Welcome & Introductions

Dr. Steensma’s slides are available for download at www.LLS.org/programs.

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Division of Hematological Malignancies
Dana-Farber Cancer Institute
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Harvard Medical School
Boston, MA
What's on the Horizon for Acute Myeloid Leukemia?

Disclosure

David P. Steensma, MD, FACP, has affiliations with Acceleron, Amgen, Incyte, Onconova, and Takeda (Consultant).

Definitions and Risk Assessment
Definitions

- AML is a hematological cancer: “Clonal” proliferation of myeloid precursor cells (immature white blood cells), unable to mature

- These malignant “blasts” accumulate in marrow, blood, and sometimes other tissues – 20% blasts = acute leukemia

- These abnormal cells inhibit production of red blood cells, platelets, and mature neutrophils / granulocytes

AML – By The Numbers

- New US cases each year: ~21,000
- Deaths each year: ~11,000
- Median age: ~67 years
Prognostication in AML

- **Who** is the patient?
  - Age
  - Medical “co-morbidities” (i.e., other problems)

- Did it evolve out of **preceding marrow disease** (e.g. myelodysplastic syndromes (MDS))?  
  - Not always easy to tell...

- Is it a consequence of **therapy for another cancer**? (“therapy-related AML”)

- What are the **biological characteristics**?
  - Cytogenetic (chromosome) analysis
  - DNA mutational analysis: FLT3, NPM1, CEBPA, etc

Cytogenetics (chromosomes, karyotype)

**“Good-risk”**
- Translocation t(15;17) - Acute promyelocytic leukemia (APML, APL)
  - ~10%... A different disease
- t(8;21) and inv(16),
  - ~15%, “Core binding factor” alterations

**“Poor-Risk”**
- Chromosome 7 deletion
- Chromosome 5 deletion
- t(6;9)
- Complex (i.e., 3 or more abnormalities)
- Chromosome 11 translocations at 11q23
- Chromosome 17p abnormalities

**“Intermediate”**
- Normal
- One or two (non-bad) abnormalities
Prognostically important genes: 
**NPM1, FLT3 and CEBPA**

NPM1 mutation: Favorable  
FLT3 ITD: Unfavorable  
FLT3 TKD (D835/D836): Controversial  
CEBPA one mutation: Unfavorable  
CEBPA two mutations: Favorable

**Table 1.** European LeukemiaNet Standardized Reporting System for Correlation of Cytogenetic and Molecular Genetic Data in AML With Clinical Data

<table>
<thead>
<tr>
<th>Genetic Group</th>
<th>Subsets</th>
</tr>
</thead>
<tbody>
<tr>
<td>Favorable</td>
<td>t(9;21) or t(15;17) or inv(16)(p13.1q22); (t;16;16)(p13.1;q22); CBF B-AML1 (mutation or complex)</td>
</tr>
<tr>
<td>Intermediate I</td>
<td>Mutated NPM1 and FLT3-ITD (normal karyotype)</td>
</tr>
<tr>
<td>Intermediate II</td>
<td>Wildtype NPM1 and FLT3-ITD (normal karyotype)</td>
</tr>
<tr>
<td>Adverse</td>
<td>inv(3)(q21q26.2) or inv(3)(q21q26.2) or inv(16)(q22.1p13.1) or inv(16)(p13.1q22)</td>
</tr>
</tbody>
</table>

Abbreviations: AML, acute myeloid leukemia; ITD, internal tandem duplication. Complex karyotype is defined as those with chromosome abnormalities in the absence of one of the WHO designated recurring translocations or inversions: t(9;21), t(15;17), t(11;19), inv(16), t(6;9), or t(8;21).
Outcome by ELN Risk Group

AML Survival by Age
Why Do Older Patients with AML Have A Poorer Prognosis?

- Frailer and more commonly have other health problems
- Less favorable and more unfavorable cytogenetics
- Higher incidence of preceding MDS
- Elevated therapy-related morbidity and mortality
- Higher incidence of treatment-resistant disease
- Lower rates and duration of complete remission, and shorter median overall survival
- Less likely to be eligible for allogeneic hematopoietic cell transplantation

AML gets more complex!

