**Introduction**

- **Normal cells**
  - Reproduce themselves only when and where they are needed.
  - Stick together in the right place in the body or freely floating.
  - Self destruct if they are damaged or too old.
  - Become specialized (mature).
  - Continuously grow and divide (No G0 Phase).
  - Ignore chemical signals from other cells.
  - Don’t need to stick together-ability to spread throughout body.
  - No specialization, repair or death of cancer cells.

**Cytarabine as Chemotherapy**

- Also known as cytosine arabinoside or Ara-C.
- Anti-metabolites mask themselves as adenine or pyrimidine - which become the building blocks of DNA [2].
- Cytarabine specifically affects the “S” phase of the cell cycle where DNA is synthesized.
- Clinical trials have shown complete remission rates of 50–60% and overall survival rates of 30–40% among (AML) patients who use Cytarabine.
- Effects both normal and cancerous cells, resulting in side effects like: hair loss, mouth ulcers, nausea & vomiting, etc…

**Mechanism of Action of Ara-CTP with DNA**

- Active form: Ara-C triphosphate.
- This active form competes with deoxycytidine triphosphate to integrate into DNA. Integration into cell results in cell death.
- Cytarabine alters the sugar component of DNA by using an arabinose instead of ribose.
- This modified sugar hinders the rotation of the molecule, stopping which stops replication of DNA. [2]

**Future Treatment of Cytarabine**

**CPX-351**

Celator Pharmaceuticals released Phase 2 induction response data which states:
- CPX-351 is a special formulation of Cytarabine and Daunorubicin designed to treat high risk elderly patients with acute myeloid leukemia.
- This drug showed a higher chance of remission and survival than the standardized "3+7" strategy.
- Drug’s main purpose is to create an option for elderly patients with AML who typically have very few options.

**Mode of Action:**
- Forms a complex with DNA by inserting itself between base pairs
- It inhibits enzyme topoisomerase II- relaxes DNA supercoils for transcription
- Prevents double helix from being reformed, stopping replication.

**Background: Acute Myeloid Leukemia**

- AML develops in white blood cells, specifically Myeloid WBC’s, and starts in the bone marrow [7].
- Diagnosed by a CBC or a Blood Smear to check for mitotic division, quality, and type of cells present.
- Mutations of the NPM1 account for 30% of AML cases.
- Chromosomal Location: 5q35.1
- NPM1: instruction for making a protein called nucleophosmin. [5]
- Nucleophosmin: attaches to another protein called ARF; a tumor suppressor. [6]

**Literature Cited**


