# Clinical Trials in Multiple Myeloma

## *IMF Living Well Teleconference*

## *November 1, 2018*

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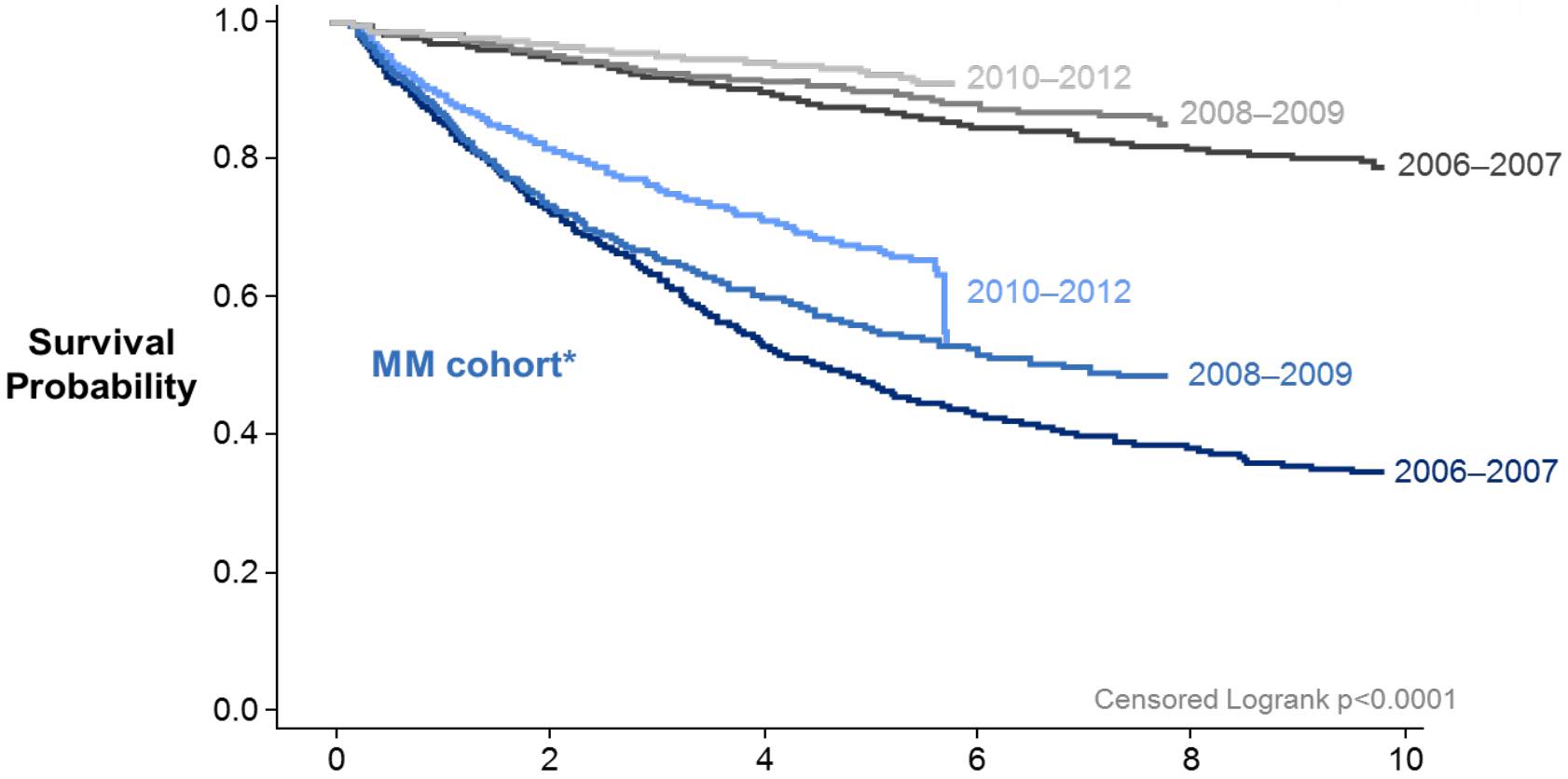
Chief Medical Officer, International Myeloma Foundation
Professor, Translational Genomics Research Institute (TGen)
City of Hope Cancer Center

# Improving Survival in MM




*Year ranges represent the year of diagnosis.

Note: By linking to the SSA Master Death File, survival was measured as time from diagnosis date to the date of death obtained from the SSA, time from diagnosis date to the date of inpatient death, or time from diagnosis date to September 30, 2015; Survival estimates were presented for multiple myeloma patients diagnosed and treated during 2006-2012 (n=9,521).

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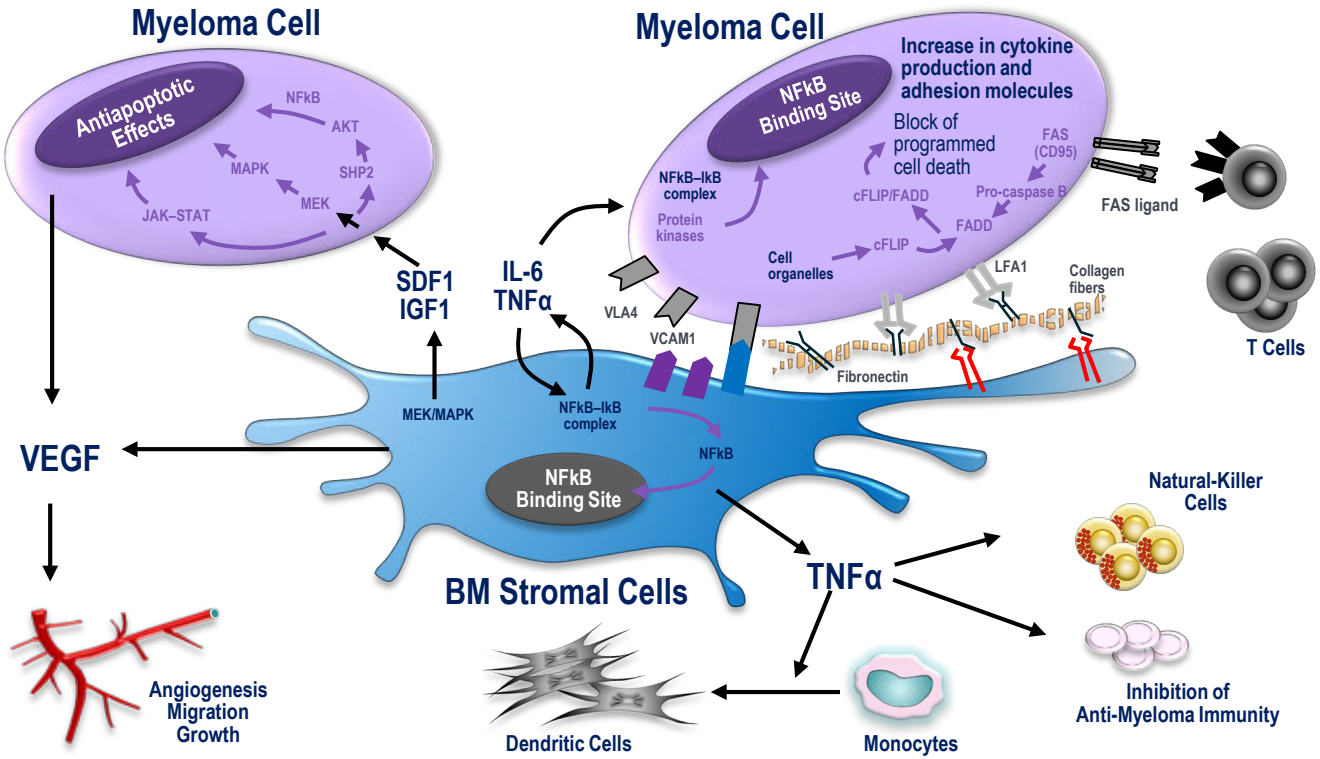
# Impact of Clinical Trials in Myeloma: Dramatic Improvements in Survival in <10 Years

- Survival rates have nearly doubled; further improvements expected in near future
- Ten new drugs approved since 2003
  - IMiDs: Thalomid, Revlimid, Pomalyst
  - Proteasome inhibitors: Velcade, Pomalyst, Ninlaro
  - Histone deacetylase inhibitor: Farydak
  - Monoclonal antibodies: Darzalex, Empliciti
  - Chemotherapy: Doxil
- Several of these have multiple indications
- Many new drugs being studied in clinical trials
- Understanding of the biology of myeloma improving, with the eventual goal of personalized medicine