*Recurring Mutations Found by Sequencing an Acute Myeloid Leukemia Genome*

*DNMT3A Mutations in Acute Myeloid Leukemia*

*EZH2 Mutations Are Related to Low Blast Percentage in Bone Marrow and -7/del(7q) in De Novo Acute Myeloid Leukemia*

*AXL mutations identify a high-risk subgroup of older patients with primary cytogenetically normal AML within the ELN Favorable genetic category*
>40 different recurrently mutated genes; 8 different major subtypes/pathways

200 Patients – 200 Different Diseases?

Each column represents a patient; each row is a gene; each colored box indicates mutation
Current Treatment

“3&7” or “7+3” – The Standard Remission Induction Approach for “Fit” Patients From 1973-2017

Standard Non-Protocol Therapy for Acute Myeloid Leukemia (AML)

Induction (Goal: achieve complete remission)

3&7 Regimen

**Cytarabine (Ara-C)** – 7 day continuous infusion

Day 1

**Daunorubicin** – daily IV x 3 days

(Some institutions use idarubicin instead of daunorubicin)

Approximately Day 14: Bone marrow aspirate/biopsy

Day 21

Recovery of healthy blood cells usually occurs after Day 21

**Conditions for hospital discharge:**
- Absolute neutrophil count >500/mm³ (>0.5 x 10⁹/L)
- Any infections are controlled (e.g., no fevers)
- Adequate oral intake

If persistent leukemia, retreatment may be necessary
If no persistent leukemia, wait for recovery of healthy cells

Remission rate: 40-75%, depending on age and disease biology
Mortality rate: 10-30%, depending on age and comorbid conditions
Complete remission rates with intensive treatment according to age and performance status

% achieving remission


Standard Post-Remission Intensive Therapy: 1993-?

Consolidation (Goal: lower risk of relapse)

High-dose or Mid-dose Cytarabine (Ara-C) -- "HIDAC" or "MIDAC" (May be repeated up to 4 times, with 2-6 week break between cycles after count recovery)

AM

PM

6 Cytarabine (Ara-C) doses total, each dose is a ~3 hour infusion
1 dose in AM and 1 dose in PM on days 1, 3, and 5 (no chemotherapy on day 2 or day 4)

Most patients can be safely discharged from the hospital on day 6, but about one-half will need to be readmitted later, usually due to fevers during the period of low white counts (neutropenia)

*First Consolidation treatment ("cycle") usually begins 2-6 weeks after recovery of blood counts following Induction chemotherapy
* For some patients, the first Consolidation cycle may be "2 & 5", a shortened version of 3 & 7, instead of HIDAC or MIDAC
* Some patients will go to stem cell transplant during or instead of Consolidation (depends on patient age, risk of relapse, and donor availability)

Created August 2010 by David Steensma MD for the Dana-Farber Cancer Institute Leukemia Group
Emerging Approaches

2017 Acute Myeloid Leukemia
FDA Approvals

• 4/28/17: Midostaurin (Rydapt™; Novartis)
  – For adult patients with newly diagnosed AML who have a FLT3 mutation
  – Companion diagnostic: Invivoscribe LeukoStrat CDx FLT3 mutation assay

• 8/1/17: Enasidenib (Idhifa™; Agios)
  – For adult patients with relapsed/refractory AML who have an IDH2 mutation
  – Companion diagnostic: Abbott m2000 RealTime IDH2 mutation assay

• 8/3/17: CPX351 / fixed ratio daunorubicin-cytarabine (Vyxeos™; Jazz)
  – For adults with either of two types of AML: newly diagnosed therapy-related AML (t-AML) or AML with myelodysplasia-related changes (AML-MRC)

• 9/1/17: Gemtuzumab ozogamicin (Mylotarg™; Pfizer)
  – For adults with newly diagnosed AML whose tumors express the CD33 antigen (CD33+), and for treatment of patients 2 years or older with relapsed/refractory CD33+ AML
CPX-351 Structure

