

Detection of *p53* Gene of Colorectal Cancer and Activating it by Stabilizing Dynamic CeRNA

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Description

Colorectal cancer (CRC) is one of the deadly cancers in the world. It stands in the third place with 1,933,600 numbers of patients according to 2020 analysis. There are other names for CRC like colon cancer, bowel cancer, rectal cancer or adenocarcinoma because they emerge from the glandular epithelial cells of large intestine at the colon and rectum.

A person may be affected with CRC due to following reasons

- Family history of CRC
- Genetic disorders
- Certain health conditions like Inflammatory bowel disease
- Type 2 Diabetes

CRC gets spread to some other parts of body by metastasis once the condition becomes severe and then no further medication can help the patient.

A tumour suppressor gene (*TP53*) is usually involved in control of apoptosis and cell cycle. This is located on the short arm of chromosome 17 (17p13.1) through this communication it is explained that how *TP53* gene is targeted by using chemically stabilized endogenous RNA [1].

Oncocers are CeRNAs taking crucial roles in oncogenic pathways processed in many types of cancer, and this study analyzes oncoer-mediated cross-talk by sponging microRNAs (miRNAs) in these pathways. The depot of genomic mutations and epigenetic alterations changing gene function and expression causes cancers [2-3].

Competitive endogenous RNA is a previously considered as junk but after several studies it is discovered that they code for linc RNA. CeRNA is a network of several mRNA's which competes for same pool of micro RNA's. To regulate the ability of miRNA and to inhibit the translation of mRNA into proteins the ceRNA binds to their target mRNA's and acts as miRNA's sponges. This is called as sponging mechanism [4].

The action site of *p53* gene in CRC is first identified and then miRNA's will block translation by binding to mRNA. By using chemically stabilized ceRNA the excess miRNA's are sponged. Since the ceRNA is a complex secondary structure with many loops and stems it is impossible to perform sponging mechanism by itself sometimes. Hence, two strategies are performed to make them able to perform sponging mechanism [5].

Designing ceRNA by chemical biology

A ceRNA has many nucleotides since it is a complex and dynamic structure. In this method the covalent bonds between all the

nucleotides are made stronger. The very first step involves the synthesis of ceRNA by designing artificial nucleotides in the stem of the loop. Later the bonds will be locked by using a light source at a particular wavelength so that they will stay intact as per requirement and do not change. As a result strong covalent bonds are formed [6-7].

Click chemistry reaction

Azide-alkyne cycloaddition which contains diazoles is used to design ceRNA by click chemistry reaction. It is stereospecific and regioselective 5 membered ring structures. To stop the conformational changes in dynamic structure of ceRNA, we will use this heterocyclic ring structure of Azide alkyne and lock the stem region at our required site on the ceRNA.

The R1 and R2 groups on the heterocyclic compound will bind at specific bases and interlock them. After this process, by using synthetic biology a cell is designed with help of genetic circuits to generate the ceRNA at the *TP53* gene site and activate it. The determination of the *TP53* mutation timing for the majority of cancer forms appears to be imprecise and varies widely amongst research, cohorts studied, and techniques of analysis.

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Conflict of Interest

The authors declare that they are no conflict of interest.

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