IMiD, immunomodulatory drug

# The Myeloma Microenvironment Is Key to Disease Pathophysiology

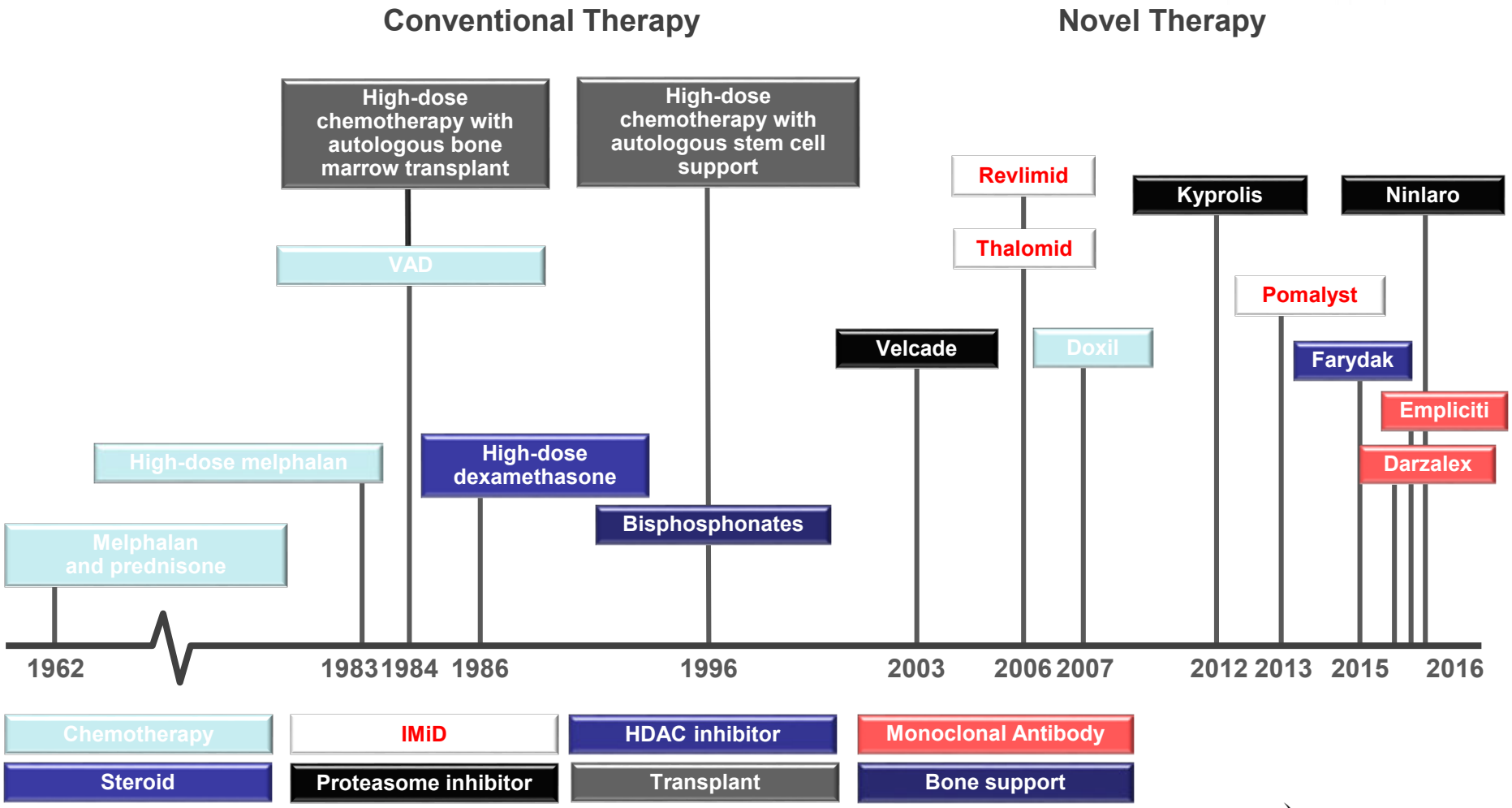



Bruno B et al. *Lancet Oncol*. 2004;5:430-442.

# Objectives

- Review the immense progress made in myeloma due to clinical trials
- Outline the basics of clinical trials and their phases
- Discuss the benefits and challenges of clinical trials
- Delineate key ongoing trials in multiple myeloma
- Preview novel molecules and approaches soon to accessible in clinical trials in myeloma

# Evolution of Multiple Myeloma Treatment




VAD, vincristine, doxorubicin, dexamethasone; IMiD, immunomodulatory drug; HDAC, histone deacetylase.

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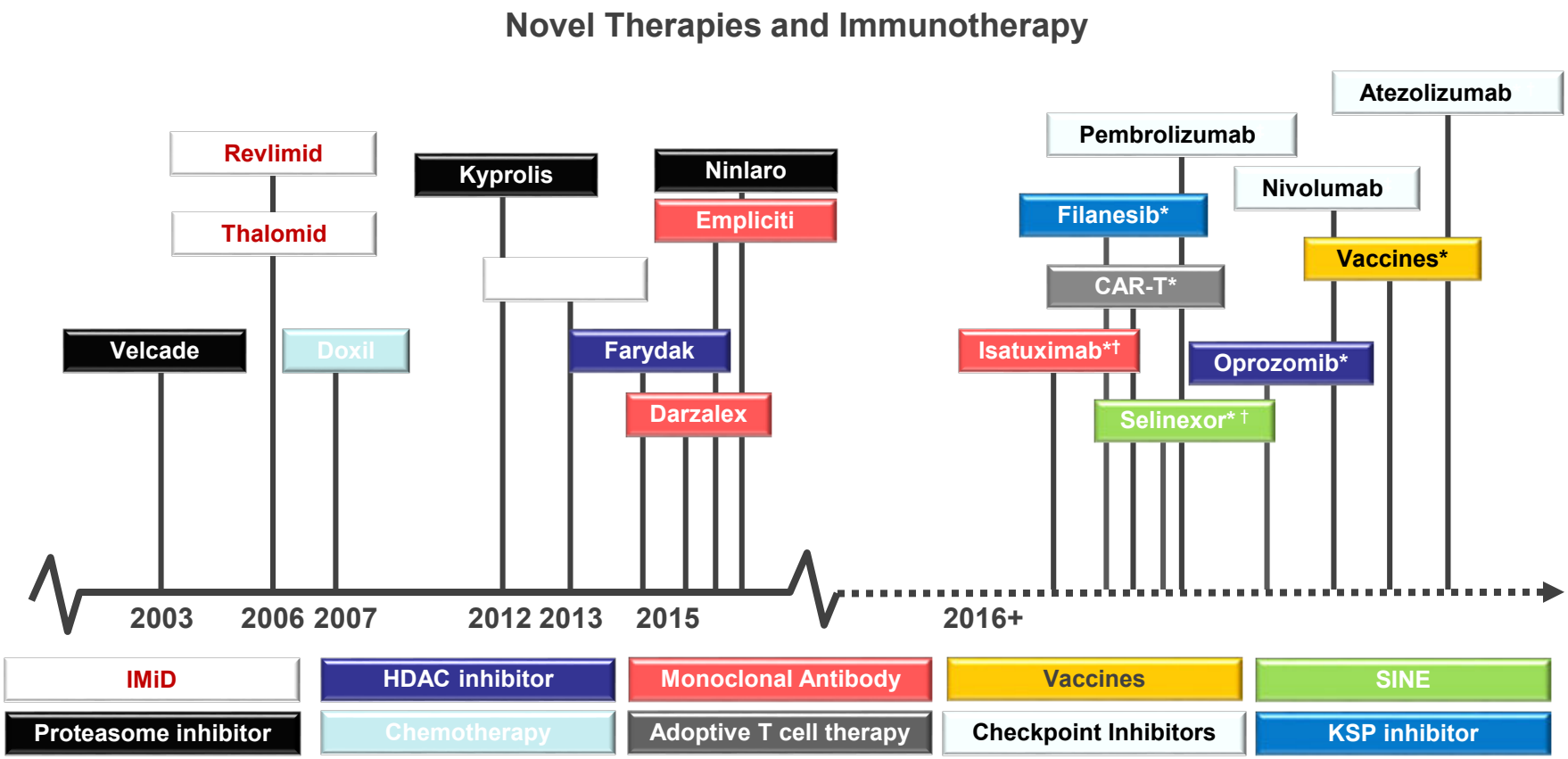
# Continuing Evolution of Multiple Myeloma Treatment: New Classes and Targets




PLD, peglylated liposomal doxorubicin; IMiD, immunomodulatory drug; HDAC, histone deacetylase; KSP, kinesin spindle protein, SINE, selective inhibitor of nuclear export
*Not yet FDA-approved; only available in clinical trials
†Treatments studied in MMRC trials
‡FDA-approved for a non-MM indication

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# Current Important Research Questions

- How can treatments be matched to patients' subtypes/genomics (personalized medicine)?
- What are the best drugs and combinations of drugs for multiple myeloma at all stages of disease?
- What new molecules could be effective in treating myeloma?