CPX-351 Uses a Nano-Scale Delivery Complex

- 100 nm bilamellar liposomes
- 5:1 molar ratio of cytarabine to daunorubicin
- 1 unit = 1.0 mg cytarabine plus 0.44 mg daunorubicin

Source: Celator

CPX-351 Trial Design

Randomized Phase 3 Trial of CPX-351 vs “7+3” in Older Patients with sAML

Primary Endpoint: Overall Survival

Eligibility
- Previously untreated sAML or tAML
- Ages 60–75
- Able to tolerate intensive therapy
- PS 0–2

CPX-351
100 units/m² IV days 1, 3, 5
Up to 2 inductions and 2 consolidations

7+3
Daunorubicin 60 mg/m²
Cytarabine 100 mg/m²

Source: Celator
CPX-351 Trial Result

Activating FLT3 Mutations in AML

ITD: 25-30%
High relapse, poor prognosis

TKD: 5-10%

A Phase III Randomized Double-blinded Study Of Chemotherapy +/- Midostaurin (PKC412) In Newly Diagnosed Adults aged 18-60 with FLT3 Mutated Acute Myeloid Leukemia (AML)


Participants: ALLIANCE/CALGB, AMLSG, CETLAM, ECOG, EORTC, GIMEMA, NCIC, OSHO, PETHEMA, SAL, SWOG

CTEP sponsored, Novartis provided drug and sponsored outside North America, and Alliance (formerly CALGB) chaired study, collected data and performed analysis

RATIFY Trial Schema

Stratify FLT3 ITD or TKD

FLT3 WILD TYPE not eligible for enrollment

Stratification: TKD; ITD with allelic ratio <0.7 ‘vs’ ≥0.7
Overall Survival (Primary Endpoint)
23% reduced risk of death in the Midostaurin arm

- Median OS: Mido 74.7 (31.7-NE); PBO 25.6 (18.6-42.9) months
  NE: not estimable
  * controlled for FLT3 subtype (TKD, ITD-Low, ITD-High)

Phase 2 Study of Quizartinib in AML:
Response Rate

- Median duration of response: 12.1 weeks FLT3-ITD(+) & 7.0 weeks FLT3-ITD(-)
- 75% of FLT3-ITD(+) and 48% of FLT3-ITD(-) patients refractory to their last prior therapy, achieved at least a PR to quizartinib

Levis et al
ASH 2015
Clinical Response to Gilteritinib Treatment by FLT3 Mutation or TKI Status

<table>
<thead>
<tr>
<th>Clinical Response</th>
<th>≥80 mg Gilteritinib</th>
<th>TKI Status</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mutation Type</td>
<td>TKI Status</td>
</tr>
<tr>
<td></td>
<td>FLT3-ITD only</td>
<td>N=142</td>
</tr>
<tr>
<td></td>
<td>FLT3-D835 only</td>
<td>N=11</td>
</tr>
<tr>
<td></td>
<td>ITD and D835</td>
<td>N=9</td>
</tr>
<tr>
<td></td>
<td>Prior TKI</td>
<td>N=40</td>
</tr>
<tr>
<td></td>
<td>TKI Naïve</td>
<td>N=127</td>
</tr>
<tr>
<td>CR</td>
<td>N=142</td>
<td></td>
</tr>
<tr>
<td>CRp</td>
<td>N=11</td>
<td></td>
</tr>
<tr>
<td>CRi</td>
<td>N=9</td>
<td></td>
</tr>
<tr>
<td>PR</td>
<td>N=40</td>
<td></td>
</tr>
<tr>
<td>CRc (CR+CRp+CRi)</td>
<td>N=127</td>
<td></td>
</tr>
<tr>
<td>ORR (CRc+PR)</td>
<td>N=127</td>
<td></td>
</tr>
</tbody>
</table>

Data presented as n (%).

CR, complete remission; CRc, composite complete remission; CRi, complete remission with incomplete hematologic recovery; CRp, complete remission with incomplete platelet recovery; ORR, overall response rate; PR, partial response.

