



Targeting Mitochondrial Respiration to Overcome Therapy Resistance Acute Myeloid Leukemia

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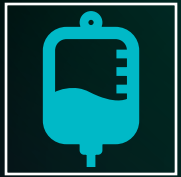
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Introduction

- Acute Myeloid Leukemia (AML) – A type of blood cancer
 - Due to undeveloped white blood cells produced in the bone marrow
- As for right now the frontline cure Chemotherapy achieves high remission rates but 70-80% relapse or wouldn't not respond the the initial therapy.

Current AML Therapy



Chemotherapy



Consolidation
Therapy



Supportive
Care



Stem Cell
Transplantation



**Targeted
Therapy**

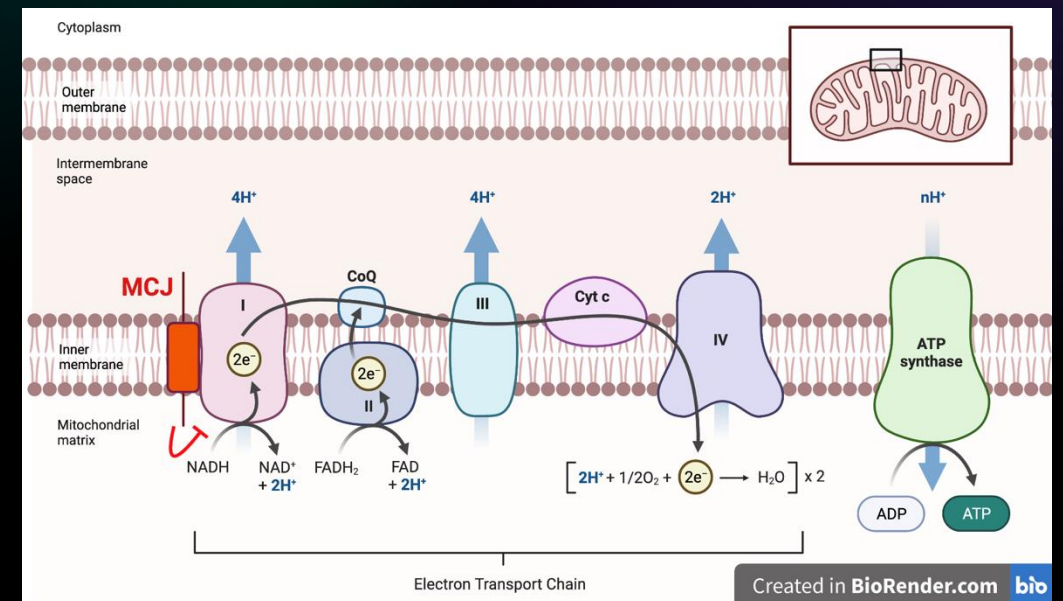
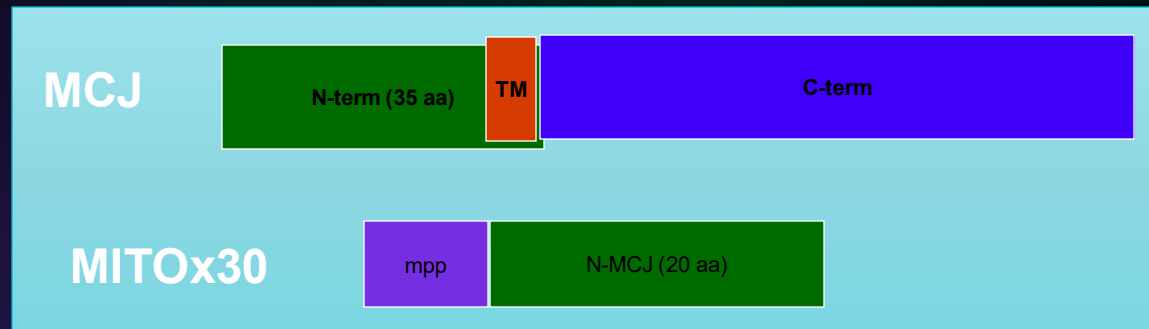
The background of the slide features a complex chemical structure diagram. It includes several fused and linked rings, with various atoms labeled (C, H, Cl, N, O). A prominent label 'meta attack' with an arrow points to a specific reaction site on a benzene ring. Other labels like 'CH3', 'NH2', and 'H' are also visible. The diagram is rendered in a light blue/teal color against a white background, which is then overlaid on a dark teal gradient on the right side of the slide.

