# New Therapies for Leukemia

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# Objectives

- Classify types of anticancer treatments
- Define and classify new approvals 2017-2018
  - Acute Myeloid Leukemia (AML)
  - Chronic Myeloid Leukemia (CML)
  - Acute Lymphoblastic Leukemia (ALL)
  - Chronic Lymphocytic Leukemia (CLL)
- Identify current place in therapy for new treatments
- Understand general administration characteristics and common toxicities

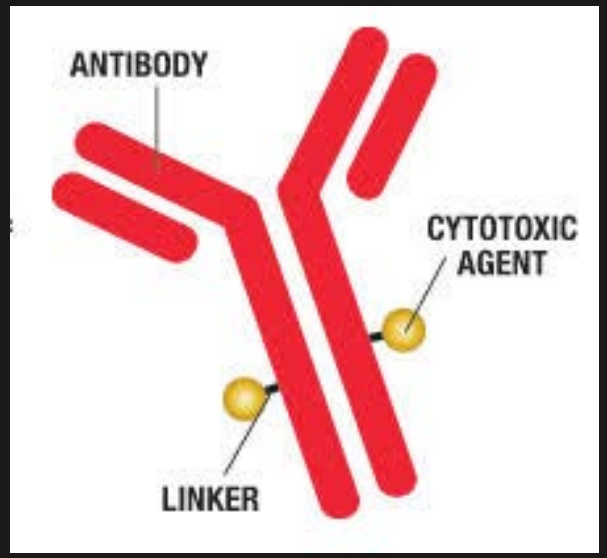
# Treatment Classification Review

| Type of therapy | Characteristics | Action |
|---|---|---|
| Traditional cytotoxic chemotherapy | Widespread action and toxicities | Within cells; prevents division and reproduction |
| Small molecule inhibitors | Inhibit enzymatic activity of specific proteins | Within cells; targeted |
| Antibodies | Proteins targeting specific antigens | Extracellular |
| Antibody conjugates | Targeted activity with selective cytotoxicity | Extracellular target; complex internalized |

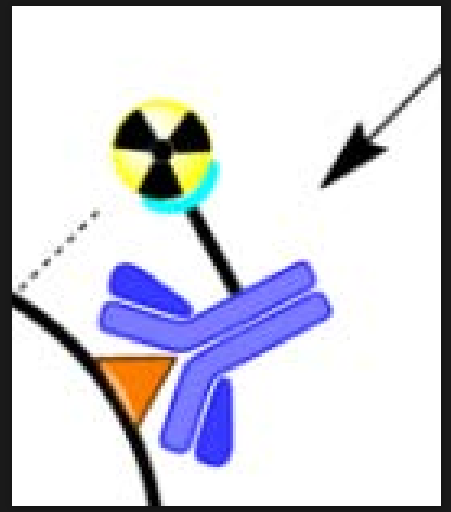
# Treatment Classification Review

| Type of therapy | Delivery | Naming convention |
|---|---|---|
| Traditional cytotoxic chemotherapy | Any; PO, IV, IM, subQ | Varies |
| Small molecule inhibitors | Usually PO, may be injected | End in “-ib” |
| Antibodies | Injected, usually IV, possibly subQ | End in “-mab” |
| Antibody conjugates | IV | Contain “-mab”; followed by cytotoxic agent name |

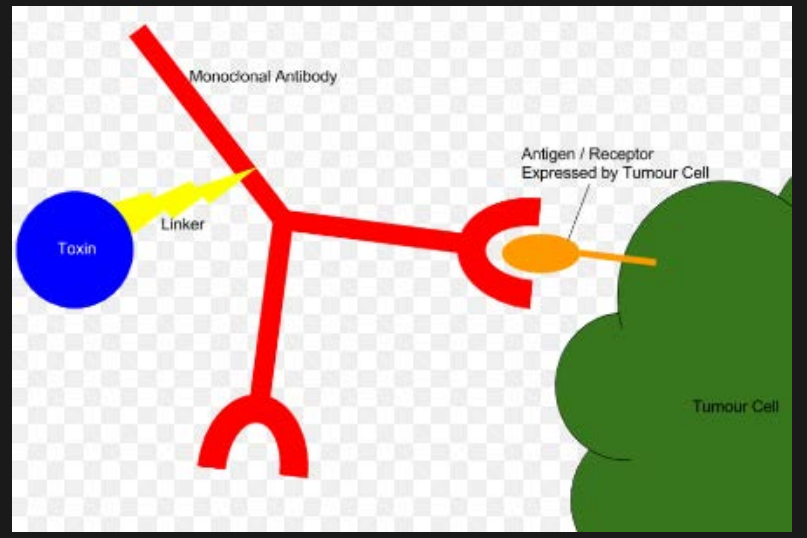
# Treatment Classification Review




Antibody Drug Conjugate



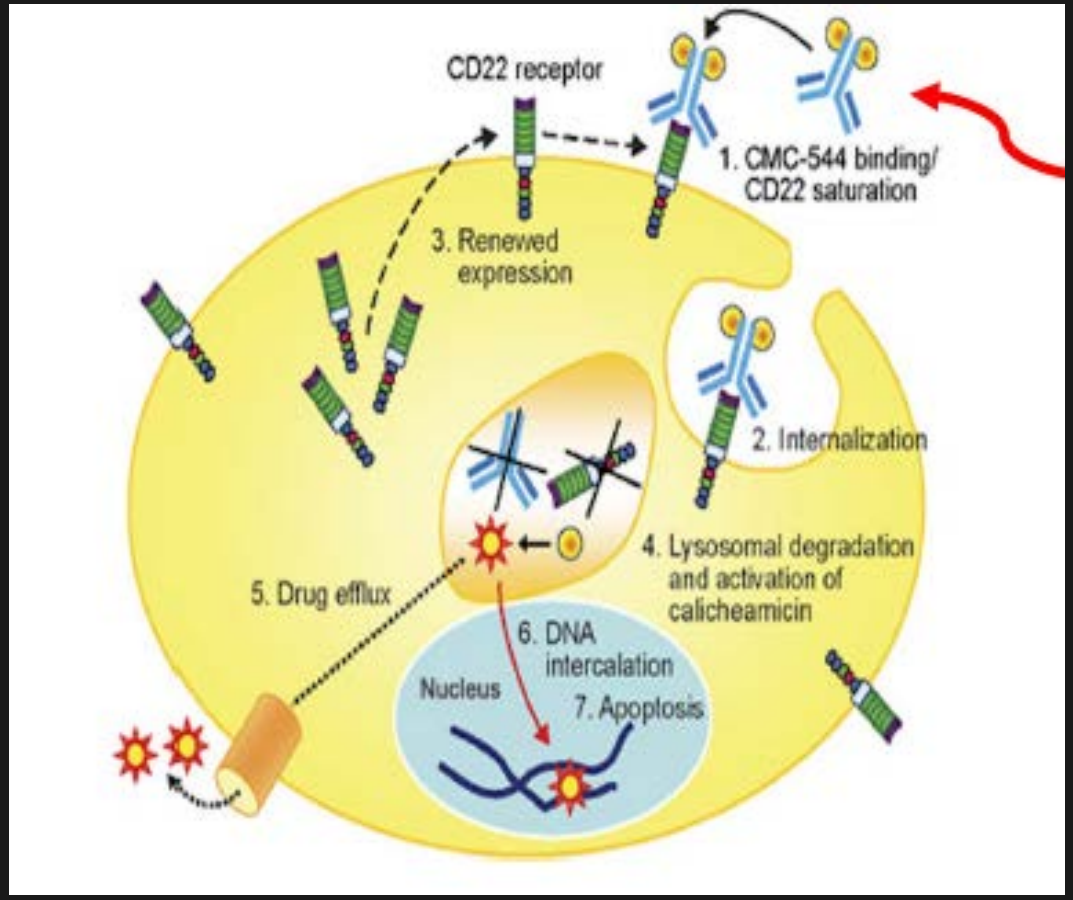
Antibody Radionuclide Conjugate




Antibody Exotoxin Conjugate

# Treatment Classification Review

- Mechanism for antibody conjugates
  - Extracellular receptor targeted
  - Taken up by endocytosis
  - Lysosomal degradation
  - Cell death
- Potential toxicity from drug efflux of cytotoxic agent



# Overview

## New approvals/uses

### AML

- midostaurin
- enasidenib
- liposomal daunorubicin and cytarabine combination
- gemtuzumab ozogamicin
- ivosidenib

### ALL

- inotuzumab ozogamicin
- blinatumomab
- tisagenlecleucel (CAR-T cells)

### CML

- nilotinib

### CLL

- rituximab and hyaluronidase
- venetoclax

# AML

## Standard treatment review

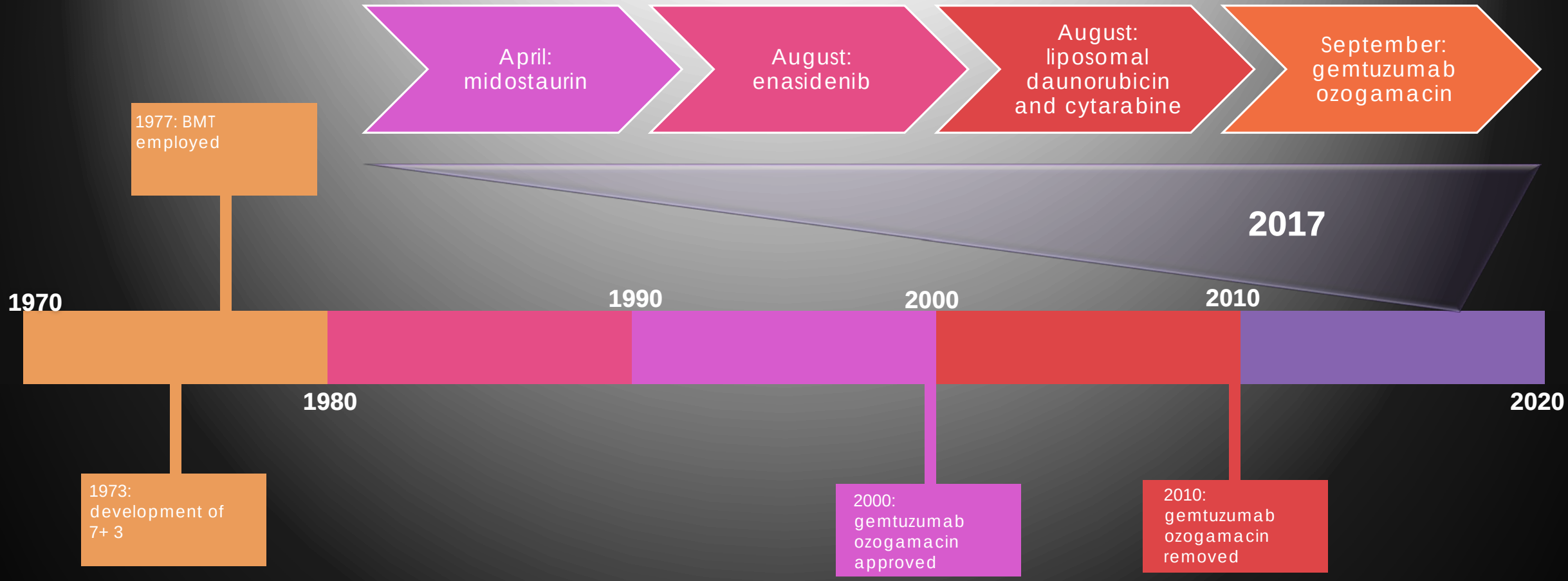
### Standard treatment

- Intensive chemotherapy:
  - Induction
    - 7 + 3
  - Consolidation
    - High dose chemo
    - BMT
- Salvage
- HMAs; azacitidine, decitabine