IDH1/2-mutant AML

- Mardis et al, NEJM 2009: First description of IDH1 mutations in ~8% of patients with AML, associated with normal cytogenetic status (cn-AML).
- Subsequent studies found a larger subset, ~15%, of patients with mutations in the IDH2 gene.
- IDH proteins, essential to the Krebs Cycle, catalyze decarboxylation of isocitrate to α-ketoglutarate (α-KG) in cytoplasm (IDH1) and mitochondria (IDH2).
- Mutant IDH enzymes catalyze an NADPH-dependent reduction of α-KG to 2-hydroxyglutarate (2-HG).
- This leads to accumulation of 2-HG onco-metabolite in IDH-mutant tumors.

- Ward et al. Cancer Cell, 2010
Response to Enasidenib (AG-221) in AML/MDS

<table>
<thead>
<tr>
<th>Overall Response (CR, CRp, CRi, mCR, PR)</th>
<th>RR-AML (n = 159)</th>
<th>Untreated AML (n = 24)</th>
<th>MDS (n = 14)</th>
<th>All (N = 209)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overall Response</td>
<td>59 (37%) [95% CI: 30%, 45%]</td>
<td>10 (42%) [22%, 62%]</td>
<td>7 (50%) [23%, 77%]</td>
<td>79 (38%) [31%, 45%]</td>
</tr>
<tr>
<td>CR</td>
<td>29 (18%) [95% CI: 13%, 25%]</td>
<td>4 (17%) [5%, 37%]</td>
<td>3 (21%) [5%, 51%]</td>
<td>37 (18%) [13%, 24%]</td>
</tr>
<tr>
<td>CRp</td>
<td>1 (1%)</td>
<td>1 (4%)</td>
<td>1 (7%)</td>
<td>3 (1%)</td>
</tr>
<tr>
<td>CRi</td>
<td>3 (2%)</td>
<td>0</td>
<td>0</td>
<td>3 (1%)</td>
</tr>
<tr>
<td>mCR</td>
<td>9 (6%)</td>
<td>1 (4%)</td>
<td>3 (21%)</td>
<td>14 (7%)</td>
</tr>
<tr>
<td>PR</td>
<td>17 (11%)</td>
<td>4 (17%)</td>
<td>0</td>
<td>22 (11%)</td>
</tr>
<tr>
<td>SD</td>
<td>72 (45%)</td>
<td>9 (38%)</td>
<td>6 (43%)</td>
<td>96 (48%)</td>
</tr>
<tr>
<td>PD</td>
<td>10 (6%)</td>
<td>1 (4%)</td>
<td>0</td>
<td>11 (5%)</td>
</tr>
<tr>
<td>Not evaluable</td>
<td>18 (11%)</td>
<td>4 (17%)</td>
<td>1 (7%)</td>
<td>23 (11%)</td>
</tr>
</tbody>
</table>

* Overall response by IDH mutation type: R140Q 36% / R172K 42%

CR, complete response; CRp, CR with incomplete platelet recovery; CRi, CR with incomplete hematologic recovery; mCR, marrow CR; PR, partial response; SD, stable disease; PD, progressive disease