Introduction

- For this research 2 type of drugs are used to see its reaction
 - Venetoclax + Azacitidine (VenAza)
 - MCJ Peptide (MITOx30)

Introduction

- Methylation- controlled J protein (MCJ) – made by DNAJC 15 gene
 - MCJ acts as a “break” it slows down mitochondrial respiration , when MCJ is missing it could cause the resistance of chemotherapy

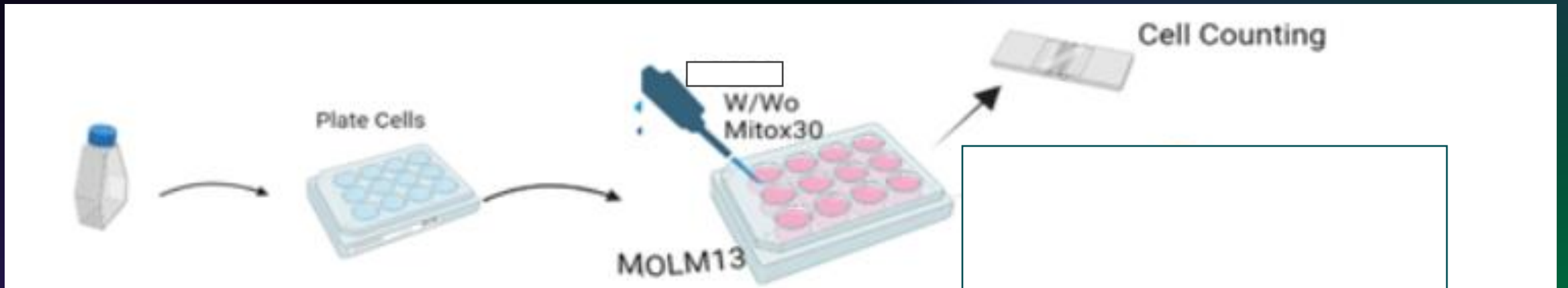


Hypothesis and objective:

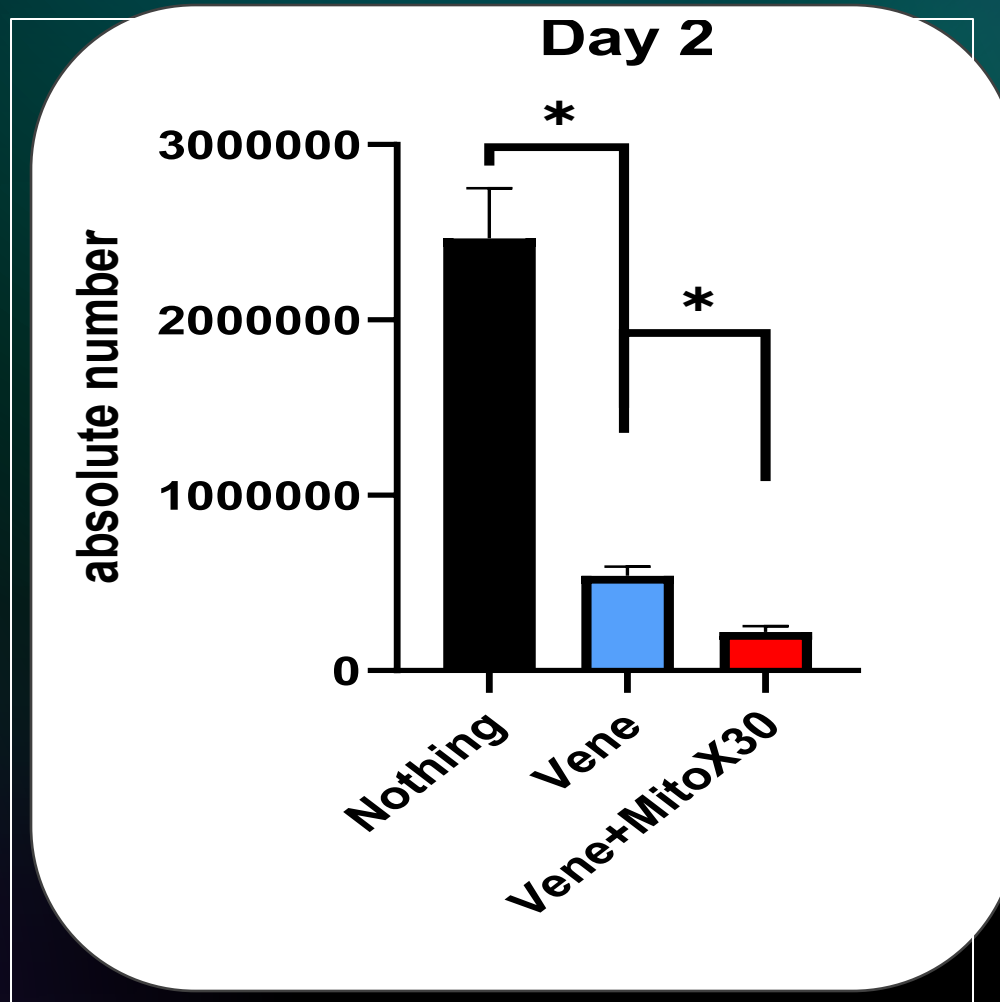
- Hypothesis: The integration of **MCJ mimetics** will restore MCJ function and augment the effectiveness of the chemotherapeutic drugs, such as Venetoclax and Azacitidine.
- Aim 1: Investigate the effect of MCJ peptide **(Mitox30)** when combined with Venetoclax + Azacitidine (VenAza).