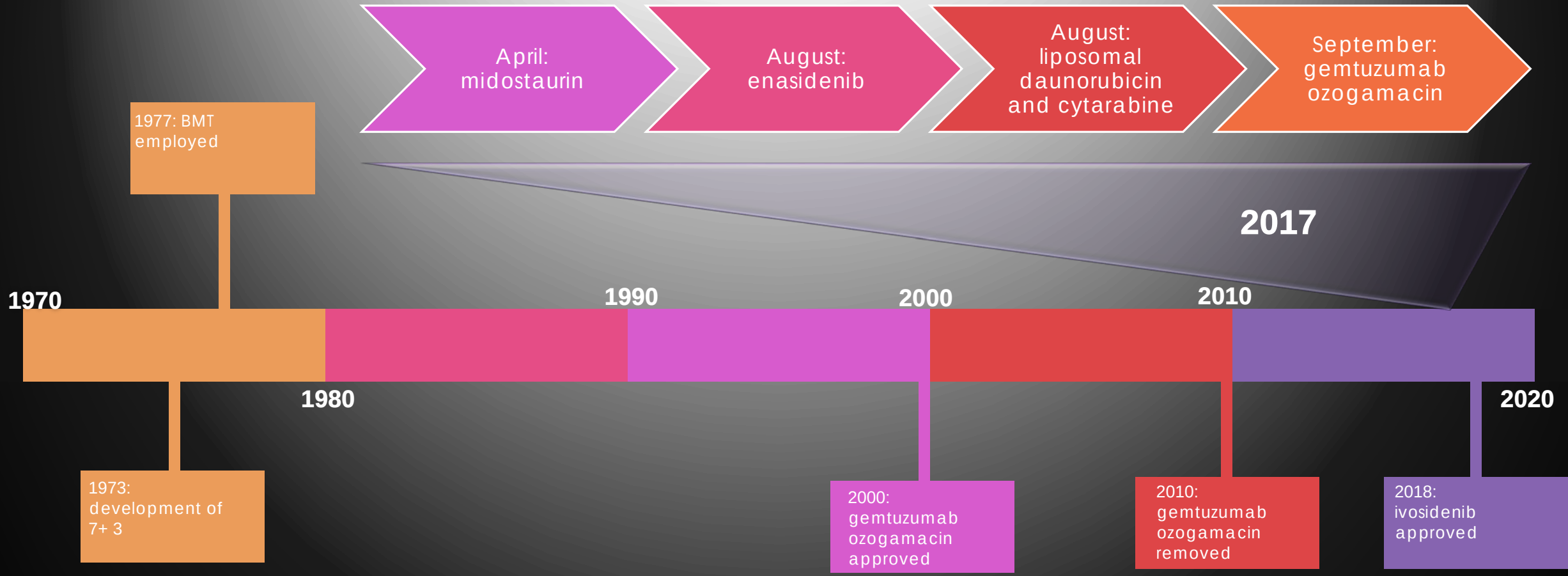
### Update to treatment options

- midostaurin
- enasidenib
- liposomal daunorubicin and cytarabine combination
- gemtuzumab ozogamicin
- ivosidenib

# Landmark Year for AML: 2017



# Landmark Year for AML: 2017



# AML

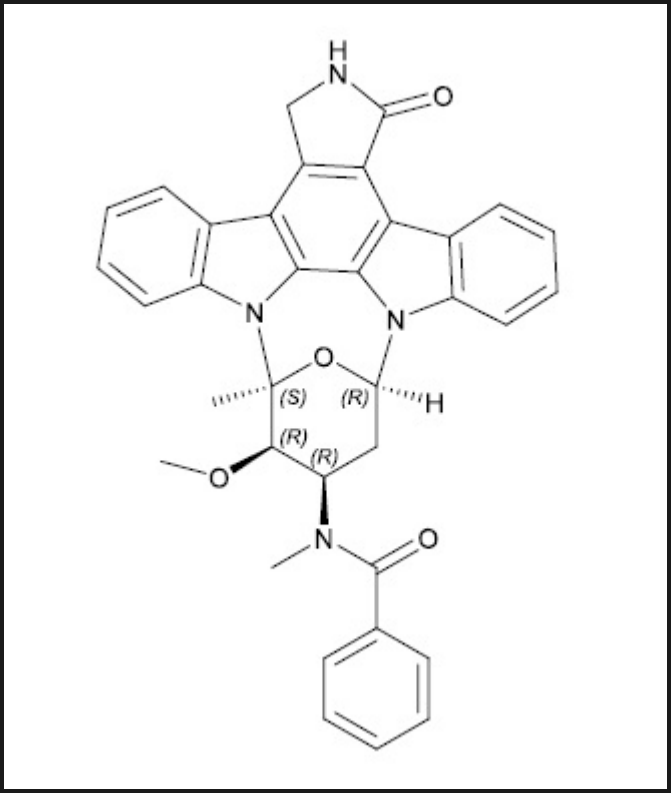
## New application of class (FLT-3 inhibitor)

- FLT-3 is a transmembrane tyrosine kinase receptor
  - Mutation in FLT-3 is known to confer poorer prognosis in AML
    - Internal tandem duplication (FLT-3 ITD)
    - Point mutation of the tyrosine kinase domain (FLT-3 TKD)
- Mechanism is to prevent FLT-3 signaling

# AML

## Midostaurin

- Midostaurin
- First approved FLT-3 inhibitor for AML
  - Small molecule multi kinase inhibitor
- More FLT-3 inhibitors are pending FDA approval



# AML

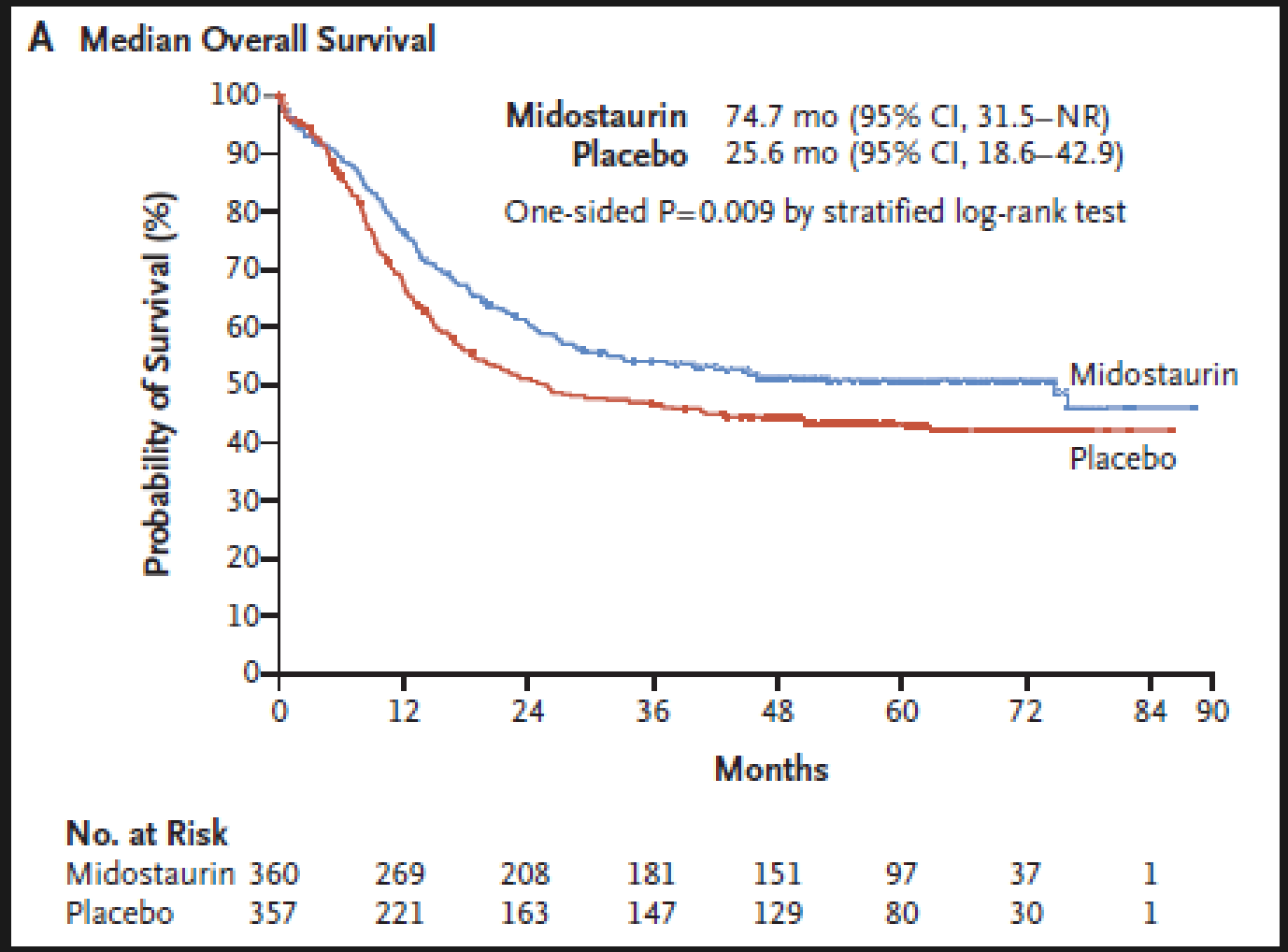
## Midostaurin

Approval was based on phase III study

Midostaurin plus chemotherapy (7+3) in previously untreated FLT3 positive AML

Hazard ratio

0.78 95% CI(0.63-0.96)