Clinical activity of AG-120 in R/R AML

<table>
<thead>
<tr>
<th>Patient achieved CR by end of Cycle 1</th>
<th>Dose escalation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Screening 44% blasts</td>
<td>R/R AML n=63</td>
</tr>
<tr>
<td>Cycle 1 Day 15 3% blasts</td>
<td>Overall N=78</td>
</tr>
<tr>
<td>Patient achieved CR by end of Cycle 1</td>
<td>CR, n (%)</td>
</tr>
<tr>
<td></td>
<td>10 (16)</td>
</tr>
<tr>
<td></td>
<td>CRi/CRp, n (%)</td>
</tr>
<tr>
<td></td>
<td>8 (13)</td>
</tr>
<tr>
<td></td>
<td>PR, n (%)</td>
</tr>
<tr>
<td></td>
<td>1 (2)</td>
</tr>
<tr>
<td></td>
<td>mCR/MLFS, n (%)</td>
</tr>
<tr>
<td></td>
<td>2 (3)</td>
</tr>
<tr>
<td></td>
<td>SD, n (%)</td>
</tr>
<tr>
<td></td>
<td>27 (43)</td>
</tr>
<tr>
<td></td>
<td>PD, n (%)</td>
</tr>
<tr>
<td></td>
<td>8 (13)</td>
</tr>
<tr>
<td></td>
<td>NE, n (%)</td>
</tr>
<tr>
<td></td>
<td>7 (11)</td>
</tr>
<tr>
<td></td>
<td>ORR, n (%) [95% CI]</td>
</tr>
<tr>
<td></td>
<td>21 (33) [22, 46]</td>
</tr>
</tbody>
</table>

Data cut off date 1 August 2016

CR = complete response; CRi = CR with incomplete neutrophil recovery; CRp = CR with incomplete platelet recovery; PR = partial response; mCR/MLFS (marrow CR/morphologic leukemia-free state) = <5% blasts in bone marrow; no hematologic recovery; SD = stable disease; NE = not evaluable; ORR = overall response rate (CR + CRi + CRp + PR + mCR/MLFS)
DNA Hypomethylating Therapies

- Less intensive treatment, increasingly used for frailer or older patients
- Typically administered in outpatient clinic
- Can lead to therapeutic responses, including transfusion independence, decrease in leukemic burden, and less commonly, remissions
- However, responses often transient, with leukemic progression and brief post-HMA survival
- **Decitabine** and **azacitidine** FDA approved for MDS (and AML with 20-30% blasts)
- Development: guadecitabine (SGI-110), CC-486, ASTX727


Hypomethylating Therapy among Older Adults

CR rate: 18 % (10/55)

Fenaux P, et al. JCO 2010


TP53 and Decitabine in Acute Myeloid Leukemia and Myelodysplastic Syndromes

Welch et al. NEJM 2016
Venetoclax was shown to synergize with HMA in preclinical models, suggesting that combination with HMA may be a promising approach in AML

- FDA approved for CLL
- Reported: phase 1b, open-label, nonrandomized, dose-escalation trial of venetoclax in combination with DEC or AZA in older (≥65 years), treatment-naive AML patients (NCT02203773)
- Ongoing: randomized trials in up-front and relapsed/refractory AML

### Venetoclax + HMA Efficacy in AML

<table>
<thead>
<tr>
<th>Overall Response, n (%)</th>
<th>Arm A (VEN + DEC)</th>
<th>Arm B (VEN + AZA)</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>VEN 400 mg (n=6)</td>
<td>VEN 800 mg (n=12)</td>
<td></td>
</tr>
<tr>
<td>CR+Cri</td>
<td>3 (50)</td>
<td>9 (75)</td>
<td></td>
</tr>
<tr>
<td>ORR (CR+Cri+PR)</td>
<td>3 (50)</td>
<td>10 (83)</td>
<td></td>
</tr>
<tr>
<td>CR+Cri+PR+MLFS</td>
<td>3 (50)</td>
<td>11 (92)</td>
<td></td>
</tr>
</tbody>
</table>

- 27/45 (60%) patients achieved CR/Cri, 1/45 (2%) had partial remission (PR), and 4/45 (9%) patients achieved morphologic leukemia free state (MLFS) when treated at all dose levels
- 23/34 (68%) patients achieved CR/Cri when treated at 400 and 800mg dose levels
- Median time to CR/Cri was 1 month (range, 0.8–3.8)
- The median duration of response was 8.4 months (95% CI = 6.8—not reached)
- 7/45 (16%) patients experienced morphologic relapse after achieving a CR or CRI
- Median time on study was 3.2 months (range, 0.2–14.6)


