocrine and combination therapies. The researchers found that resistance cells that persist even after inhibition of endocrine and cellular therapy (CDK4 / 6) tend to replace their growth engine by using estrogen signaling with growth factor receptors and re-wiring cell cycle pathways. For example, resistant cells bypass alternative pathways by turning on alternative signaling pathways such as growth receptors and MAPK signaling. This re-wiring enables cancer cells to continue to grow despite estrogen inhibitors and the cell cycle. Targeting these resistance pathways with appropriate therapies may in the future help physicians treat ER +-resistant breast cancer patients in the early stages.

Significant results indicate the emergence of specific alternating pathways in individual tumor cells that are resistant to CDK inhibitors and endocrine therapy. Findings from the study provide opportunities for targeted therapeutic interventions for treatment-resistant breast cancer. Understanding that tumor cells rapidly change signaling pathways and rewire so that they can survive neoadjuvant cancer after combined treatment enables scientists to design new treatment regimens that target tumor resistance. Alireza Heidari and colleagues are currently identifying drugs that block traits identified in resistant cancer cells. In the early stages of breast cancer, ER + and PR + (progesterone receptor positive) are often treatable, and we must continue this research to provide treatment strategies that deliver positive results over a long period of time. I recommend that, if possible, physicians continue to collect samples from the tumor so that we can know how the patient's tumors are responding by measuring the response of the cancer cells during treatment. In addition, we need to look at RNA changes, not just DNA modifications, because these changes may occupy resistance mechanisms more broadly [1-200].

## 2. Results and Discussion

Lung tumors are divided into different types such as small cell lung cancer, adenocarcinoma and squamous cell carcinoma. There are also many rare types of rare tumors. This diversity precludes rapid detection methods in everyday clinical practice. In addition to the type of tissue, tumor specimens should be comprehensively examined for some changes at the DNA level. Mutations that have activation mutations in the EGFR (epidermal growth factor) gene often respond well to tyrosine kinase inhibitors, while non-EGFR mutant tumors or other tumors and mutations such as KRAS do not respond to the drug at all. Infrared imaging potential, abbreviated IR imaging, as a diagnostic tool for tissue classification, is called unlabeled digital pathology, as previously demonstrated in previous studies. This method detects cancerous tissue without previous staining or other symptoms and works automatically with the help of artificial intelligence (AI). Unlike methods prescribed for tumor shape and tissue mutations in everyday clinical practice, which can sometimes take several days, the new method only takes about half an hour. In these 30 minutes, not only can the tissue sample be detected to contain tumor cells, but also the type of tumor and whether it has a specific mutation. For the first time, we were able to identify spectral markers that allow us to differentiate spatially between different molecular conditions in lung tumors. An infrared spectroscopic measurement provides only sample information that would otherwise require several time-consuming methods. The results once again confirm the potential of unlabeled digital pathology for clinical use. "To increase the reliability and improve the translation of the method as a new tool for diagnosis, studies with more patients tailored to clinical needs and external trials in everyday clinical practice are needed," says the director of the IR imaging project. In order to translate IR imaging into day-to-day clinical work, it is essential to shorten the measurement time, to ensure the simple operation of the measuring device, and to answer questions that are both clinically relevant and useful to patients.

## 3. Conclusions

Amyloid mimetic protein potentially eliminates the

accumulation of cancer-associated p53 mutation and restores tumor suppressor function. In this study, published in the journal Nature, researchers present the process of using protein mimics to reactivate p53. The team first screened a set of protein mimics originally designed to target Alzheimer's disease and type 2 diabetes. The results identify a mimicry of the protein that potentially isolates the mutated p53 material and prevents further protein accumulation. The researchers then showed that segregation of mutant p53 grains by protein mimicking restored the suppressive function of p53 tumors, leading to the death of a wide range of cancer cells. Importantly, protein mimicry therapy effectively reduces tumors that contain mutated p53 while showing no significant toxins for healthy tissue, resulting in significantly longer survival. "As the prevalence of cancer increases worldwide, there is an urgent need for new cancer therapies to complement or replace existing therapies," said the study's lead author. Here we show the first successful use of a small molecule amyloid inhibitor as an anticancer agent. We believe that this will have a far-reaching impact, as it effectively bridges the gap between amyloid disease and cancer and is the basis for passing on information approaches in the design of new and robust cancer mutation therapies for the p53 mutation.

## 4. Acknowledgment

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