Stone RM, et al. **N Engl J Med 2017;377:454-64.**

# AML

## Midostaurin

- May be used in combination with standard 7+3 for newly diagnosed FLT3 positive AML

| Drug | Dose | Route | Schedule | Toxicities | Other |
|---|---|---|---|---|---|
| midostaurin | 50 mg BID | PO | Days 8-21 of induction and consolidation cycles | ▪ GI toxicity<br>▪ Pulmonary toxicity<br>▪ May cause QT prolongation | ▪ Potential drug interactions<br>• Antifungals<br>▪ Logistical challenges |

# AML

## New class (isocitrate dehydrogenase inhibitor)

- IDH performs an enzymatic step for myeloid differentiation
  - Mutated IDH produces an oncometabolite 2-HG
    - 2-HG blocks differentiation
- Oral small molecule inhibitor of the IDH protein
  - Mechanism is to inhibit mutant IDH variants
    - Allows for differentiation without cytotoxicity

# AML
## Enasidenib

- Enasidenib
- First in class
  - Inhibits the IDH2 protein
    - Mutant IDH2 is inhibited at much lower concentrations than the wild type enzyme
    - Intracellular levels of 2-HG are reduced in blood samples of patients with IDH2 mutated AML
- Accelerated approval is for use in r/r AML with IDH2 mutation
- Approval is based on phase 1/2 (non-randomized) data

# AML

## Enasidenib

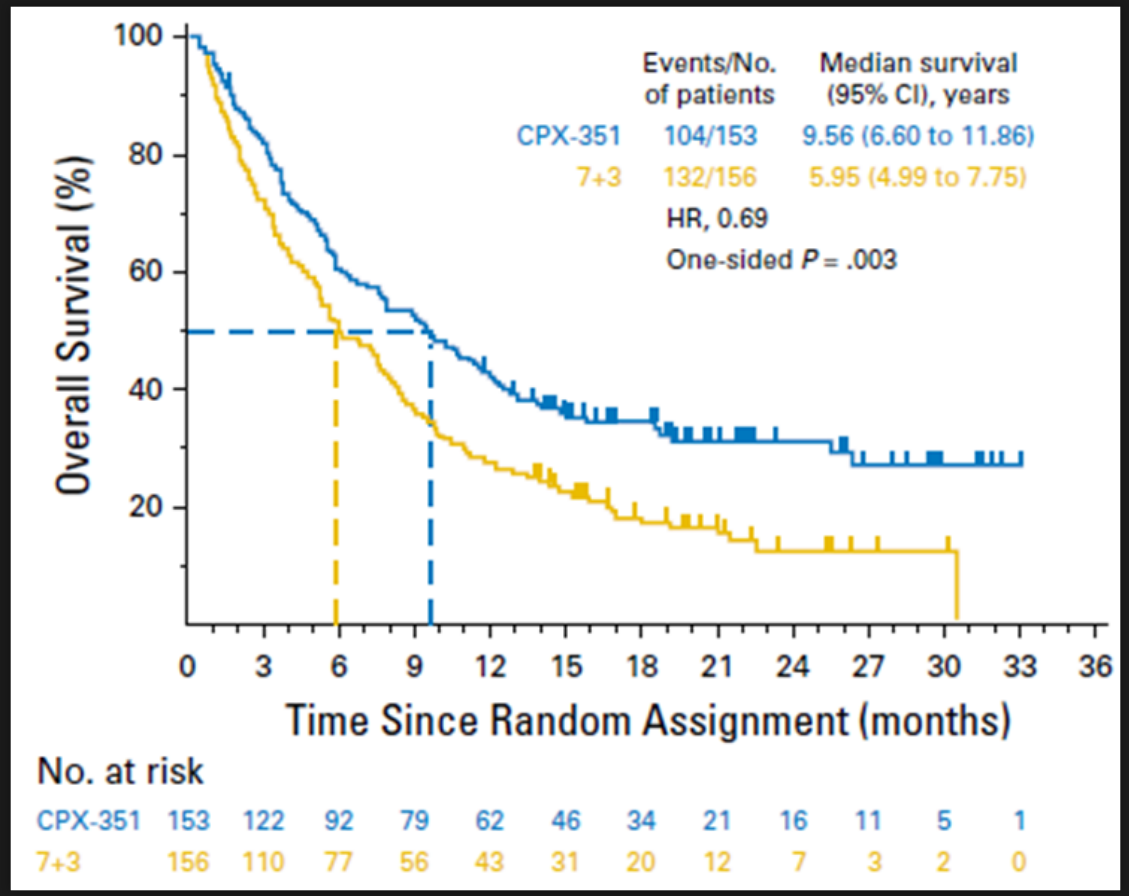
- FDA approved for use in IDH2 mutated relapsed or refractory disease

| Drug | Dose | Route | Schedule | Toxicities | Other |
|---|---|---|---|---|---|
| enasidenib | 100 mg daily | PO | Until progression | ▪ Differentiation syndrome<br>▪ Leukocytosis<br>▪ Electrolyte imbalance<br>▪ GI disturbance<br>▪ Hepatotoxicity<br>▪ TLS | ▪ Response may be delayed |

# AML

## Liposomal daunorubicin and cytarabine

- liposomal daunorubicin and cytarabine
  - Dual drug liposomal encapsulation of cytarabine and daunorubicin
  - Fixed ratio of 1:5 molar equivalents for maximum synergy
  - Better uptake by leukemia cells




Lancet JE, et al. **JCO. 2018;** https://doi.org/10.1200/JCO.2017.77.6112.

# AML

## Liposomal daunorubicin and cytarabine administration

- May be used in therapy related AML or AML with myelodysplastic features

| Drug | Dose | Route | Schedule | Toxicities | Other |
|---|---|---|---|---|---|
| liposomal daunorubicin and cytarabine (CPX-351) | 44 mg/m2 daunorubicin and 100 mg/m2 cytarabine (liposomal) IV over 90 minutes on days 1, 3, 5 of induction<br><br>29 mg/m2 daunorubicin and 65 mg/m2 cytarabine (liposomal) IV over 90 minutes on days 1 and 3 of consolidation | | | ▪ Typical for 7+3<br>▪ Extended hematologic toxicity<br>• Bleeding<br>• CNS<br>▪ Theoretical copper toxicity | ▪ Not interchangeable with standard daunorubicin and cytarabine<br>▪ Opportunity for outpatient administration? |

# AML

## Gemtuzumab ozogamicin

- Gemtuzumab ozogamicin
  - Antibody targets CD33; delivers ozogamicin
- History
  - Approved based on phase II data in 2000
  - Post marketing data could not confirm benefit
  - Concerning high rate of treatment related mortality
    - Removed from market in 2010

WSJ

HEALTH

**Pfizer to Withdraw a Cancer Drug Mylotarg**

*By Jennifer Corbett Dooren*

Updated June 22, 2010 12:01 a.m. ET

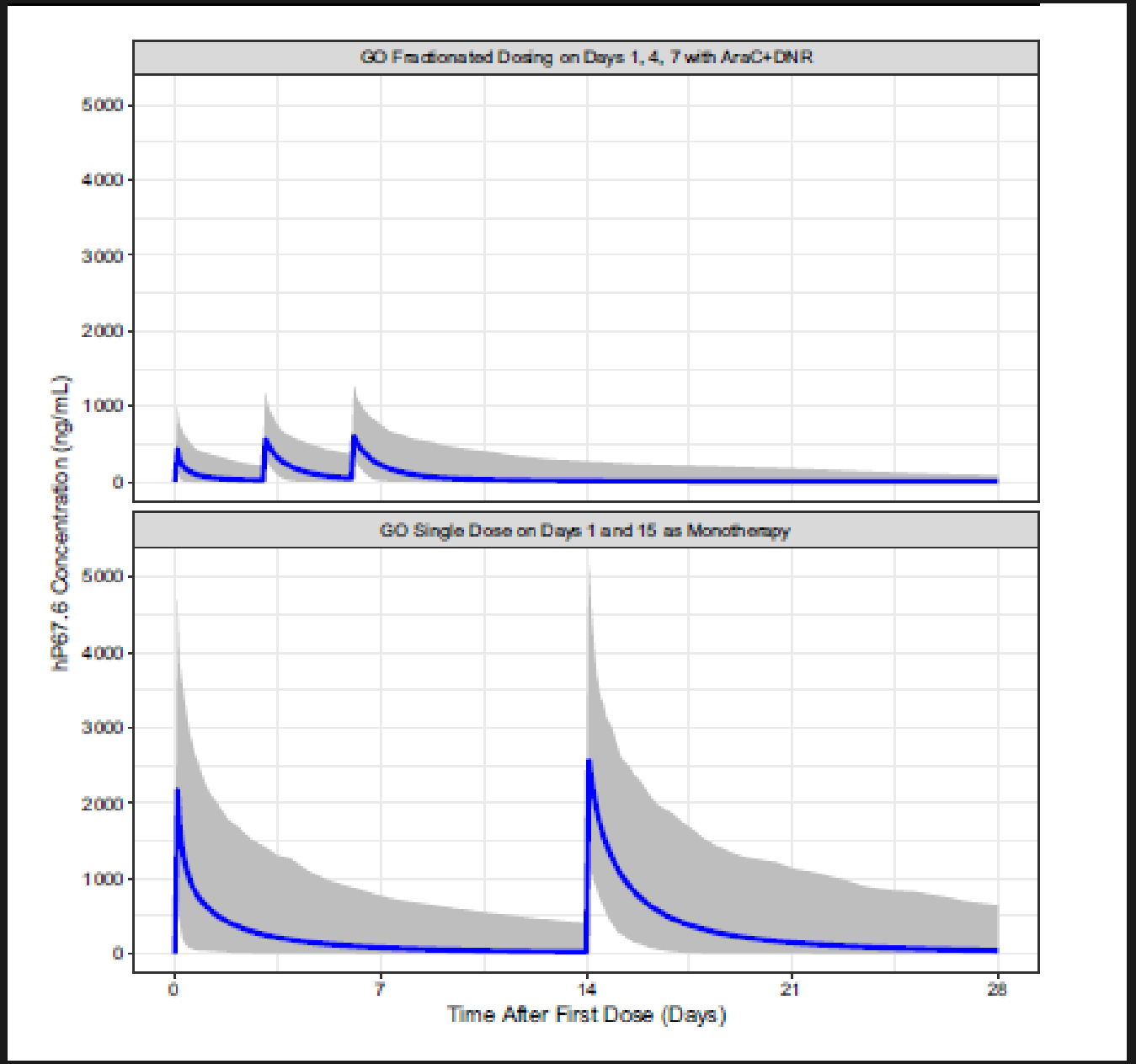
WASHINGTON—The Food and Drug Administration said Monday Pfizer Inc. PFE 0.59% ▲ is withdrawing its cancer drug Mylotarg from the U.S. market after a clinical study showed the drug wasn't effective and had more safety problems.