Venetoclax: An Oral Selective BCL-2 Inhibitor

- Venetoclax was shown to synergize with HMA in preclinical models, suggesting that combination with HMA may be a promising approach in AML
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Dinardo C, ASH 2015, Abstract 327
Pollyea D, ASCO 2016, Abstract 7009.
Vadastuximab Talirine (SGN-CD33A; 33A)
Proposed Mechanism of Action in Combination with HMA

Anti-CD33 antibody, engineered cysteines to enable uniform site-specific conjugation
Cleavable dipeptide linker, highly stable in circulation
Pyrolobenzodiazepine (PBD) dimer, binds DNA with high intrinsic affinity

But... 7/2017, development on hold due to excess deaths in randomized study; FDA and sponsor evaluating

Vadastuximab Best Clinical Response: Efficacy Evaluable Patients

<table>
<thead>
<tr>
<th>Efficacy Evaluable</th>
<th>All N=49</th>
<th>Secondary AML N=22</th>
<th>FLT3/ITD+ N=5</th>
<th>Age ≥75 years N=26</th>
</tr>
</thead>
<tbody>
<tr>
<td>Remission Rate (CR + CRi)</td>
<td>73%</td>
<td>77%</td>
<td>100%</td>
<td>65%</td>
</tr>
<tr>
<td>CR</td>
<td>47%</td>
<td>50%</td>
<td>80%</td>
<td>38%</td>
</tr>
<tr>
<td>CRi (p)a</td>
<td>20%</td>
<td>18%</td>
<td>20%</td>
<td>19%</td>
</tr>
<tr>
<td>CRi (n)b</td>
<td>6%</td>
<td>9%</td>
<td>0</td>
<td>8%</td>
</tr>
<tr>
<td>mLFSc</td>
<td>2%</td>
<td>5%</td>
<td>0</td>
<td>4%</td>
</tr>
<tr>
<td>ORR (CR + CRi + mLFS)</td>
<td>76%</td>
<td>82%</td>
<td>100%</td>
<td>69%</td>
</tr>
</tbody>
</table>

a CRi (p)= CR with ANC ≥1000/uL, incomplete platelet recovery
b CRi (n)= CR with platelets ≥100,000/uL, incomplete neutrophil recovery
c mLFS = morphologic leukemia-free state
d Defined as therapy-related AML, AML evolved from prior MDS, or de novo AML with MDS-related cytogenetics
Chimeric Antigen Receptor T (CAR-T) Cells: An Advance for ALL; Will They Be Useful Someday for AML?


Immune Checkpoint Inhibitors: A Breakthrough In Solid Tumors, What About AML?

Image Source: NCI
What’s Next For AML?
“Personalized” Medicine

Personalized medicine approach to treating AML

1. Personalized medicine approach to treating AML
   - In collaboration with the Knight Cancer Institute, Oregon Health & Science University and other researchers, personalized medicine is being developed to target AML.

2. Drug and biological companies will work with the collaboration to identify specific targets.
   - Tailored inhibitors will be developed for each target.

3. Humanized mice with transplanted human AML cells will be used for testing.
   - These mice can model the human disease and help in the development of personalized treatments.

4. Blood and bone marrow samples will inform personalized treatment.
   - Further research will be conducted to refine the treatments.

Gojo I, Karp J. *Clin Can Res* Dec 2014 43
Transplant ≈ “Rebooting the System”

What Needs To Be Done Before Transplant?
Donor Identification

Likelihood of Finding a Matching Adult Donor

- African American or Black: 76%
- Hispanic or Latino: 83%
- Asian or Pacific Islander: 84%
- Americans Indian and Alaska Native: 90%
- Caucasian: 97%

SOURCE: National Marrow Donor Program / Be The Match 2013 fiscal year reports.
Typical Pre-Transplant Testing

- Electrocardiogram (ECG)
- Echocardiogram
- Pulmonary function tests (PFTs, spirometry)
- Dental exam and cleaning
- Updated marrow biopsy
- Blood tests: tissue type, antibodies, blood type, infection markers, organ function

Allogeneic Hematopoietic Stem Cell Transplant (Allo-HSCT)