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# Misconceptions About Cancer Clinical Trials

| Misconceptions | Facts |
| --- | --- |
| I may get a sugar pill (placebo) instead of real therapy. | No placebos are given alone — every patient receives treatment. |
| I'll be treated like a guinea pig. | Most patients receive care that exceeds expectations. |
| Clinical studies are for people with no other options. | Many involve an adjustment to a standard of care that may improve outcome or quality of life |

***The more people who participate, helps to speed drug development.***

Available at http://health.clevelandclinic.org/2014/04/10-biggest-cancer-clinical-trial-myths-busted/.

# Overview of New Drug Development

**STEP 1**

Identify a target for therapy in the laboratory

**STEP 2**

Confirm the anticancer activity in laboratory and animal studies

**STEP 3**

Clinical trials (human studies) to determine safety, dosing and effectiveness

*The whole process costs millions of dollars and years of effort!*

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# Clinical Trials

Remember some of the important principles of clinical trials:

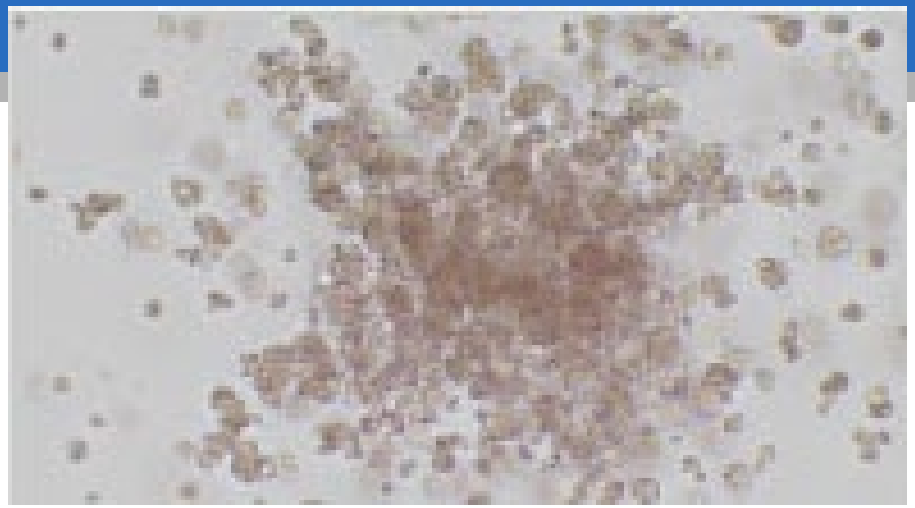
- The drive of research has brought us to where we are
- No one is expected to be a “guinea pig” with no potential benefit to them
- Research is under very tight supervision and standards
- Open, clear communication between the physician and the patient is fundamental

# Clinical Trials – Why Me??

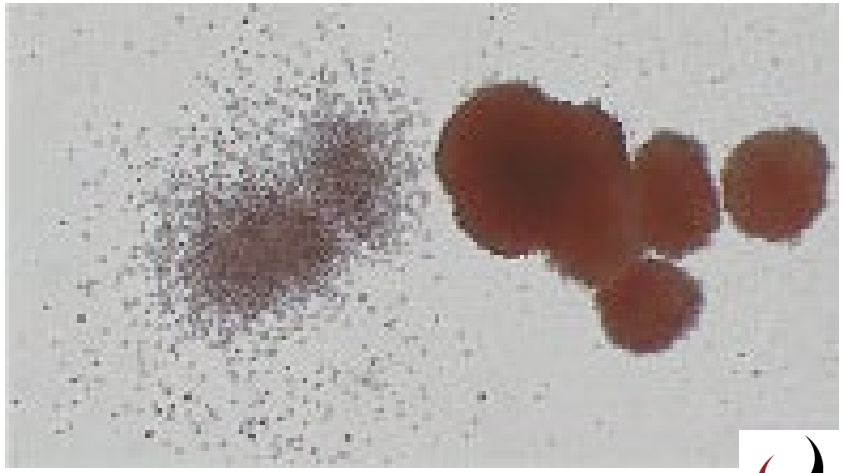
- Every patient is unique and must be viewed that way
- Benefits of trials are numerous and include:
  - Early access to “new” therapy
  - Delay use of standard therapy
  - Contribution to myeloma world – present and future
  - Financial access to certain agents
- Must be balanced with potential risks
  - “toxicity” of side effects
  - Possibility of lack of efficacy

# Even Before Phase I

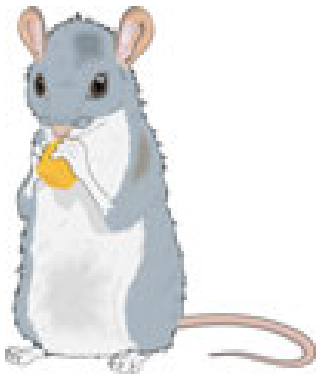
- Most agents are tested in lab models
  - Various “myeloma cell lines” = in vitro
- Next step is animal model
  - We are more like mice than you think!!
- Earliest study in phase I is called “First in Human”
  - Often uses extremely low dose of drug to ensure safety



# *In Vitro* Activity




# Murine Activity



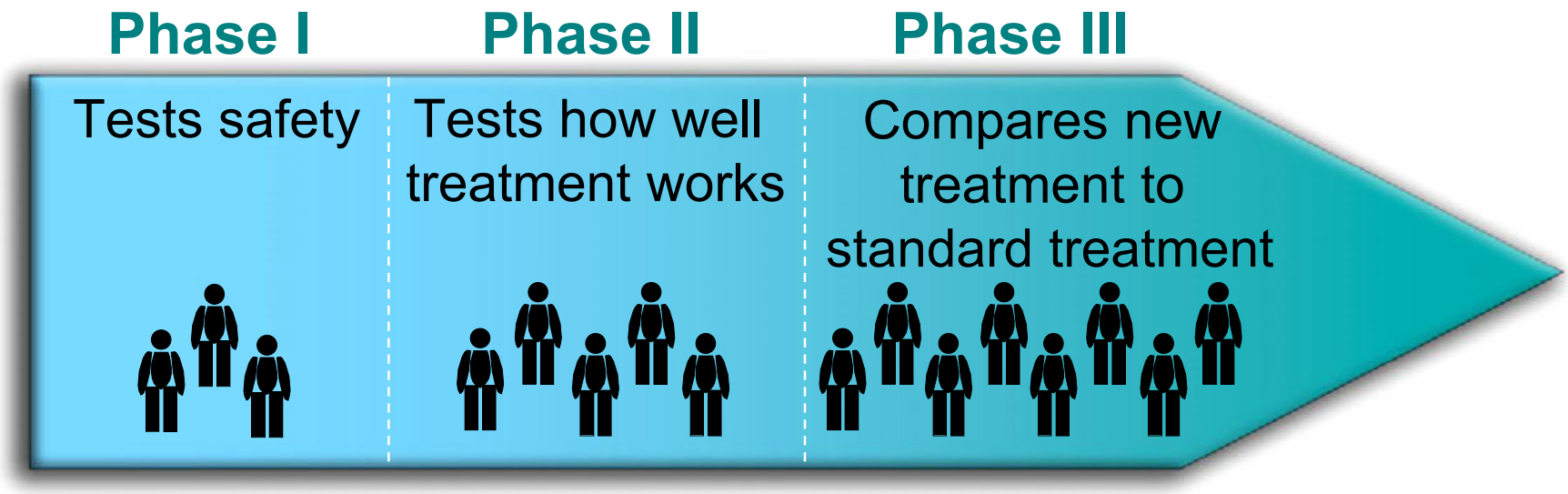
# Types of Trials

Phase 1: designed to test the safety of a drug (possibly efficacy)