# AML
# Gemtuzumab ozogamicin

Subsequent trials evaluated different dosing schemes

Fractionated dosing

- Smaller doses
- More frequent

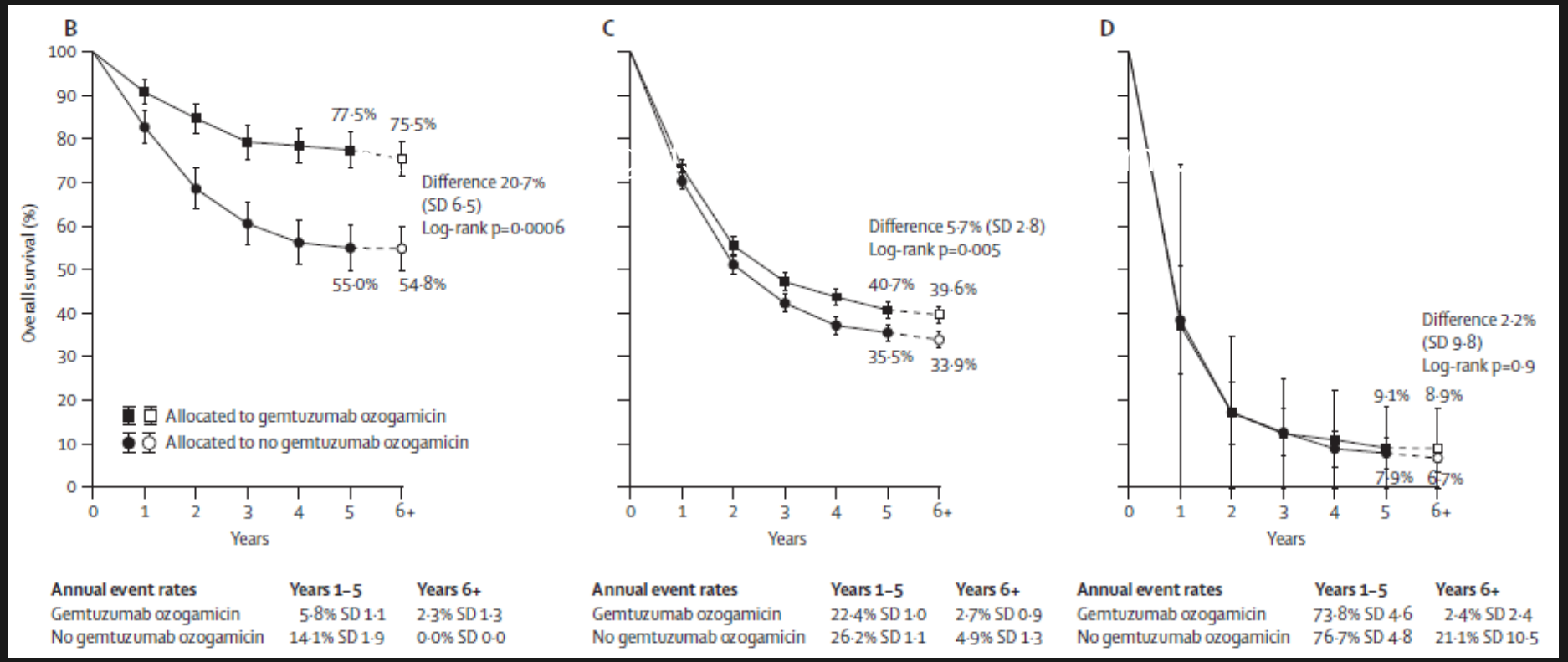



Hibma J, Knight, B. **Clinical Pharmacokinetics 2018;** https://doi.org/10.1007/s40262-018-0699-5

# AML

## Gemtuzumab ozogamicin

2014 meta-analysis evaluating GO in combination with chemotherapy




| Annual event rates | Years 1–5 | Years 6+ |
|---|---|---|
| Gemtuzumab ozogamicin | 5·8% SD 1·1 | 2·3% SD 1·3 |
| No gemtuzumab ozogamicin | 14·1% SD 1·9 | 0·0% SD 0·0 |

| Annual event rates | Years 1–5 | Years 6+ |
|---|---|---|
| Gemtuzumab ozogamicin | 22·4% SD 1·0 | 2·7% SD 0·9 |
| No gemtuzumab ozogamicin | 26·2% SD 1·1 | 4·9% SD 1·3 |

| Annual event rates | Years 1–5 | Years 6+ |
|---|---|---|
| Gemtuzumab ozogamicin | 73·8% SD 4·6 | 2·4% SD 2·4 |
| No gemtuzumab ozogamicin | 76·7% SD 4·8 | 21·1% SD 10·5 |

Hills RK, et al. Lancet Oncology 2014;15:986-996.

# AML

## Gemtuzumab ozogamicin

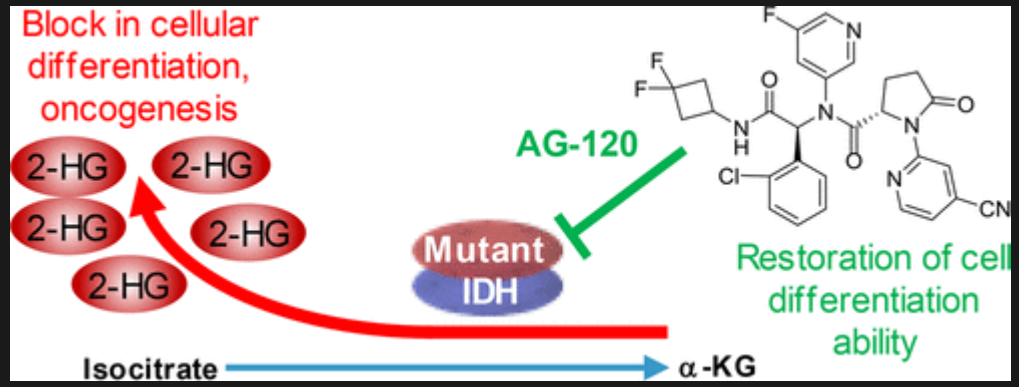
- May be used in CD33 positive AML in combination with 7+3 or as monotherapy

| Drug | Dose | Route | Schedule | Toxicities | Other |
|---|---|---|---|---|---|
| gemtuzumab ozogamacin | Combination: 3 mg/m2 (max 4.5 mg) IV over 2 hours on days 1, 4 and 7 of induction; 3 mg/m2 IV on day 1 of 2 consolidation cycles<br><br>Monotherapy: 6 mg/m2 IV on day 1 and 3 mg/m2 IV on day 8 of induction; 2 mg/m2 IV every 4 weeks x 8 doses | | | ▪ Hepatotoxicity; veno-occlusive disease (sinusoidal obstruction syndrome)<br>▪ Myelosuppression<br>▪ Potential QT prolongation<br>▪ Hemorrhage | ▪ Premedicate for infusion reaction<br>▪ Potential outpatient treatment? |

# AML

## Ivosidenib

- Ivosidenib
- First in class (inhibits IDH1 protein)
- Developed for use in AML with IDH1 mutation
- Approval is based on phase 1/2 (non-randomized) data



# AML
## Ivosidenib

- FDA approved for use in IDH1 mutated relapsed or refractory disease

| Drug | Dose | Route | Schedule | Toxicities | Other |
|---|---|---|---|---|---|
| ivosidenib | 500 mg daily | PO | Until progression | ▪ Differentiation syndrome<br>▪ Leukocytosis<br>▪ Prolonged QT<br>▪ GI disturbance<br>▪ Hepatotoxicity<br>▪ TLS | ▪ Response may be delayed<br>▪ Drug interactions |

# CML

## Standard treatment review

### Standard treatment

- Chronic phase
- Tyrosine kinase inhibitors
  - imatinib
  - dasatinib
  - nilotinib
  - bosutinib
  - ponatanib

### Update to treatment options

- nilotinib
  - FDA approved labeling for potential discontinuation

# CML

## TKIs

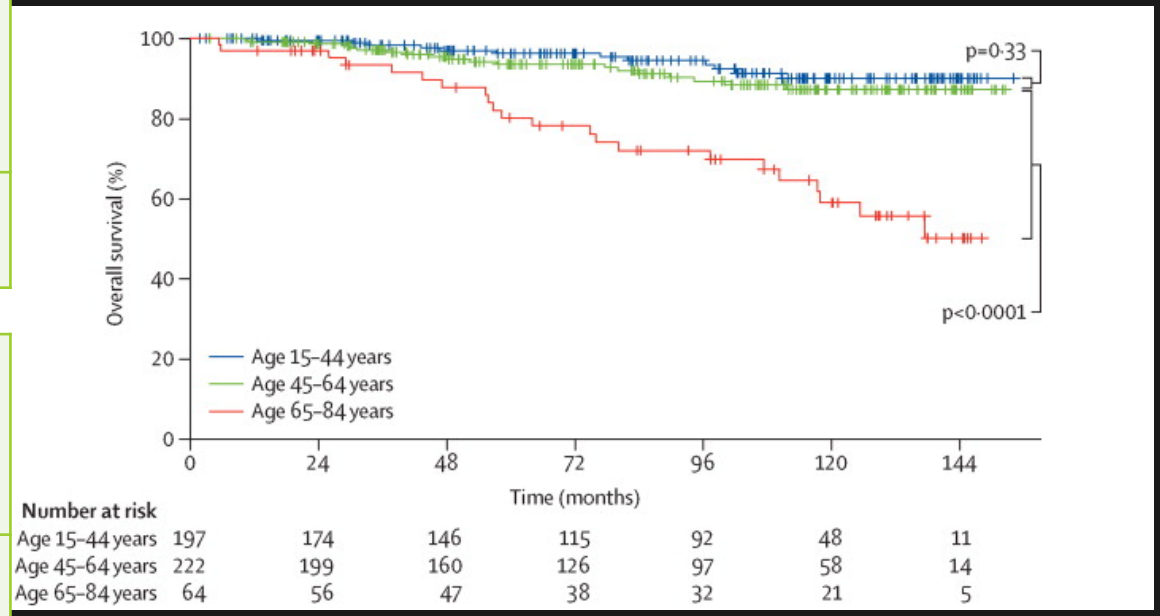
- The Philadelphia chromosome associated tyrosine kinase was first inhibited clinically with a small molecule compound – imatinib
- "2nd generation" tyrosine kinase inhibitors were then developed and provided alternatives when variants of the BCR-ABL gene demonstrated resistance

