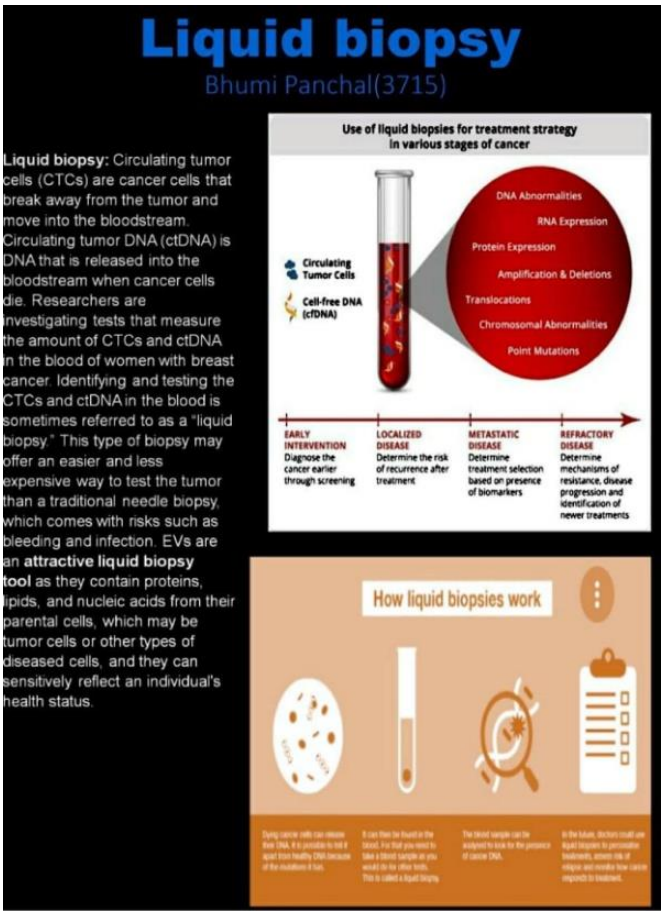
SHAYONA
PRESIDENT SCIENCE COLLEGE

BIOTECHNOLOGY DEPARTMENT
PRESIDENT SCIENCE COLLEGE
DIGITAL POSTER COMPETITION 2021

Our Biotechnology students of semester 5 displayed their thoughts through an array of posters on the recent trends in biotechnological research. Total of 28 participants were there who showcase their talent, among them top 5 best posters were selected portraying their outstanding performance irrespective of their ranks are as follows:

1. **Ms. Bhumi Panchal (3715)- Liquid Biopsy**
2. **Ms. Mansi Kanji (3737)- Therapeutic Protein – FC-fusion protein**
3. **Mr. Suhas Singh Rao (3734)- Nanobiotechnology Endoscopy**
4. **Ms. Zeel Patel (3726)- CAR T- cell Cancer Therapy**
5. **Ms. Bansi Singhala (3732)- Potential Role of Junk DNA in Ageing- Cancer**

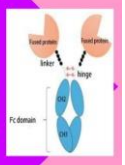
Selected Top 5 Best Posters showing varied research fields of Biotechnology are as follows-



Theraputic Protien

FC-FUSION PROTIEN

- Most protein therapeutics currently on the market are recombinant and hundreds of them are in clinical trials for therapy of cancers, immune disorders, infections, and other diseases.
- New engineered proteins, including bispecific mAbs and multispecific fusion proteins, mAbs conjugated with small molecule drugs, and proteins with optimized pharmacokinetics, are currently under development.
- However, in the last several decades, there are no conceptually new methodological developments comparable, e.g., to genetic engineering leading to the development of recombinant therapeutic proteins.
- It appears that a paradigm change in methodologies and understanding of mechanisms is needed to overcome major challenges, including resistance to therapy, access to targets, complexity of biological systems, and individual variations.



They can be divided into five groups: [a] replacing a protein that is deficient or abnormal

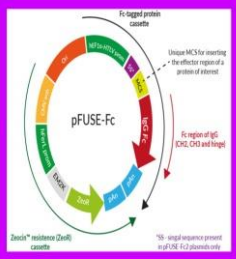
[b] Augmenting an existing pathway;

[c] Providing a novel function or activity;

[d] Interfering with a molecule or organism; and

[e] Delivering other compounds or proteins, such as a radionuclide, cytotoxic drug, or effectors proteins.

They can also be classified based on their molecular mechanism of activity as [a] binding non- covalently to target, e.g. mAbs;[b]affecting covalent bonds;e.g. enzymes; and [c] exerting activity activity without specific interactions, e.g. serum albumin.



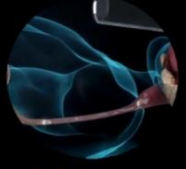
REFERANCE: https://link.springer.com/protocol/10.1007%2F978-1-61779-921-1_1

NAME- SUHAS SINGH RAO ROLL NO.- 3734
SUBJECT- BIOTECHNOLOGY

NANOBIOTECHNOLOGY ENDOSCOPY

- Researchers have now developed a m ultifunctional endoscope-like device, using individual CNTs for prolonged intracellular probing at the single-organelle level, without any recordable disturbance to the m etabolism of the cell

NANO CAPSULE



CAPSULE INSERTION

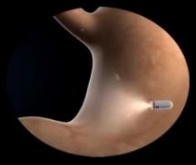
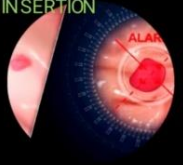
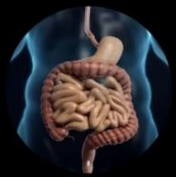


