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Nucleic Acids Aptamer Application in Diagnosis and Therapy of Cancer Based on Cell-SELEX Technology

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Abstract

The "labeling" of nucleic acids (cell genetic information such as RNA or DNA) is not a new technology for monitoring them. However, current capabilities do not provide a complete picture of how tumor cells develop. What this platform, known as Clon Mapper, can do that was not possible before, is to go back in time and track how tumor cells change over time. This gives researchers the ability to see which cells "win" over fewer resistant cells, continue to clone themselves, and make the tumor more dangerous. By isolating these cells, researchers can better test which therapies work against them. Monitoring changes over time is the key to successful transmission therapies. Tumor cells adapt and become resistant to therapies, which is why patients can recover but experience a relapse later. This is one of the reasons why cancer treatment is so challenging; we do not have very good methods for early detection of cells sensitive to a drug and measuring their resistance. This resistance is the main cause of treatment failure in many cancer patients. CLL is a low-grade B cancer that is often monitored for months or even years before active treatment is needed. This treatment depends a lot on the patient's close supervision. In this study, Clon Mapper focuses on identifying cells that mimic themselves, the speed at which this process takes place, and its effect on the growth rate of surrounding cells over time. This allows for a more accurate analysis of the cell population and may lead to more customized treatment plans for patients.

Keywords: Cancer; Cells; Tissues; Tumors; Prevention; Prognosis; Diagnosis; Imaging; Screening; Treatment; Management

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Introduction

Angiosarcoma is an ultra-thin tumor that usually develops on the skin and is highly invasive. After metastasis, it is usually treated with chemotherapy, but the cancer's response to it is not long-term and angiosarcoma has a high mortality rate. Separate reports of angiosarcoma tumors responding to immunotherapy have been excluded from other clinical trials, but this marks the first prospective immunotherapy trial in the disease. The drug has been tested in 53 groups of rare cancer patients, but testing is still ongoing in 10 of these groups. Other rare cancers that have been successfully treated in the trial include thyroid tumors, metaplastic breast cancer, and neuroendocrine tumors. The trial has so far involved 773 patients and is approaching its ultimate goal of 818 patients. DC testing is supported by the NCI, led by SWOG, and conducted by the National Network of Clinical Trials (NCTN) with funding from the NIH. Outside of DART, the study of angiosarcoma took years to progress. Adding it to DART helped speed up the achievement of these results. During treatment, the size of the tumor shrank in at least six people, three of whom had criteria known as partial response to treatment, and one had complete response criteria. The other two patients had tumors that were stable after starting treatment. Both patients are still being treated for chronic disease. Among five patients with primary facial or scalp disease, three (60%) recovered with treatment. This is a subset of patients who have very little chance of receiving current treatment. Patients with immunosuppressive inhibitors often experience side effects, and the toxicity in this group of patients is comparable to the toxicity seen in other experiments with ipilimumab and nivolumab in sarcoma. Among these patients, 75% reported treatment-related adverse events and 25% reported treatment-related adverse events of grade 3 or higher [1-490].

Results and Discussion

The enzyme APOBEC3A is a vital part of the innate immune system that protects cells from viral infection by causing mutations and prevents viruses from multiplying; APOBEC3A, on the other hand, increases the level of DNA mutations by directly attacking the cancer cell genome, leading to cancer progression, metastasis, and drug resistance. In previous studies, we have shown that APOBEC3A-induced DNA mutations are very common in cancer patients. In fact, we have found that they are up to 80% present in certain types of cancer, such as lung, breast or bladder cancer. The study, entitled Genotoxic stress and viral infection causes transient mutations in APOBEC3A and proinflammatory genes through two distinct pathways, was published in the journal Nature. In this study, researchers transiently adjusted APOBEC3A for viral infection and genotoxic stress caused by chemotherapy drugs. Their work demonstrates how a viral infection triggers a specific innate immune response to activate APOBEC3A expression in human cells and how it is an important step in eliminating the virus; This study also shows how different chemotherapeutic drugs stimulate APOBEC3A, but increase the cancer invasion through a completely different type of immune response that this time triggers mutations. Our results show different methods for cells to regulate APOBEC3A expression to resolve the different types of stresses that a cell may face. By understanding how APOBEC3A-expressing cancer cells and viral infections are regulated, we are ready to take an important step toward developing new therapeutic strategies to combat cancer and new antiviral therapies. Further studies are needed to develop strategies to prevent APOBEC3A-induced DNA mutations in the cancer genome that increase tumor heterogeneity and increase disease progression and resistance to therapies. In the case of viral infections, the next step is to determine whether certain types of mutations previously

detected in viruses, such as SARS-Cov-2 (Covid-19), are the result of APOBEC3A activity and affect virus replication in cells.

Conclusion

While cancer vaccines are an antitumor treatment option, they should inhibit postoperative recurrence and metastasis by activating the patient's immune system. Therefore, it is important for scientists to find possible ways to train the patient's immune system to find these tumor cells. One option is to use the tumor tissue to make a vaccine for the patient, but because there is little difference between the tumor antigens and the body proteins, the tumor antigens may be considered "native" by the patient's immune system. Membrane-coated hybrid enzymes offer a new vaccine production strategy that simultaneously introduces antigens and adjuvants to the dendritic cell to stimulate tumor-specific immune responses. In mouse tumor models, the researchers found that the strategy protected the mouse by prolonging the survival of tumor-bearing animals and long-term protection against the challenge of tumor recurrence. Thus, this hybrid membrane-based anti-tumor immune system offers a new opportunity to develop personalized cancer vaccines that could target a wide range of tumors in the future.

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