# CML

## TKIs

| 5-year absolute OS (95% CI); 312 assessed | 5-year relative OS (95% CI) | 5-year OS in general population (95% CI) |
|---|---|---|
| 92·7% (90·1–95·3) | 94·7% (92·1–97·4) | 97·8% (97·6–98·2) |

| 10-year absolute OS (95% CI); 127 assessed | 10-year relative OS (95% CI) | 10-year OS in general population (95% CI) |
|---|---|---|
| 83·5% (79·2–87·9) | 88·2% (83·7–92·9) | 94·6% (94·2–95·4) |




Sasaki K, et al. **Lancet Haematol 2015;2: e186–93.**

# CML

## TKIs

- Improved overall survival changes the focus of management
  - Agent selection is important
    - Consider adverse effect profile and patient comorbidities
    - Resistance
  - Long term management strategies should emphasize adherence
  - Teratogenicity
  - Trials have investigated some discontinuation strategies

# CML

## TKI discontinuation

As of 2017; NCCN has developed criteria for potential discontinuation. Outside of a clinical trial, TKI discontinuation should be considered only if ALL of the criteria included in the list below are met.

- Chronic phase; no history of accelerated or blast phase
- Previously quantifiable BCR-ABL1 transcript
- Stable MR; greater than 2 years
- Access to reliable qPCR with results available within 2 weeks
- Monthly initial molecular monitoring
- Ability to restart TKI within 4 weeks of loss of response
- Consultation with a CML specialty center

NCCN Guidelines for Chronic Myeloid Leukemia. Version 4.2018.

# CML

## Nilotinib

- May be used as initial treatment for newly diagnosed CML chronic phase

| Drug | Dose | Route | Schedule | Toxicities | Other |
|---|---|---|---|---|---|
| nilotinib | 400 mg BID | PO | Until progression; may consider d/c after 3 years in certain circumstances | ▪ Bone marrow suppression<br>▪ Fluid retention<br>▪ QT prolongation<br>▪ Electrolyte imbalance<br>▪ Vascular disease | ▪ May be used for some mutations demonstrating resistance to TKIs |

# ALL

## Standard treatment review

### Standard treatment

- Induction
  - Vincristine, anthracycline, cyclophosphamide, corticosteroids and pegaspargase
  - Hyper CVAD
  - +/- TKI
- Consolidation
  - Multi-agent chemotherapy, BMT
- Maintenance
- CNS prophylaxis throughout
- Relapsed/refractory
  - Blinatumomab

### Update to treatment options

- Inotuzumab ozogamicin
- Blinatumomab
- Tisagenlecleucel (CAR-T cells)

# ALL
## Inotuzumab ozogamacin

- Inotuzumab ozogamicin
  - Developed to target cell surface glycoprotein CD22 (90% of B cell ALL)
  - Antibody targets CD22 expressing leukemic cells; delivers ozogamicin
- InO was associated with better remission rates and when compared with SOC in patients with relapsed disease

# ALL

## Inotuzumab ozogamacin

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

### Inotuzumab Ozogamicin versus Standard Therapy for Acute Lymphoblastic Leukemia

Hagop M. Kantarjian, M.D., Daniel J. DeAngelo, M.D., Ph.D., Matthias Stelljes, M.D., Giovanni Martinelli, M.D., Michaela Liedtke, M.D., Wendy Stock, M.D., Nicola Gökbuget, M.D., Susan O'Brien, M.D., Kongming Wang, Ph.D., Tao Wang, Ph.D., M. Luisa Paccagnella, Ph.D., Barbara Sleight, M.D., Erik Vandendries, M.D., Ph.D., and Anjali S. Advani, M.D.

| End Point | Inotuzumab Ozogamicin Group | | Standard-Therapy Group | | Between-Group Difference (97.5% CI) | P Value† |
|---|---|---|---|---|---|---|
| | *no./total no.* | *% (95% CI)* | *no./total no.* | *% (95% CI)* | *percentage points* | |
| Complete remission or complete remission with incomplete hematologic recovery | | | | | | |
| Total | 88/109 | 80.7 (72.1–87.7) | 32/109 | 29.4 (21.0–38.8) | 51.4 (38.4–64.3) | <0.001 |
| Bone marrow blast results below threshold for minimal residual disease | 69/88 | 78.4 (68.4–86.5) | 9/32 | 28.1 (13.7–46.7) | 50.3 (29.9–70.6) | <0.001 |

Kantarjian HM, et al. N Engl J Med 2016;375:740-53.

# ALL

## Inotuzumab ozogamacin

The NEW ENGLAND JOURNAL of MEDICINE

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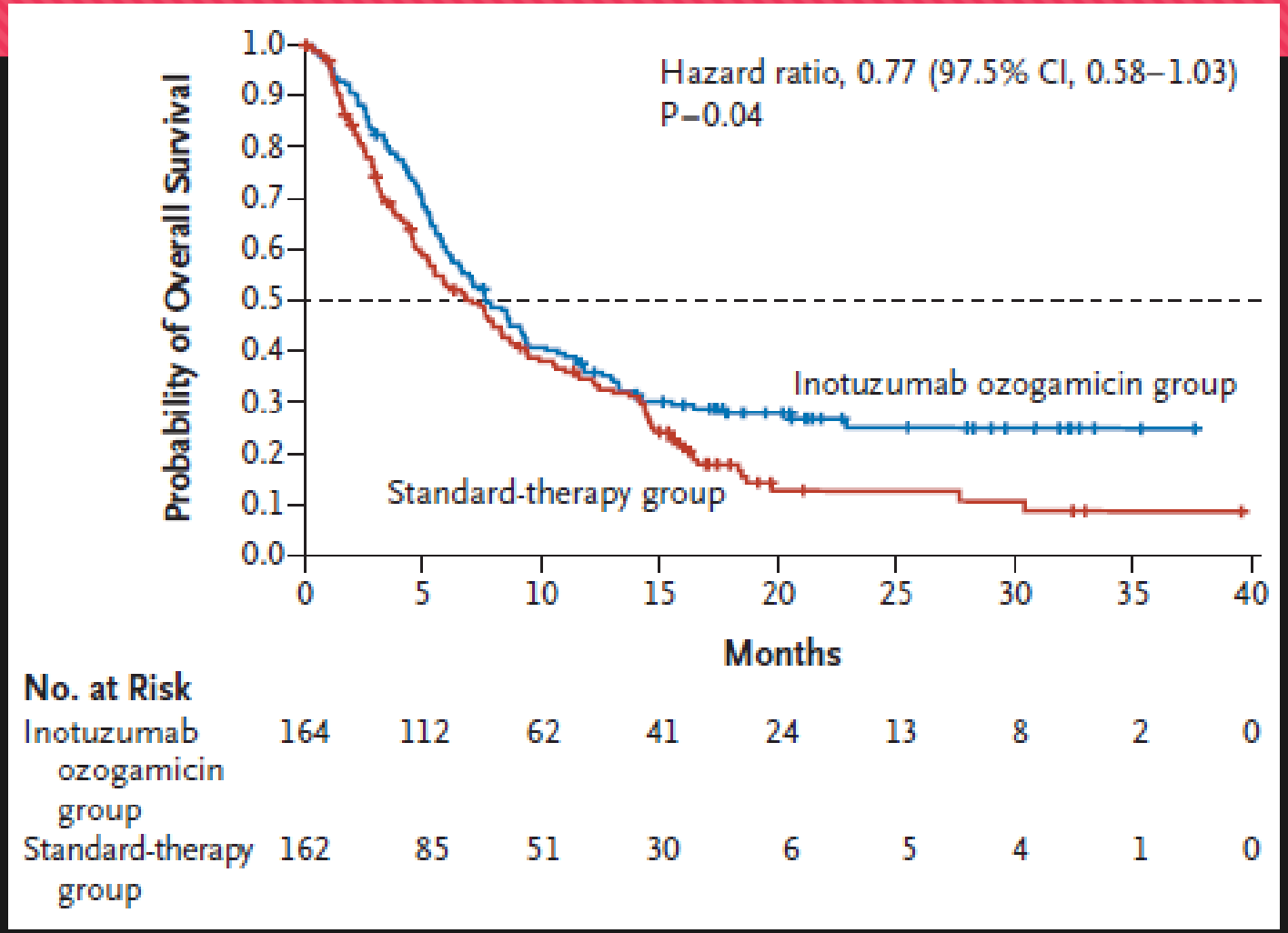
Kantarjian HM, et al. N Engl J Med 2016;375:740-53.

# ALL

## Inotuzumab ozogamacin

- Median OS 7.7 months for InO (95% CI, 6.0 to 9.2) versus 6.7 months for SOC (95% CI, 4.9 to 8.3)




| No. at Risk | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| Inotuzumab ozogamicin group | 164 | 112 | 62 | 41 | 24 | 13 | 8 | 2 | 0 |
| Standard-therapy group | 162 | 85 | 51 | 30 | 6 | 5 | 4 | 1 | 0 |

Kantarjian HM, et al. N Engl J Med 2016;375:740-53.