Phase 2: test efficacy of established drug

Phase 3: test the agent in direct comparison with the current standard of care

# Clinical Trials in the Treatment of Myeloma



# Phase 1 Clinical Trials

- All patients receive the experimental therapy
- Phase 1 trials find the optimal dose of a new drug or drug combination
- Patients get higher doses as the study continues
- Determine side effects of new drugs or combinations
- Explore how the drug is metabolized by the body
- Important for all stages of myeloma

# Phase 2 Clinical Trials

- Determine if a new drug or combination is effective against the cancer
- May be added to a phase 1 study once the ideal dose is found
- Patients usually receive the experimental therapy
- In some cases, the study may include two “arms” comparing either two different doses or a different treatment (another combination of drugs)

# Phase 3 Clinical Trials

- Highest form of clinical evidence. Typically a large number of patients are required…usually required for FDA approval
- Patients receive either an experimental therapy (one or more drugs) or the current standard treatment
  - The patient is randomly assigned to a treatment—a process called randomization
  - Neither the physician or the patient can determine which treatment is given
- May be placebo controlled, if no standard treatments are available
- Very closely monitored for effectiveness and side effects

# Considering Entering a Clinical Trial?

- Discuss whether or not you are eligible for a clinical trial with your physician
- Work with your physician to determine the best trial for you
- Meet with the clinical research nurse or trials administrator to discuss the trial
- Carefully review the provided “Informed Consent”
  - Describes the study and any potential safety concerns related to the experimental medication


INFORMED CONSENT

# Commonly Asked Questions

How does the study work? How often will I need to see my doctor or visit the cancer center?

Will I need to undergo additional tests?

What is currently known about the new drug or combination?

What benefits can I expect?

What side effects should I expect? Who should I notify if I have side effects?

Can I take my vitamins or other medications?

Can I get the treatment with my local doctor?

Will my insurance pay for my participation in the clinical trial?

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# Other Types of Clinical Trials

## Longitudinal Studies

- Long-term studies with a large number of patients
- Usually to track outcomes of a large "cohort" of patients

## Registry Studies

- Patients are treated using available therapies
- Efficacy and safety are analyzed following treatment
- Typically involve a large number of patients

## Expanded Access Programs

- Allow early access to experimental therapies when no alternatives are available
- Often precedes formal approval of a drug

# Future Directions

- **Next generation of novel therapies in myeloma**
  - **Isatuximab** (SAR650984)/CD-38 Mab
  - Marizomib/high-potency proteasome inhibitor
  - Ricolinostat (ACY-1215)/selective histone deacetylase inhibitor
  - **Selinexor** (KPT-330)/selective inhibitor of nuclear transport
  - KPT-8602/selective inhibitor of nuclear transport
  - Filanesib (ARRY-520)/kinesin spindle kinase inhibitor
  - Indatuximab (BT-062)/anti CD-138 MAb/maytansinoid conjugate
  - **Venetoclax** (ABT-199)/Bcl-2 inhibitor
  - CC220 – next generation Immunomodulatory drugs
  - CC-122/pleiotropic pathway modulator
  - **Chimeric antigen receptor/CAR-T** (CD-19, BCMA)
  - Bispecific antibodies (CD3/CD38, CD138, CS1, BCMA)
  - BITE therapies – monoclonal Ab plus a T cell engager
  - Engineered autologous stem cell products
  - Engineered allogeneic stem cell products

# Promising agents

- Monoclonal Antibodies:  CD38, SLAMF6, others
- Immune Modulators – CC220
- Novel mechanisms: Venetoclax, Selinexor
- Immunotherapies: BCMA CAR-T, BITE

# Venetoclax

Not currently indicated in myeloma

May herald the first truly “targeted” therapy in MM

t(11;14) and BCL-2 expression

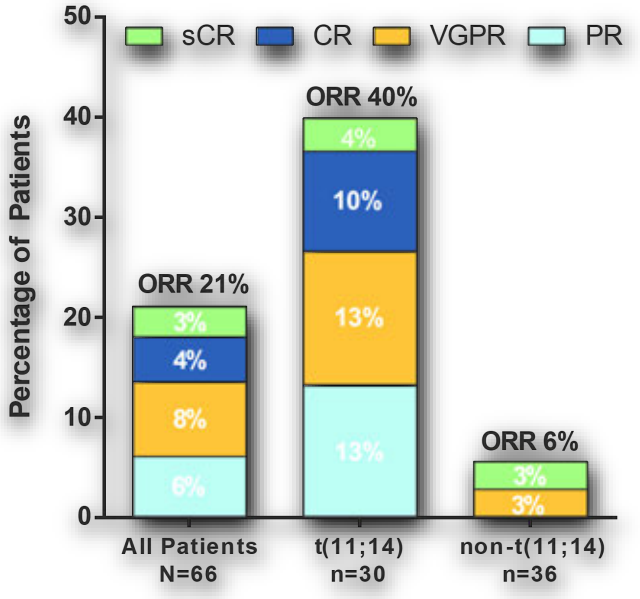
Recall that about 15% of patients have t(11;14) and even more have overexpression of BCL-2

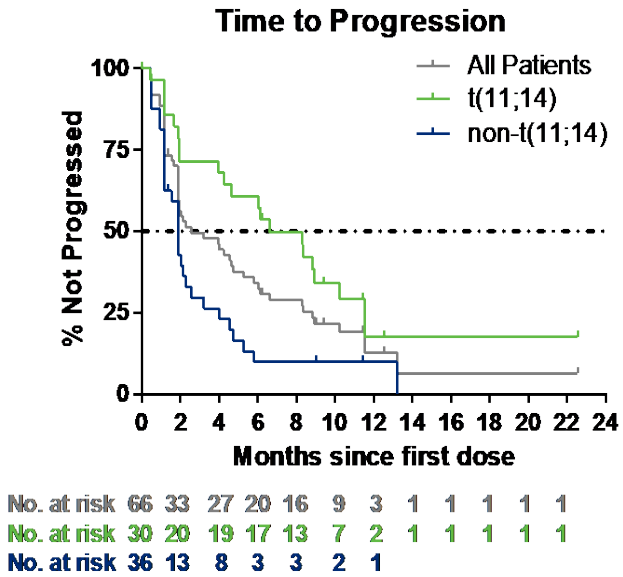
Very promising single agent trials, then with proteasome inhibitors…

# Venetoclax monotherapy: Ph1 in RRMM patients

30-1200 mg oral admin (MTD: 1200 mg)

66 pts after a median of 5 prior lines of therapy: 79% refractory to last line of therapy; 61% double refractory to bortezomib and lenalidomide




**Higher ORR (88% vs 20%) were seen in t(11;14) with a high BCL2:BCL2L1 ratio**

Main toxicities are thrombocytopenia (26% G3-4) and neutropenia (21% G3-4)

Serious AEs: pneumoniae (8%) and sepsis (5%)