Methods

- For this research two type of cells
 - Primary and MOLM-13 cells
- Primary Cell
 - Comes directly from patients who are resistant
- Molm-13 Cells
 - The MOLM-13 cell line is a human acute myeloid leukemia (AML) cell line that was initially established in 1986 by the Rolf Marschalek lab.

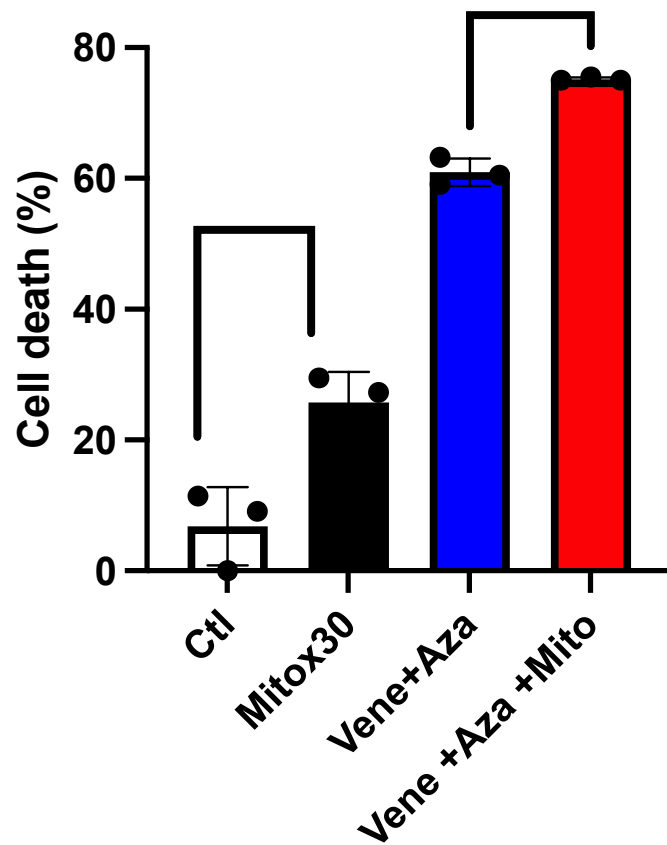


MCJ peptide (mitox30) and Venetoclax synergy increases MOLM-13 Cell Death

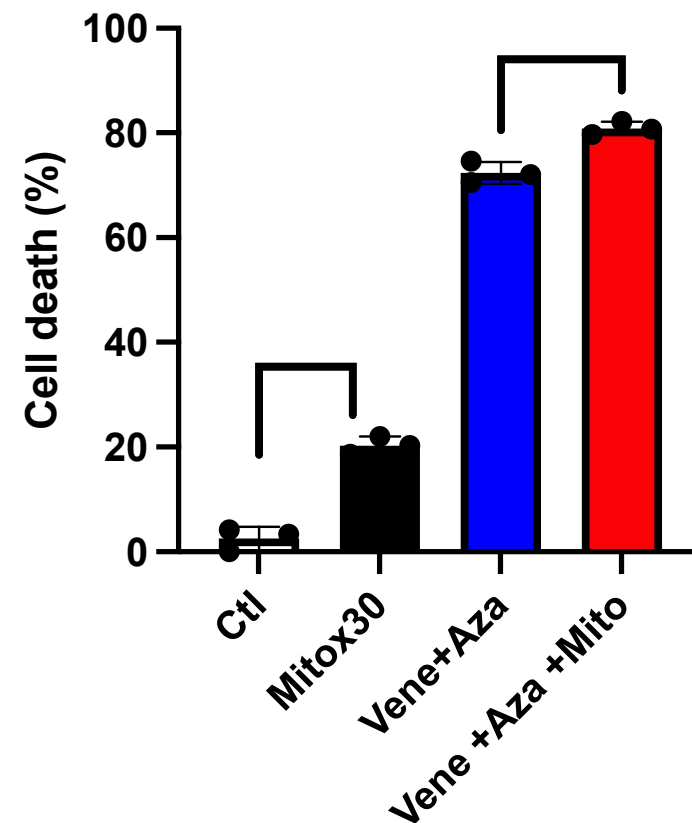


MCJ peptide (MitoX30) increases the response to VenAza in the primary AML cells

1032- 48h



1032- 72h



Limitations

Heterogeneity of AML

Bone Marrow Microenvironment

Clonal Evolution

Immunological Evasion

Chemotherapy Resistance

Targeted Therapy Resistance



Cultural Exchange

Language is very similar

Similar cultural Experience

Education Very different from here

Tried Japanese food

Thank
You!!!!!!

- Mercedes Rincon
- Felipe Pereira
- Daniela Ortiz-Chavez
- Fahima Abdullahi
- Qian Fang
- Maureen Hoen
- Cristina Cenciarelli