# ALL

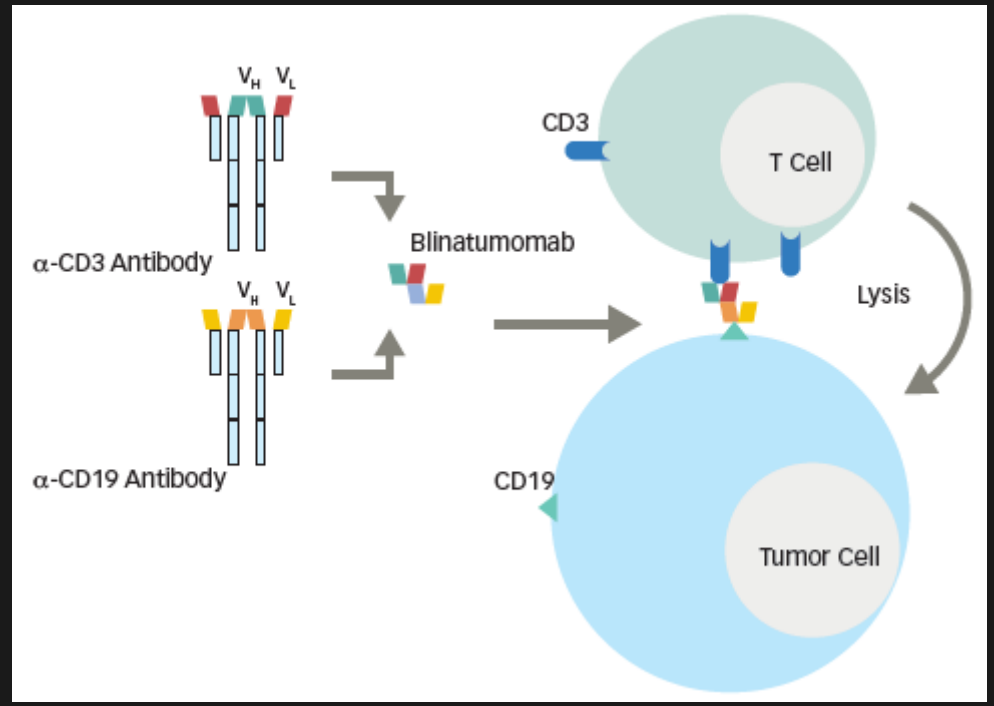
## Inotuzumab ozogamacin

- May be used in relapsed/refractory B cell ALL

| Drug | Dose | Route | Schedule | Toxicities | Other |
|---|---|---|---|---|---|
| inotuzumab ozogamacin | Induction: 0.8 mg/m2 IV on day 1, followed by 0.5 mg/m2 on days 8 and 15 of a 21 day cycle (may repeat as a 28 day induction cycle)<br><br>Subsequent cycles with a complete response: 0.5 mg/m2 IV on days 1, 8 and 15 of a 28 day cycle (up to 6 cycles for those not proceeding to HCT) | | | ▪ Hepatotoxicity; veno-occlusive disease (sinusoidal obstruction syndrome)<br>▪ Myelosuppression<br>▪ Potential QT prolongation<br>▪ Hemorrhage | ▪ Premedicate for infusion reaction<br>▪ Potential outpatient treatment? |

# ALL
## Blinatumomab

- Blinatumomab
  - Bi-specific T cell engager (BiTE)
  - Linker between T cells and tumor cells expressing CD19
- Previously approved for relapsed/refractory ALL
  - New use is for first remission with minimal residual disease positivity



# ALL

## Blinatumomab

- May be used following first induction for previously untreated ALL with minimal residual disease or for relapsed or refractory ALL

| Drug | Dose | Route | Schedule | Toxicities | Other |
|---|---|---|---|---|---|
| blinatumomab | IV continuous infusion<br><br>Induction cycle: 9 mcg daily on days 1-7, followed by 28 mcg daily on days 8-28 of a 6 week treatment cycle<br><br>Subsequent cycles: 28 mcg daily on days 1-28 of 6 or 12 week treatment cycles | | | ▪ Cytokine release syndrome<br>▪ Neurotoxicity | ▪ Admission required for start of each cycle<br>▪ Special warning for preparation and administration errors |

# ALL

## Tisagenlecleucel CAR-T cells

- CAR-T cells
  - Lymphodepleting chemotherapy followed by autologous tisagenlecleucel infusion
    - T cells are removed and modified in the lab for the addition of a chimeric antigen receptor (CAR)
    - T cells are re-infused and recognize cells expressing CD19 to eliminate tumor cells
- Who can get this treatment?
  - B cell ALL in second or greater relapse, not eligible for transplant
  - Age < 26
  - Not required to be in remission
- Major toxicities
  - Cytokine release syndrome
  - Neurotoxicity

# ALL

## Cytokine release syndrome

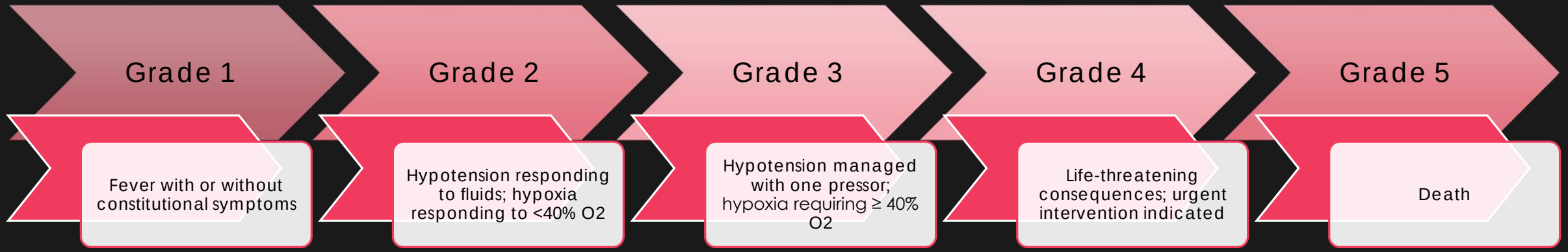
- Cytokine release syndrome is an immunologic reaction
- Significant (temporary) inflammatory cytokine production
  - IL-6, IL-10 and INF-γ
- Fever, hypotension, respiratory failure, coagulopathies
  - May progress to multi-organ failure and death
- Management
  - Stop infusion
  - Corticosteroids
  - Tocilizumab

# ALL

## Cytokine release syndrome

### Common Terminology Criteria for Adverse Events

**Definition:** A disorder characterized by fever, tachypnea, headache, tachycardia, hypotension, rash, and/or hypoxia caused by the release of cytokines.




CTCAE v5.0 – November 27, 2017 . Accessed April 5, 2018.

# CLL

## Standard treatment review

### Standard treatment

- First line regimens
  - fludarabine, cyclophosphamide and rituximab (FCR)
  - bendamustine with CD20 monoclonal antibody
  - chlorambucil with CD20 monoclonal antibody
  - ibrutinib
- Relapsed/refractory
  - venetoclax
  - idelalisib +/- rituximab

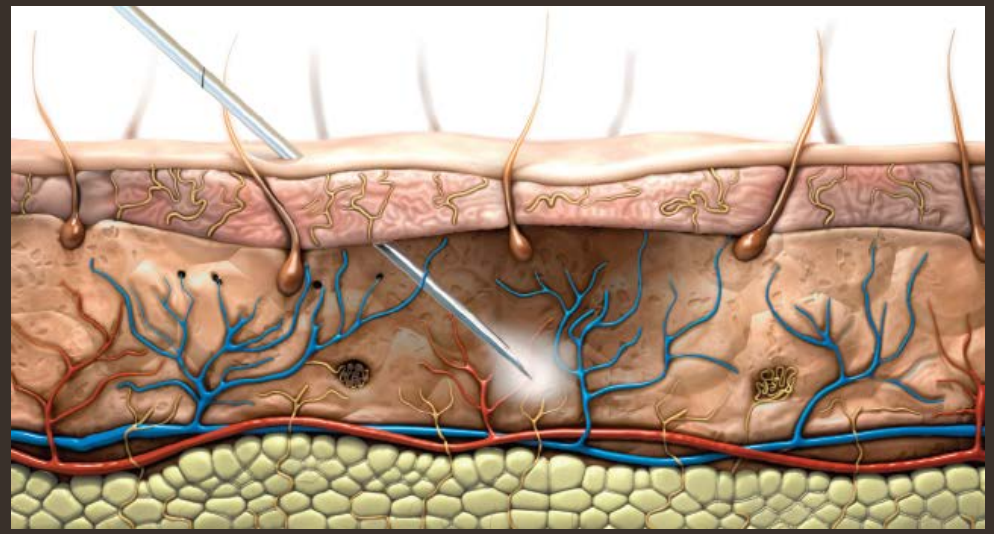
### Update to treatment options

- New formulation of rituximab
  - rituximab and hyaluronidase
- venetoclax (with rituximab)

# CLL

## Rituximab and hyaluronidase

- Rituximab and hyaluronidase for subcutaneous administration
  - Aims to increase patient satisfaction
  - Efficacy and safety appear comparable
  - Uses hyaluronidase
    - Facilitates absorption



# CLL

## Rituximab and hyaluronidase

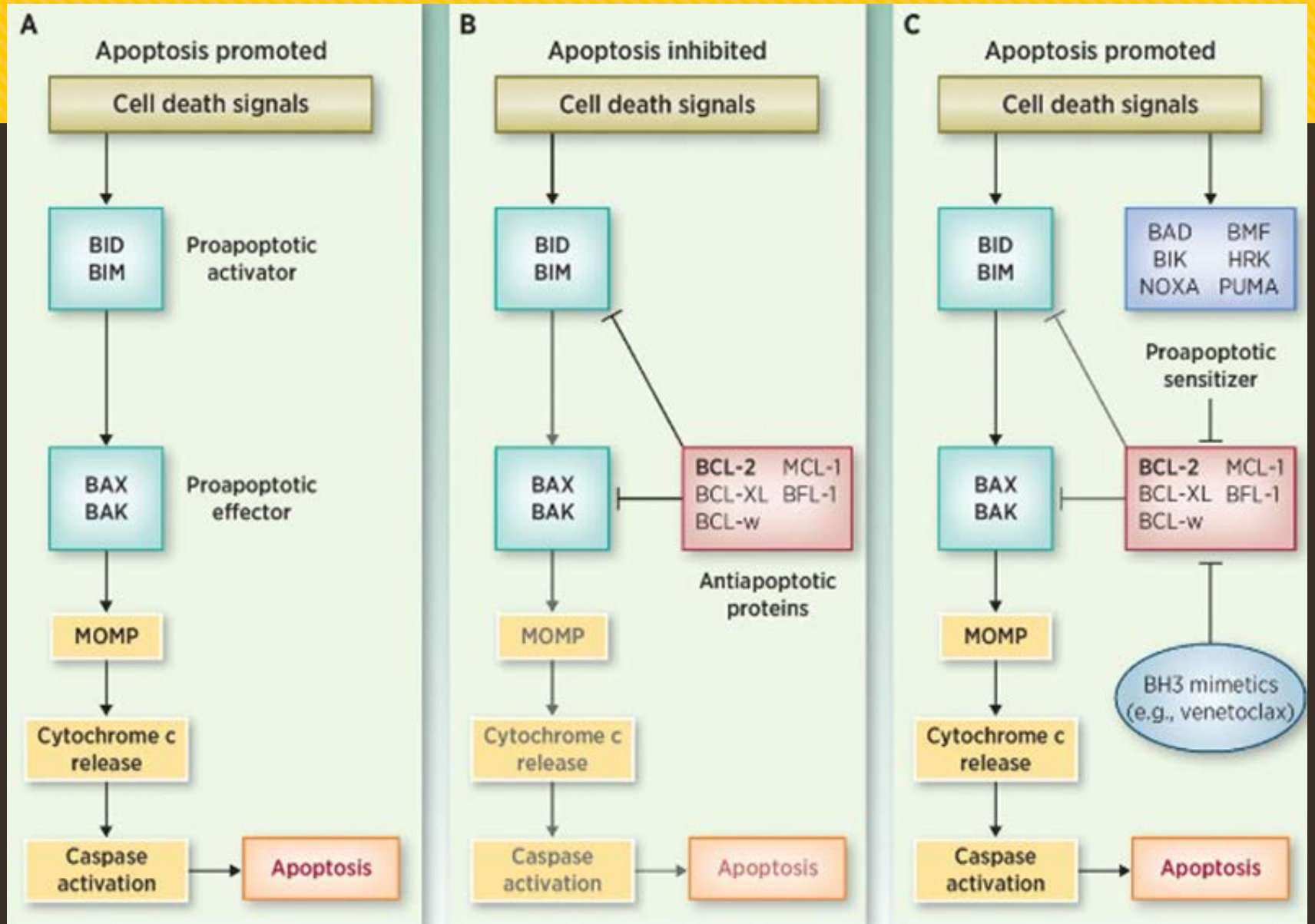
- May be used in CLL in combination with chemotherapy (FCR)