Kumar, et al. Presented at ASH 2016 (Abstract 977), oral presentation

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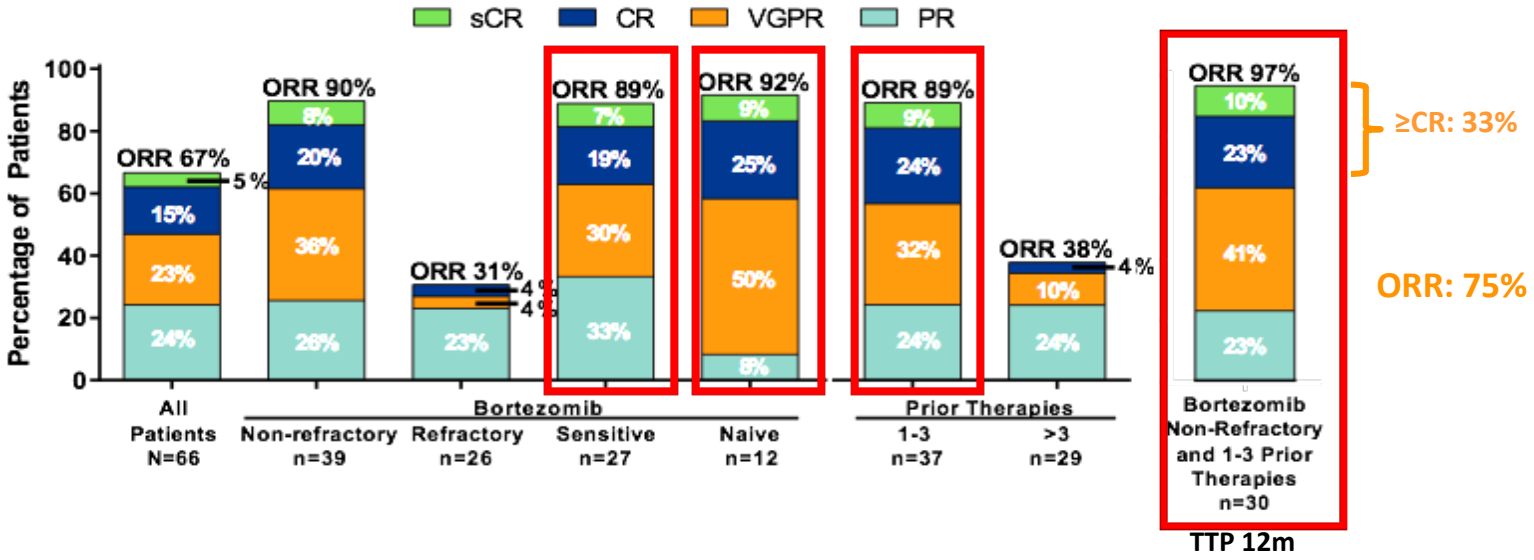
# Venetoclax plus bortezomib and dexamethasone

50-1200 mg oral daily + 1.3 mg/m$^2$ SC TW x cycles1-8, QW 9-11 + 20-20 mg (days 1,2,4,5,8,9,11,12) x cycles 1-8

66 patients after >=1 prior lines of therapy (median 3). 61% refractory to the last line




AEs were manageable. G3-4 AEs: Thrombocytopenia (29%), anemia (15%), neutropenia (14%), diarrea (6%), PN(3%), dyspnea (6%)

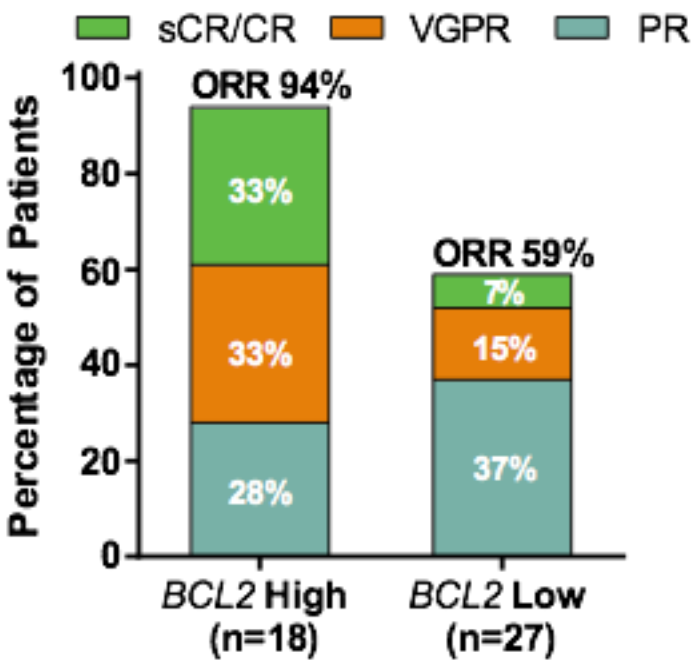
**Rationale for a phase 3 trial: Vd +/- Venetoclax**

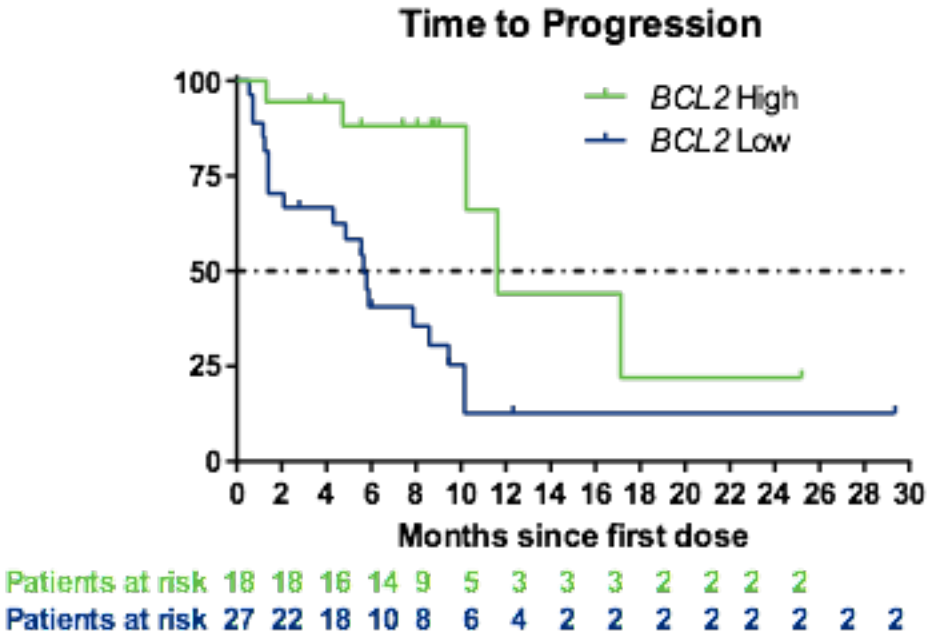

Moreau P, et al. Presented at ASH 2016 (Abstract 975), oral presentation
Dimopoulos MA; Haematologica 2015; Epub 2014 Sep 26.

# Venetoclax plus bortezomib and dexamethasone

50-1200 mg oral daily + 1.3 mg/m$^2$ SC TW x cycles1-8, QW 9-11 + 20-20 mg x cycles 1-8

## BCL2 Gene Expression and Clinical Response

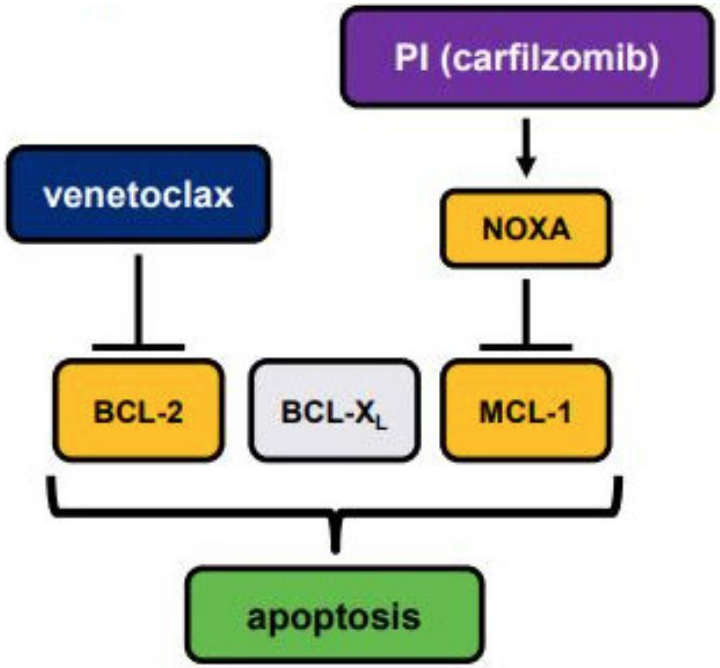



*BCL2* quantitation using ddPCR performed on CD138-selected bone marrow mononuclear cells collected at baseline. BATTing was used to estimate a threshold of *BCL2* to provide optimum selection of patients likely to have a response.

