

POSTER TITLE
APPLICATION OF CRISPR–CAS9 TECHNOLOGY IN THE TREATMENT OF CHRONIC LYMPHOCYTIC LEUKEMIA WITH MUTANT P53 GENE

aurelianu2007@yahoo.com

Aurelian Udriștioiu, Saint Ecaterina Laboratory of Medical Analyses, Associate Professor in Titu Maiorescu University of Bucharest, Faculty of Medicine, Assistance General Care, City Târgu-Jiu, District Gorj, Romania. -Member of American Association for Cancer Research, (AACR), Current Emeritus Member. Member Correspondent in American Romanian Academy of Art and Sciences, (ARA), Manole Cojocaru, Titu Maiorescu University of Bucharest, Faculty of Medicine, Romanian Academy of Sientist, (AOSR)

INTRODUCTION

The status of P53 suppressor gene, from double strands DNA, can be tested in cooperation with partners at IARC (International Agency for Research on Cancer).

In solid malignant tumors, the P53 gene is mutated or deleted in approximately 50% of all tumors, but in Chronic Lymphocytic Leukemia, (CLL), P53 gene aberrations are ranging from 5-10% at the time of diagnosis

AIM

This study looks at using CRISPR–Cas9 gene-editing technology to fix mutations in the white blood cells of patients with CLL with mutant P53 Gene. It’s a promising new way to treat this complex disease.

METHOD

CRISPR–Cas9 works like a GPS for DNA. It uses a guide RNA (gRNA) to lead a specific enzyme to a target spot in the genetic code. Once there, the enzyme cuts the DNA to disable or fix the TP53 gene. This guide is usually made of two parts (crRNA and tracrRNA), but researchers found they can combine them into one single, efficient "chimeric" RNA molecule.

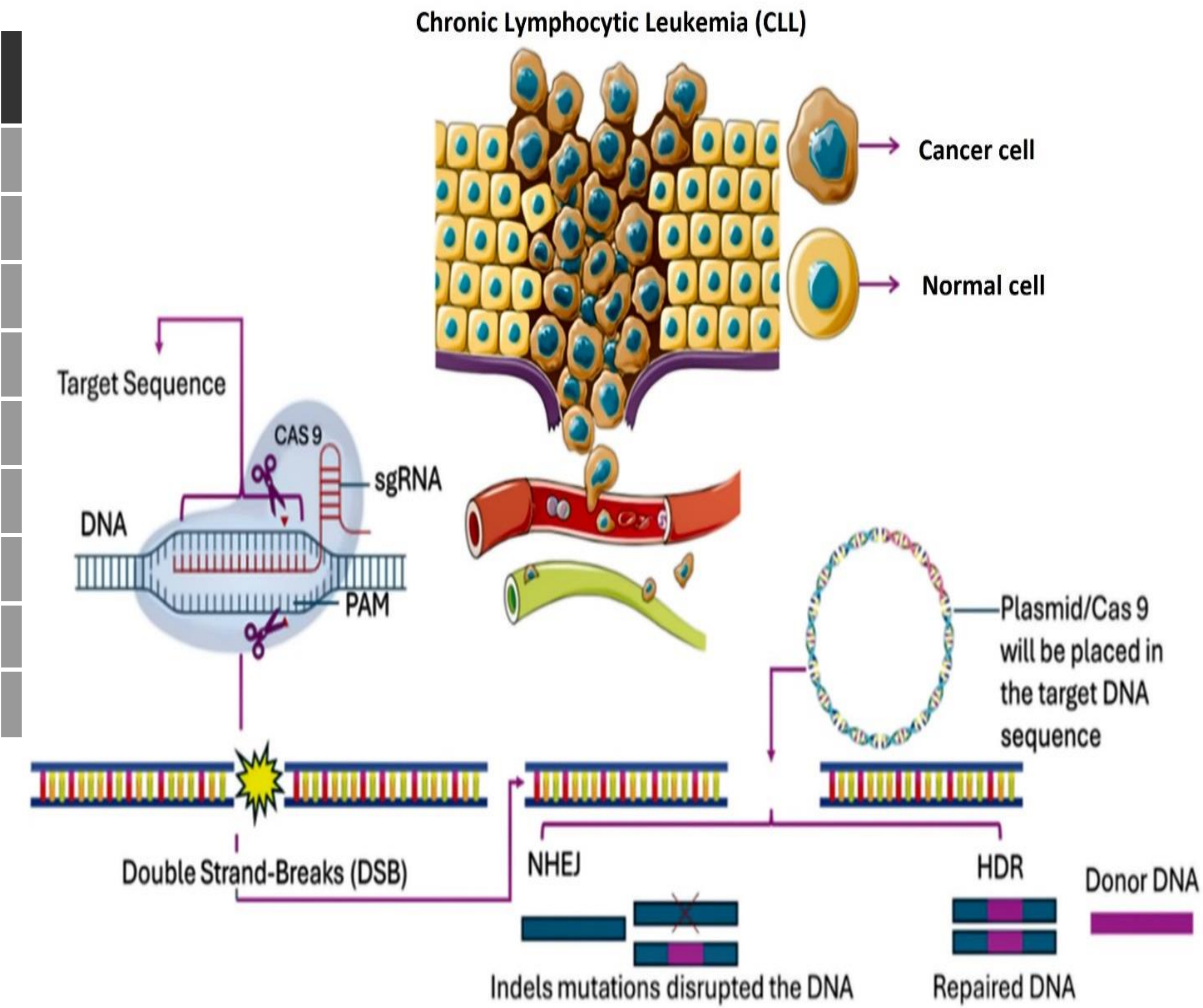
CONCLUSIONS

Basically, CRISPR–Cas9 acts like "molecular scissors," allowing scientists to cut, paste, or delete DNA. It’s a game changer for the future of genetic medicine. This study demonstrates that technology CRISPR–Cas9 gene editing holds significant promise as a therapeutic strategy for TP53-mutant CLL by enabling precise gene correction of functional replacement.

RESULTS

While the natural two-part system works well, using a single-guide system is much easier for programming DNA edits.

The system is very precis and it only cuts if it finds a specific "docking code" (called a PAM) next to the target. By creating these precise breaks, we hope to replace the mutated genes in CLL cells with healthy ones.



Contact information

Dr. Aurelian Udriștioiu, MD/PhD,
 -Primary Physician of Laboratory Medicine, Department of Hematology, Associate University Professor in Titu Maiorescu University of Bucharest, Faculty of General Medical Care, City Targu Jiu, Romania. E-mail: aurelianu2007@yahoo.com

REFERENCES

1. Lander Eric S (2016) The heroes of CRISPR. Cell 164(1):18–28.
 2. Aurelian Udristioiu, Ioan Ovidiu Gheorghe and Delia Nica-Badea. Book, Isoform p53 Protein's Major Role in the Pathophysiology of Malignant Hematologic Diseases, Chapter three, Structure of p53 protein; British Library. ISBN (10): 1-5275-7243-9; ISBN (13): 978-1-5275-7243-9, House Cambridge Publishing Group, 2024, UK.
 3. Udristioiu A, Liviu M, Cojocaru M et al. Application of CRISPR–Cas9 technology in the treatment of chronic lymphocytic leukemia with TP53 mutations. Oncolog-Hematolog ro. 2026; 74: 40-50, ISSN 2066-8716; SSN 2066-8260, ISSN-L 2066-8260.