| Drug | Dose | Route | Schedule | Toxicities | Other |
|---|---|---|---|---|---|
| rituximab and hyaluronidase | 1600 mg/ 26800 units (fixed dose)<br><br>Volume: 13.4 mL | SubQ over 7 min | Day 1 of each cycle | ▪ Similar to IV rituximab<br>  ▪ local site reactions | ▪ Cycle 1 should be given as IV rituximab<br>▪ Observe patients for 15 min following SubQ injection |

# CLL

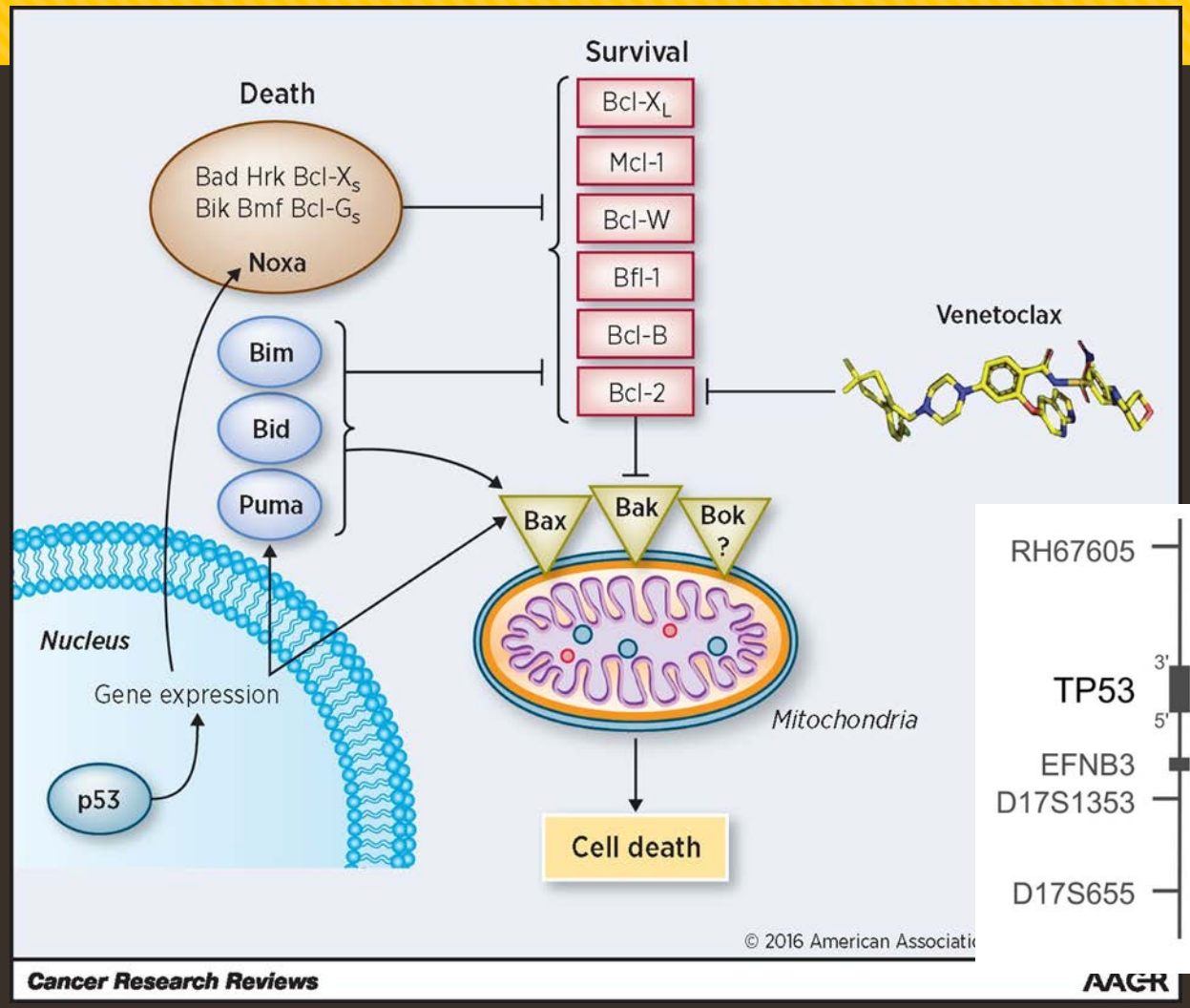
## Venetoclax

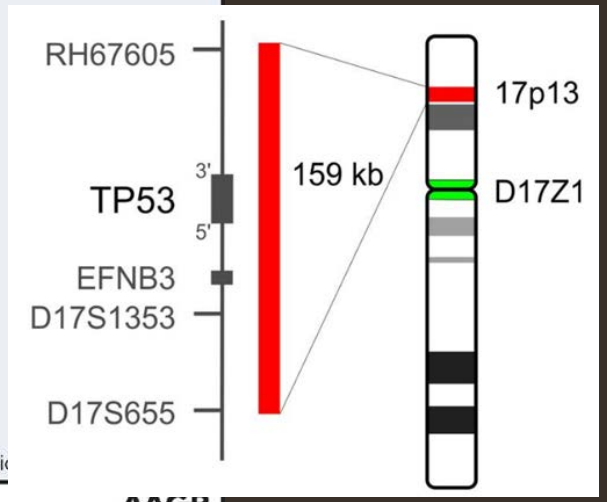
- Small molecule BCL2 inhibitor
  - Targets mitochondrial pathway of apoptosis
  - Inhibits antiapoptotic proteins



# CLL

## Venetoclax




- Identified as target for CLL with chromosomal abnormality (deletion of 17p)
  - Approved for use in refractory CLL with 17p deletion in 2016
  - Approval was based on non-randomized phase 2 data

# CLL

## Venetoclax

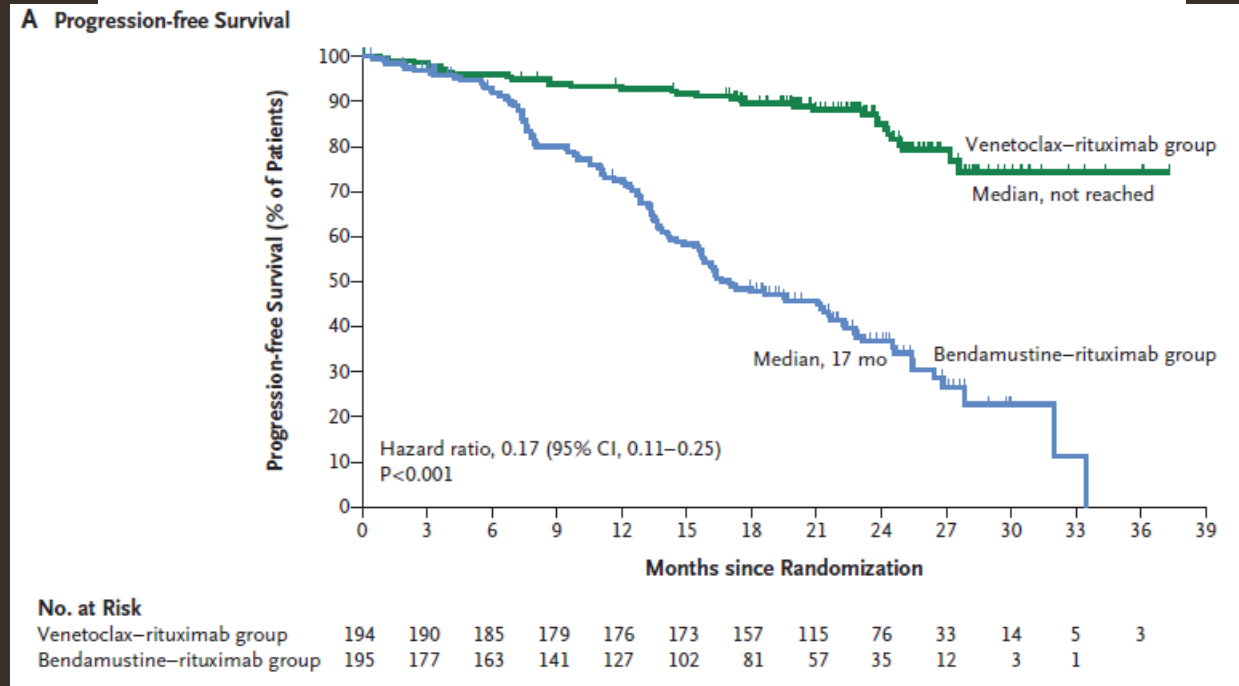
- Randomized, open-label, phase 3 trial
  - 389 participants with relapsed or refractory CLL
  - bendamustine/rituximab v. venetoclax/ rituximab
  - 2 yr rates of PFS were 84.9% for VR and 36.3% for BR
    - hazard ratio 0.17, 95% CI 0.11 to 0.25; P < 0.001

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

### Venetoclax–Rituximab in Relapsed or Refractory Chronic Lymphocytic Leukemia

J.F. Seymour, T.J. Kipps, B. Eichhorst, P. Hillmen, J. D'Rozario, S. Assouline, C. Owen, J. Gerecitano, T. Robak, J. De la Serna, U. Jaeger, G. Cartron, M. Montillo, R. Humerickhouse, E.A. Punnoose, Y. Li, M. Boyer, K. Humphrey, M. Mobasher, and A.P. Kater




Seymour JF, et al. N Engl J Med 2018;378:1107-20.