Moreau P, et al. Presented at ASH 2016 (Abstract 975), oral presentation

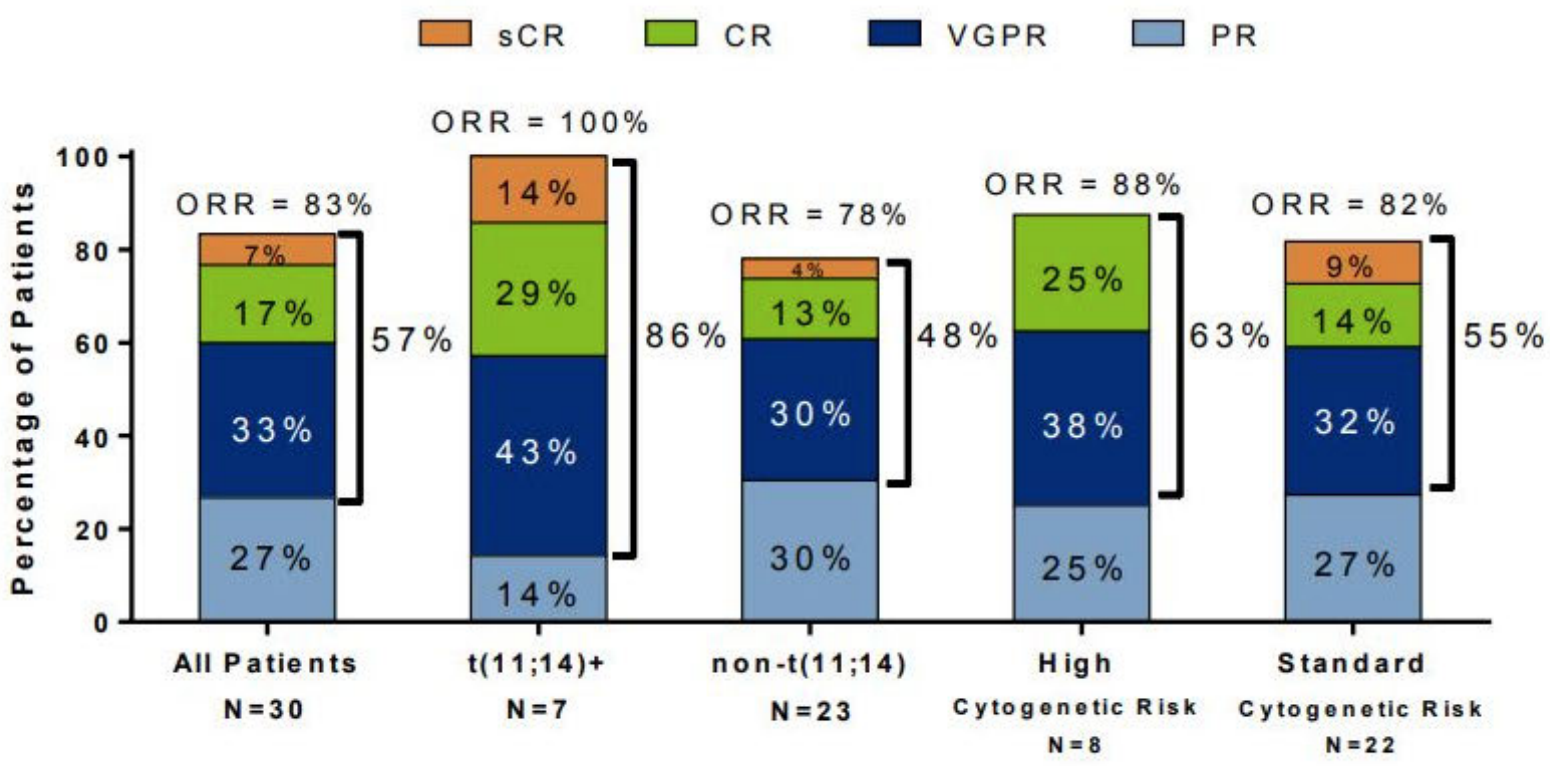
# Synergy Between Carfilzomib and Venetoclax



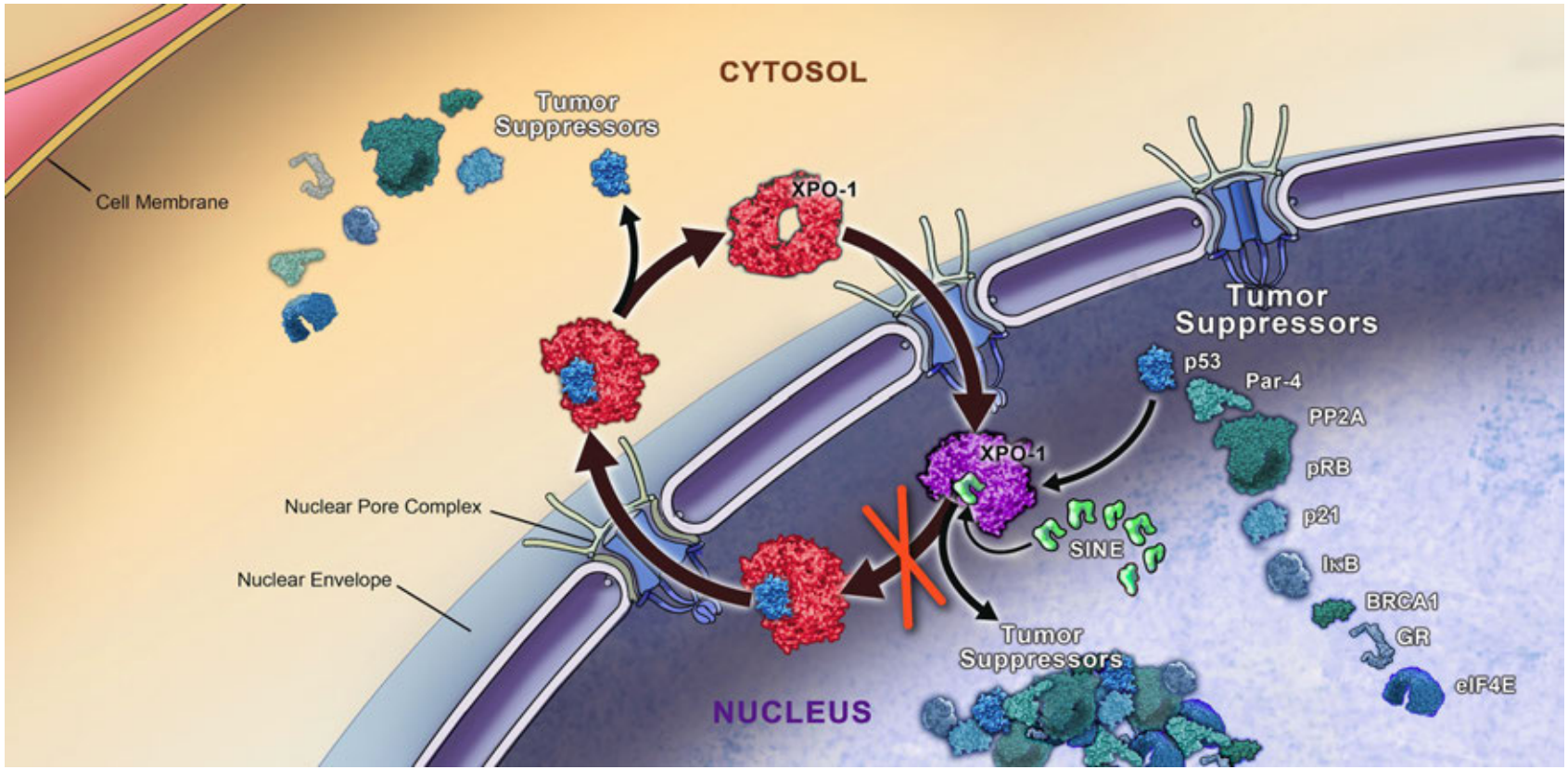

**7.** Roberts AW et al. N Eng J Med 2015; 374:311-22. **8.** Fan F et al. Cancer Lett 2014;343(2):286-94. **9.** Ponder K., et al. Cancer Bio & Ther 2016; 17(7):769-777

# Venetoclax and Carfilzomib

## Objective Responses in Patients Based on Cytogenetic Risk Status



# **Selinexor:** Novel Oral Anti-Cancer Agent
# Restores Tumor Suppressors & Reduces Oncoproteins




**Selinexor and Low Dose Dexamethasone (Sd) in Patients with Lenalidomide, Pomalidomide, Bortezomib, Carfilzomib & anti-CD38 mAb Refractory MM: STORM Study**