- Stem cells removed from donor
- Patient receives treatment to destroy blood-forming cells or prevent rejection of donated cells
- Patient receives stem cells

Source: NCI
Transplant Trends

- **Older fit patients** are increasingly considered eligible (up to ~75 years)
- Increasing use of half-matched “haplo” transplant (usually parent/child)
  - Randomize transplant of haplo vs cord blood
- Increasing elective use of **bone marrow rather than blood stem cells** as donor source
  - Requires an operation, but may reduce graft-versus-host (GVH)
- Increasing use of post-transplant **preventive or pre-emptive therapy**
  - e.g., sorafenib after FLT3 AML allo-SCT
- Monitoring and treatment of graft-versus-host and infection is improving

Conclusions

- FDA approval of 4 new AML drugs in 2017!
  - Hopefully the beginning of a wave
- Biological understanding of AML is improving
- More than 300 AML clinical trials ongoing, testing >40 different novel compounds
  - Clinical trials are the only way to move the field forward
- Transplant is more broadly applied, outcomes improving
Thank you!

DFCI Adult Leukemia Clinical Program
Richard Stone MD
Daniel Deangelio MD PhD
Martha Wadleigh MD
Gregory (Goyo) Abel MD MPH
Jacqueline S Garcia MD
Eric S Winer MD
Marlise Luskin MD
Ilene Galinsky RN NP
Mary Gerard PA-C
Adriana Penicaul PA-C
Katherine Edmonds RN NP

Research Collaborators
Benjamin Ebert MD PhD
R. Coleman Lindsley MD PhD
Andrew Lane MD PhD
David Frank MD PhD
Tony Letai MD PhD

As of February 2017, Dana-Farber currently has
533 therapeutic clinical trials
in progress to treat cancers like:
Metastatic breast cancer
Lung cancer
Multiple myeloma
Leukemia

2,156 adults participated in a clinical trial at Dana-Farber in 2016

What’s on the Horizon for Acute Myeloid Leukemia?

Q&A Session

Ask a question by phone:
• Press star (*) then the number 1 on your keypad.

Ask a question by web:
• Click “Ask a question”
• Type your question
• Click “Submit”

Due to time constraints, we can only take one question per person. Once you’ve asked your question, the operator will transfer you back into the audience line.
The Leukemia & Lymphoma Society Offers:

- **Information Resource Center:** Information Specialists, who are master’s level oncology professionals, are available to help cancer survivors navigate the best route from diagnosis through treatment, clinical trials and survivorship.
  - EMAIL: infocenter@LLS.org
  - TOLL-FREE PHONE: 1-800-955-4572

- **Free Education Booklets:**
  - [www.LLS.org/booklets](http://www.LLS.org/booklets)

- **Free Telephone/Web Programs:**
  - [www.LLS.org/programs](http://www.LLS.org/programs)

- **Live, weekly Online Chats:**
  - [www.LLS.org/chat](http://www.LLS.org/chat)

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The Leukemia & Lymphoma Society Offers:

- **Support Resources:** LLS Community, discussion boards, blogs, support groups, financial assistance and more: [www.LLS.org/support](http://www.LLS.org/support)
  - *NEW LLS Podcast, The Bloodline with LLS!* Listen in as experts and patients guide listeners in understanding diagnosis, treatment, and resources available to blood cancer patients. [www.thebloodline.org](http://www.thebloodline.org)

- **Education Video:** Free education videos about survivorship, treatment, disease updates and other topics: [www.LLS.org/educationvideos](http://www.LLS.org/educationvideos)

- **Patti Robinson Kaufmann First Connection Program:** Peer-to-peer program that matches newly diagnosed patients and their families: [www.LLS.org/firstconnection](http://www.LLS.org/firstconnection)

- **Free Nutrition Consults:** Telephone and email consultations with a Registered Dietitian: [www.LLS.org/nutrition](http://www.LLS.org/nutrition)

- **What to ask:** Questions to ask your treatment team: [www.LLS.org/whattoask](http://www.LLS.org/whattoask)
We have one goal: A world without blood cancers