# CLL

## Venetoclax

- May be used in relapsed/refractory CLL

| Drug | Dose | Toxicities | Other |
|---|---|---|---|
| venetoclax | Start at 20 mg PO daily, titrate up to 400 mg PO daily | ▪ TLS<br>▪ Neutropenia<br>▪ Severe thrombocytopenia | ▪ Dose is escalated in weekly increments over 5 weeks to mitigate risk for TLS<br>▪ Premeds may include hydration and TLS prophylaxis depending on risk<br>▪ Potential for drug-drug interactions<br>• May require dose reduction |

# Investigational

## Venetoclax and HMAs in AML

- Venetoclax in combination with hypomethylating agents in AML
  - The anti-apoptotic protein BCL2 is over expressed in most AMLs
  - HMAs have established use in AML for patients not able to tolerate intensive chemotherapy
  - The combination has synergistic potential

# Investigational

## Venetoclax and HMAs in AML

- Data from a phase 1b trial was published in January 2018
  - 57 previously untreated patients were enrolled in a dose finding study

| | Group A (n=23) | Group B (n=22) | Group C (n=12) |
|---|---|---|---|
| Complete remission | 8 (35%) | 6 (27%) | 0 |
| CRi | 6 (26%) | 7 (32%) | 8 (67%) |
| Partial remission | 1 (4%) | 0 | 0 |
| MLFS* | 2 (9%) | 5 (23%) | 0 |
| Resistant disease | 3 (13%) | 2 (9%) | 3 (25%) |
| Non-evaluable† | 3 (13%) | 2 (9%) | 1 (8%) |
| Complete remission and CRi | 14 (61%) | 13 (59%) | 8 (67%) |
| Overall response‡ | 15 (65%) | 13 (59%) | 8 (67%) |
| Overall outcome§ | 17 (74%) | 18 (82%) | 8 (67%) |

Data are n (%). CRi=complete remission with incomplete marrow recovery. MLFS=morphologically leukaemia-free state. *Less than 5% blasts in an aspirate sample with marrow spicules and a count of 200 or more nucleated cells. †Includes five patients who discontinued before end of cycle 1 because of adverse events of infections; one patient was found to have CNS leukaemia on day 7. ‡Including complete remission, CRi, and partial remission. §Including overall response and MLFS.

**DiNardo CD, et al. Lancet Oncology 2018.** Published Online; January 12, 2018 http://dx.doi.org/10.1016/.

# Investigational

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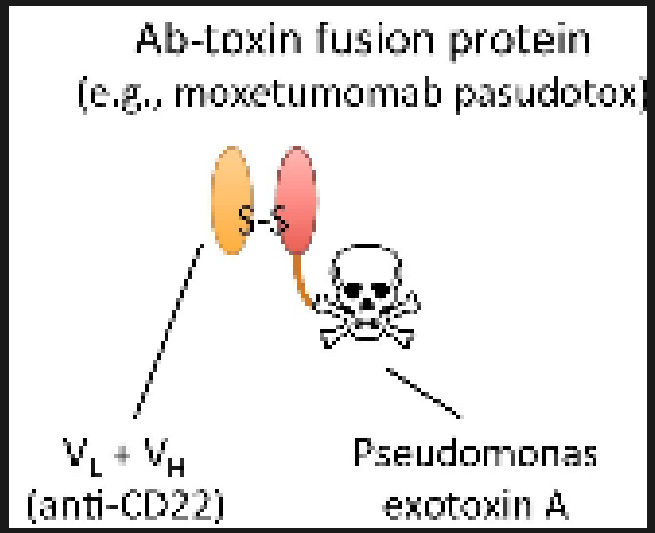
# Investigational

## New combinations not FDA approved

- Venetoclax in combination with hypomethylating agents in AML
  - Investigational
  - Phase III evaluation with comparator group is ongoing
- Other potential for venetoclax
  - Combination with FLT-3 inhibitors; liposomal daunorubicin and cytarabine
  - Younger patients

# New FDA Approval in Hairy Cell Leukemia

- Moxetumomab pasudotox
  - Antibody toxin fusion protein targeting CD22
- Approved for relapsed or refractory hairy cell leukemia
- Given as a 30 minute IV infusion on days 1, 3 and 5 of a 28 day cycle for up to 6 cycles
- Warnings:
  - Capillary leak syndrome
  - Hemolytic uremic syndrome
  - Renal toxicity

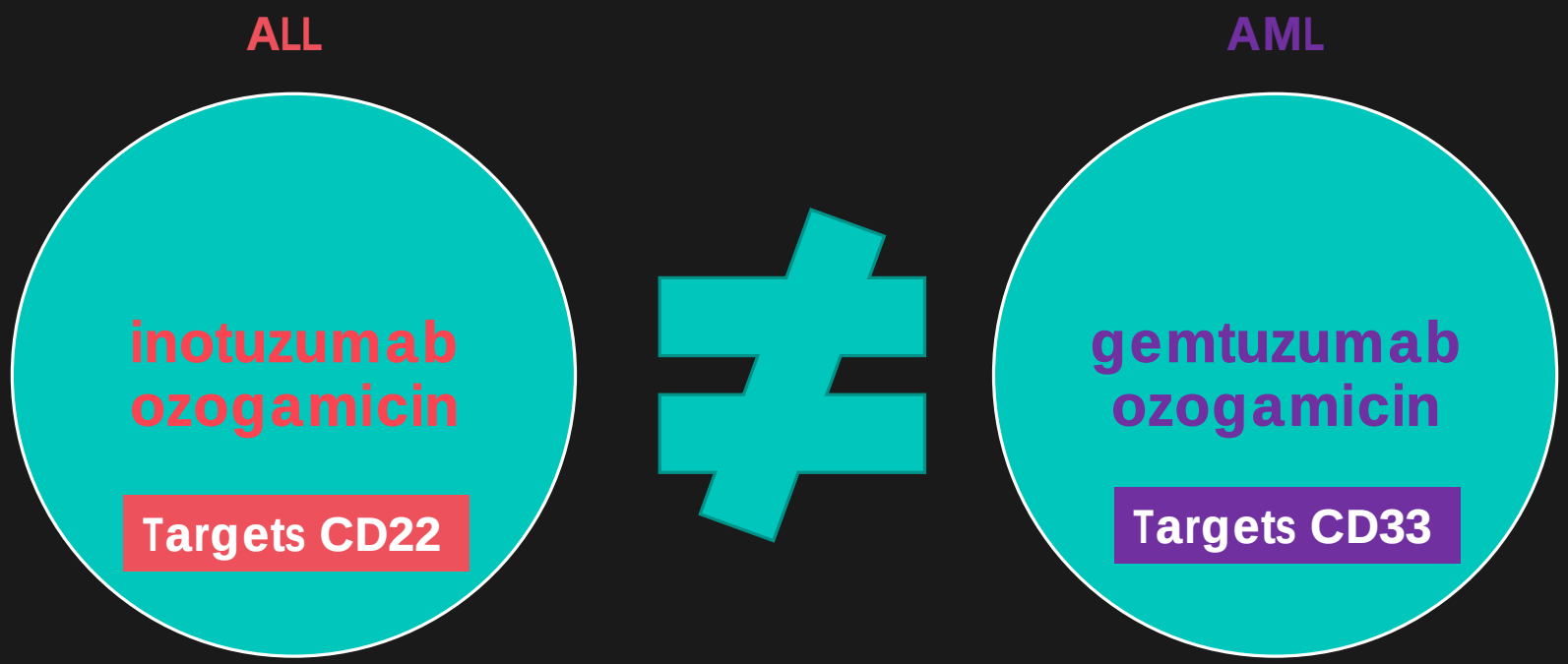


# Safety Point

**inotuzumab ozogamicin**

**gemtuzumab ozogamicin**

# Safety Point



# Safety Point

# Safety Point

# Safety Point

# Safety Point

**gemtuzumab ozogamacin**

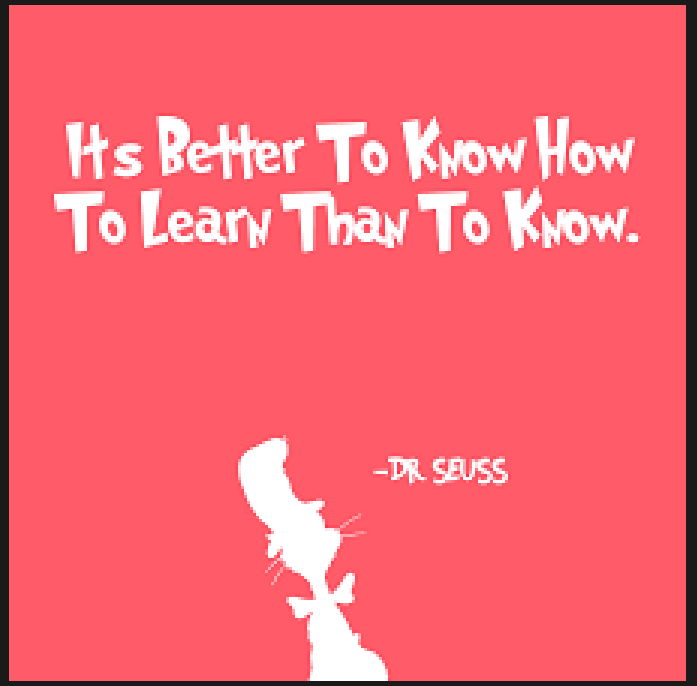

≠

**gemtuzumab**

# Drug Information and Approval Notifications

- New drugs:
  - Check the manufacturer's website for the most recent version of the prescribing information
- New literature:
  - Follow on social media professional organizations and journals
- Notifications for FDA approvals:
  - Sign up at FDA.gov to get updates on new oncology approvals

# Questions?



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