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# Selinexor and Low Dose Dexamethasone (Sd) in Patients with Lenalidomide, Pomalidomide, Bortezomib, Carfilzomib & anti-CD38 mAb Refractory MM: STORM Study

**Dan T. Vogl**, D. Dingli, RF. Cornell, CA. Huff, S. Jagannath, D. Bhutani, R. Baz, A. Nooka, J. Richter, C. Cole, R. Vij, A. Jakubowiak, R. Abonour, G. Schiller, TL. Parker, LJ. Costa, D. Kaminetzky, J. Hoffman, AJ. Yee, A. Chari, D. Siegel, R. Fonseca, S. VanWier, G. Ahmann, I. Lopez, M. Kauffman, S. Shacham, JR. Saint-Martin, C. Picklesimer, C. Choe-Juliak, and A. Keith Stewart

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# Independent Review Committee (IRC) Assessed Efficacy

| Category | N* | ORR (%) | CBR (%) | VGPR (%) | PR (%) | MR (%) | SD (%) | PD (%) | NE (%) |
|---|---|---|---|---|---|---|---|---|---|
| Overall | 78 | **16 (21%)** | 26 (33%) | 4 (5%) | 12 (15%) | 10 (13%) | 27 (35%) | 9 (12%) | 16 (21%) |
| Quad Refractory | 48 | **10 (21%)** | 14 (29%) | 2 (4%) | 8 (17%) | 4 (8%) | 21 (44%) | 4 (8%) | 9 (19%) |
| Penta Refractory | 30 | **6 (20%)** | 12 (40%) | 2 (7%) | 4 (13%) | 6 (20%) | 6 (20%) | 5 (17%) | 7 (23%) |
| 6 Doses / Month | 51 | **10 (20%)** | 15 (29%) | 3 (6%) | 7 (14%) | 5 (10%) | 21 (41%) | 4 (8%) | 11 (22%) |
| 8 Doses / Month | 27 | **6 (22%)** | 11 (41%) | 1 (4%) | 5 (19%) | 5 (19%) | 6 (22%) | 5 (19%) | 5 (19%) |

*1 patient did not have measurable disease at baseline

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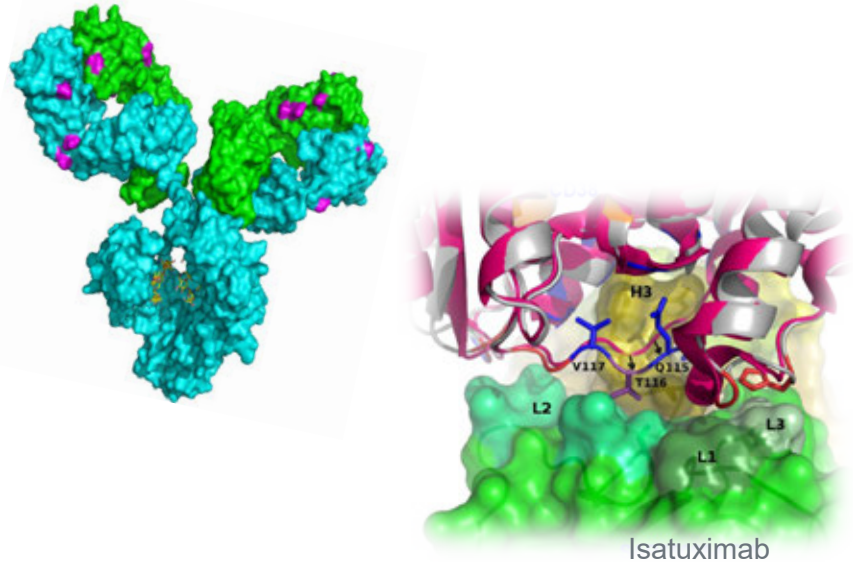
# Isatuximab: An anti-CD38 monoclonal antibody

Isatuximab, a humanized IgG1 mAb, has distinctive properties compared with other anti-CD38 antibodies

1. Isatuximab targets a unique epitope on CD38, distinct from the binding sites of other anti-CD38 mAbs[1]
2. Isatuximab is a potent inhibitor of CD38 enzyme activity and works via an allosteric mechanism[1]
3. Isatuximab can induce apoptosis in the absence of cross-linking agents[1]
4. Binding studies suggest limited internalization and that most isatuximab remains bound on the cell surface[2]