IMAGE CAPTURING



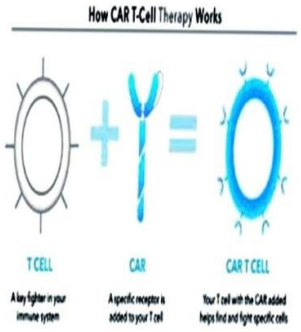
ULCER DETECTION

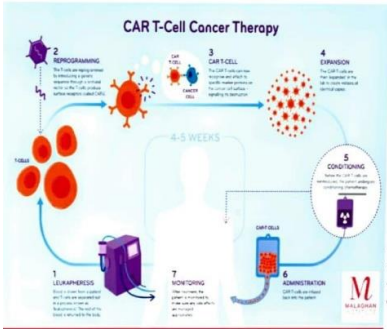
DIAGNOSIS



EXTRACTION OF CAPSULE

AR (for Chimeric Antigen Receptor) T-cell therapy uses specially engineered white blood cells called T cells to lead an assault on cancer. T cells' role in the immune system is to hunt down and destroy abnormal cells, including cancer cells. For a variety of reasons, however, they don't always recognize cancer cells, or don't mount an all-out attack on them, potentially allowing tumors to take root and expand. Turning them into CAR T cells seeks to overcome those deficiencies. To make CAR T cells, technicians collect a sample of a patient's T cells from the blood and engineer them to sprout special structures called chimeric antigen receptors on their surface. When these CAR T cells are reinjected into the patient, the receptors may help the T cells identify and attack cancer cells throughout the body.





SIDE EFFECTS

CAR T-cell therapy can cause cytokine release syndrome (CRS), which can cause dangerously high fevers, extreme fatigue, difficulty breathing, and a sharp drop in blood pressure..

Other general side effects can include:

- •Tremors
- •Headaches
- •Loss of balance
- •Trouble speaking
- •Seizures
- •Sometimes, hallucinations

POTENTIAL ROLE OF JUNK DNA SEQUENCE IN AGEING - CANCER

INTRODUCTION

DNA region known as VNTR2-1 that appears to drive the activity of the telomerase gene, which has been shown to prevent aging in certain types of cells. Knowing how the telomerase gene is regulated and activated and why it is only active in certain cell types could someday be the key to understanding how humans age and how to stop the spread of cancer. The human body is essentially made up of trillions of living cells. It ages as its cells age, which happens when those cells eventually stop replicating and dividing. genes influence how cells age and how long humans live, but how that works exactly remains unclear. Latest finding that VNTR2-1 helps to drive the activity of the telomerase gene is especially notable because of the type of DNA sequence it represents.

METHOD

Recently identified a DNA region known as VNTR2-1 that appears to drive the activity of the telomerase gene, which has been shown to prevent aging in certain type of the cell.The telomerase gene controls the activity of the telomerase enzyme, which helps produce telomeres, the caps at the end of each strand of DNA that protect the chromosomes within our cells. However, in certain cell types -- including reproductive cells and cancer cells -- the activity of the telomerase gene ensures that telomeres are reset to the same length when DNA is copied. This is essentially what restarts the aging clock in new offspring but is also the reason why cancer cells can continue to multiply and form tumorAlmost 50% of our genome consists of repetitive DNA that does not code for protein, "These DNA sequences tend to be considered as 'junk DNA' or dark matters in our genome, and they are difficult to study. Our study describes that one of those units actually has a function in that it enhances the activity of the telomerase genes.

DISCUSSION

Their finding is based on a series of experiments that found that deleting the DNA sequence from cancer cells – both in a human cell line and in mice – caused telomeres to shorten, cells to age, and tumors to stop growing. Subsequently, they conducted a study that looked at the length of the sequence in DNA samples taken from Caucasian and African American centenarians and control participants in the Georgia Centenarian Study, a study that followed a group of people aged 100 or above between 1988 and 2008. The researchers found that the length of the sequence ranged from as short as 53 repeats – or copies – of the DNA to as long as 160 repeats."It veries a lot, and our study actually shows that the telomerase gene is more active in people with a longer sequence."

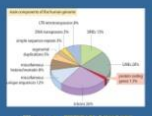


Figure 1. HUMAN GENOME

Figure 2. JUNK DNA

Figure 3. MASSEURO THE FUNCTIONAL GENOME

1. FIGURE 4. TRENDS IN GENETICS

EXPERIMENT

Since very short sequences were found only in African American participants, they looked more closely at that group and found that there were relatively few centenarians with a short VNTR2-1 sequence as compared to control participants. was worth noting that having a shorter sequence does not necessarily mean your lifespan will be shorter, because it means the telomerase gene is less active and your telomere length may be shorter, which could make you less likely to develop cancer. findings are telling us that this VNTR2-1 sequence contributes to the genetic diversity of how we age and how we get cancer. We know that oncogenes – or cancer genes – and tumor suppressor genes don't account for all the reasons why we get cancer.

CONCLUSIONS

Research shows that the picture is a lot more complicated than a mutation of an oncogene and makes a strong case for expanding our research to look more closely at this so-called junk DNA.

REFERENCES

African Americans have been in the United States for generations, many of them have Caucasian ancestors from whom they may have inherited some of this sequence. So as a next step, he and his team hope to be able to study the sequence in an African population.

CONTACT

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