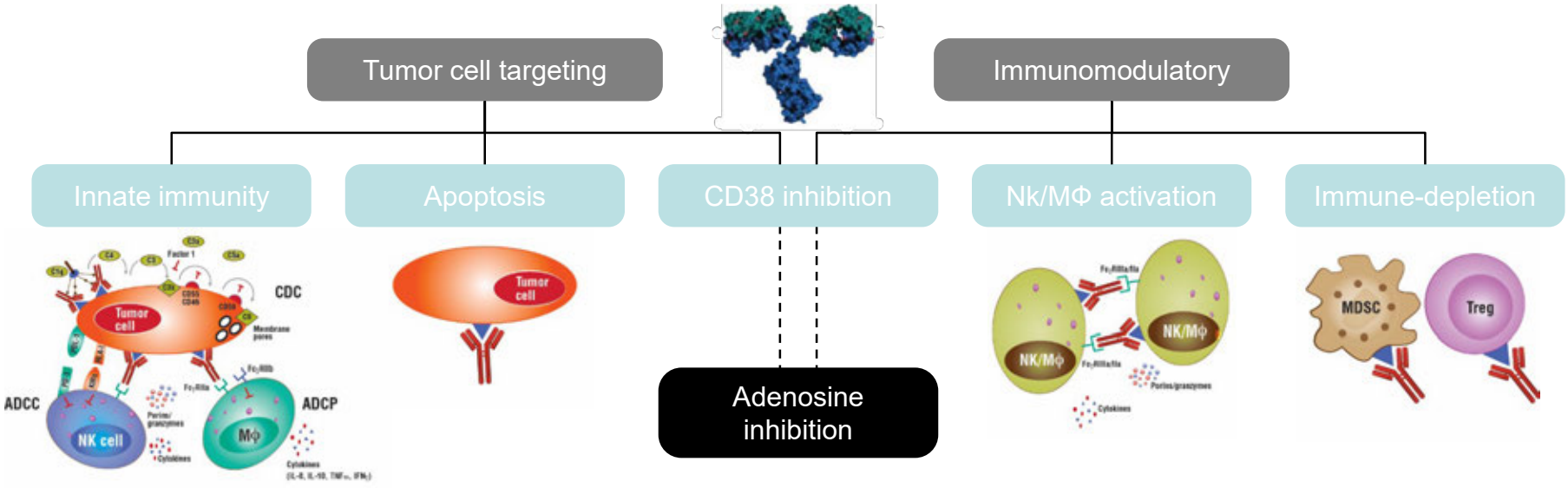
Isatuximab

mAb, monoclonal antibody

1. Deckert J, et al. Clin Cancer Res 2014;20:4574–83; 2. Sanofi data on file

# Isatuximab: Multiple mechanisms of action

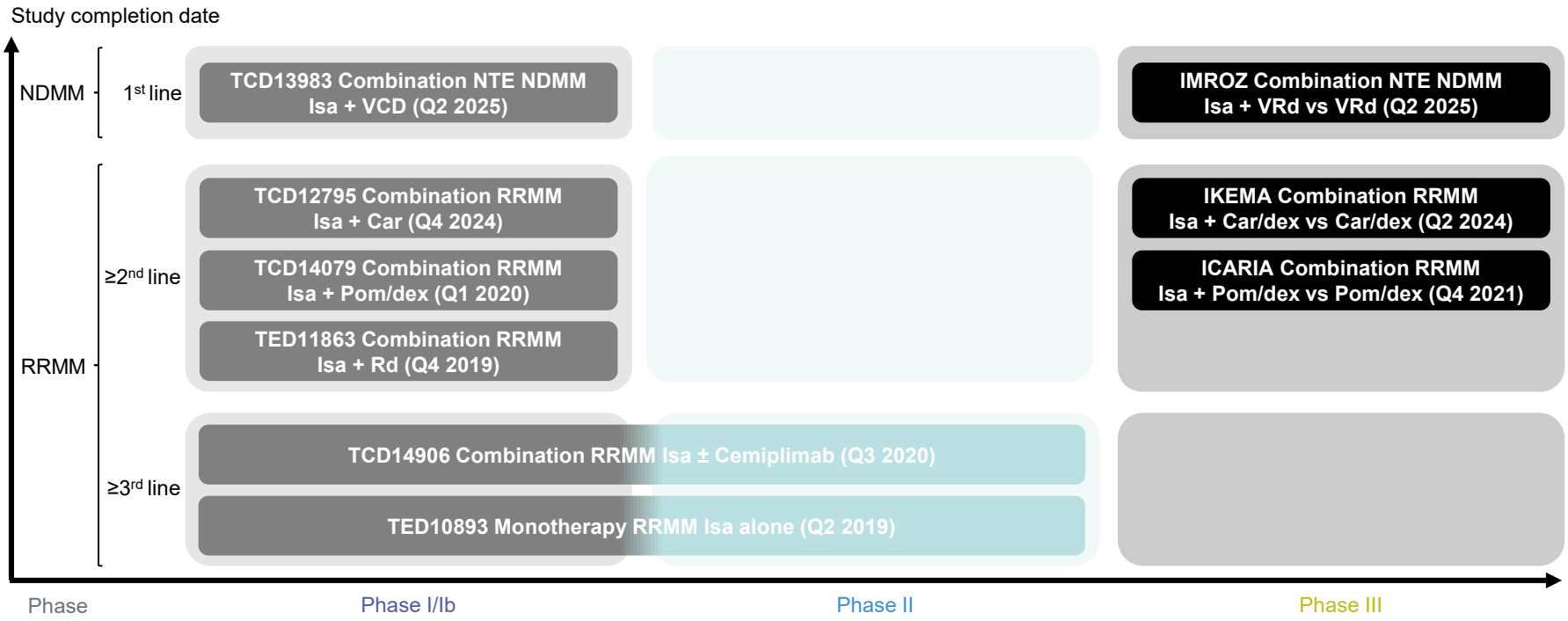
Preclinical studies suggests that NK cell-mediated ADCC is the most important mechanism of action contributing to the efficacy of isatuximab[1]




ADCC, antibody-dependent cell-mediated cytotoxicity; ADCP, antibody-dependent cell-mediated phagocytosis; CDC, complement-dependent cytotoxicity; IFN, interferon; IL, interleukin; Mφ, macrophage; MDSC, myeloid-derived suppressor cell; NK, natural killer cell; TNF, tumor necrosis factor; Treg, regulatory T-cell

Moreno L, et al. Blood 2016;128:2105

# Isatuximab clinical development




C, cyclophosphamide; Car, carfilzomib; D/d/dex, dexamethasone; Isa, isatuximab; NDMM, newly diagnosed multiple myeloma; NTE, non-transplant eligible; Pom, pomalidomide; R, lenalidomide; RRMM, relapsed/refractory multiple myeloma; V, bortezomib

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# Phase Ib
## TCD14079 Isa plus Pom/dex combination: Study design

**Standard dose escalation (3 + 3 design)**

Isatuximab IV QW Cycle 1, then Q2W (per 28-day cycle)

Cohort 1
5 mg/kg
(N=8)

Cohort 2
10 mg/kg
(N=9)

Cohort 3
20 mg/kg
(N=6)

**Expansion cohort**
**22 patients**

Based on PK/PD modeling and simulations, isatuximab 10 mg/kg QWx4/Q2W was chosen as the recommended dose

Pomalidomide 4 mg (Days 1–21 per 28-day cycle)
Dexamethasone 40 mg (20 mg if ≥75 years) QW (Days 1, 8, 15 & 22 per 28-day cycle)

Treatment continued until unacceptable toxicity, progressive disease or patient withdrawal
To mitigate IARs the protocol mandated use of standard premedications

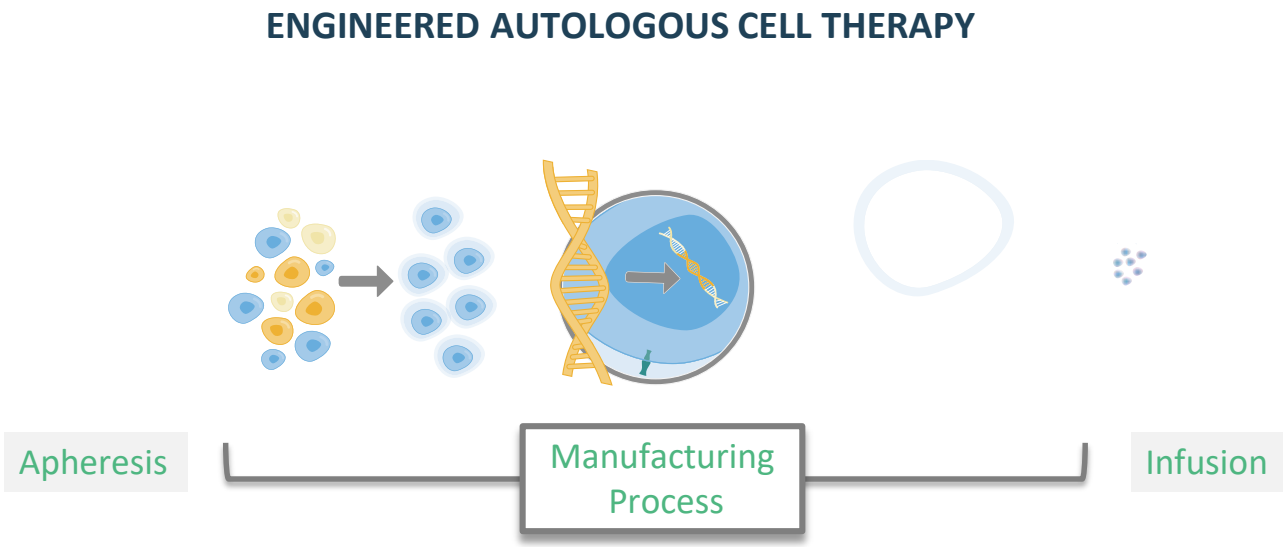
dex, dexamethasone; IAR, infusion-associated reaction; Isa, isatuximab; IV, intravenous; PD, pharmacodynamics; PK, pharmacokinetics; Pom, pomalidomide; QW, weekly; Q2W, every 2 weeks



Mikhael J, et al. Presented at: ASCO; Jun 1–6, 2018; Chicago, IL

# CAR T Cell Therapy

# Harnessing the Power of a Patient's Own Immune System to Target and Kill Myeloma Cells



# BCMA CAR T Myeloma Trial Data

| | MSK | U Penn | bb2121 | LCAR-B38M |
|---|---|---|---|---|
| **Source** | Phase I Interim Analysis ASH 2017 | Phase 1 Interim Analysis ASH 2017 | Phase 1 Interim Analysis ASCO 2018 | Phase I Interim Analysis EHA 2017 |
| **Enrollment** | 6 | 28 (24 evaluable) | 43 (39 evaluable) | 40 (22 evaluable) |
| **Efficacy** | **ORR 3 (50%)**<br>VGPR 2 | **ORR 11 (46%)**<br>CR/sCR 2, VGPR 3 | **ORR 30 (77%)**<br>CR/scR 17, VGPR 9 | **ORR 22 (100%)**<br>sCR 14, VGPR 4 |
| **Safety** | • Any CRS: 3 (50%)<br>• ≥ Gr 3 CRS: 0 ≥ Gr 3 NE: 0 | • Any CRS: 20 (83%)<br>• ≥ Gr 3 CRS: 8<br>• ≥ Gr 3 NE: 3<br>2 DLT: PRES, Pleural hemorrh | • Any CRS: 27 (63%)<br>• ≥ Gr 3 CRS: 2 (5%)<br>• ≥ Gr 3 NE: 1 | • Any CRS: 28 (85%)<br>• ≥ Gr 3 CRS: 3 (8.6%)<br>• ≥ Gr 3 NE: 0 |
| **Toci-Steroid Use** | • Toci: 2<br>• Steroid: 0 | • Toci/Silt: 6 | • Toci: 9<br>• Steroid: 4 | |

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**ABSTRACT 8007**

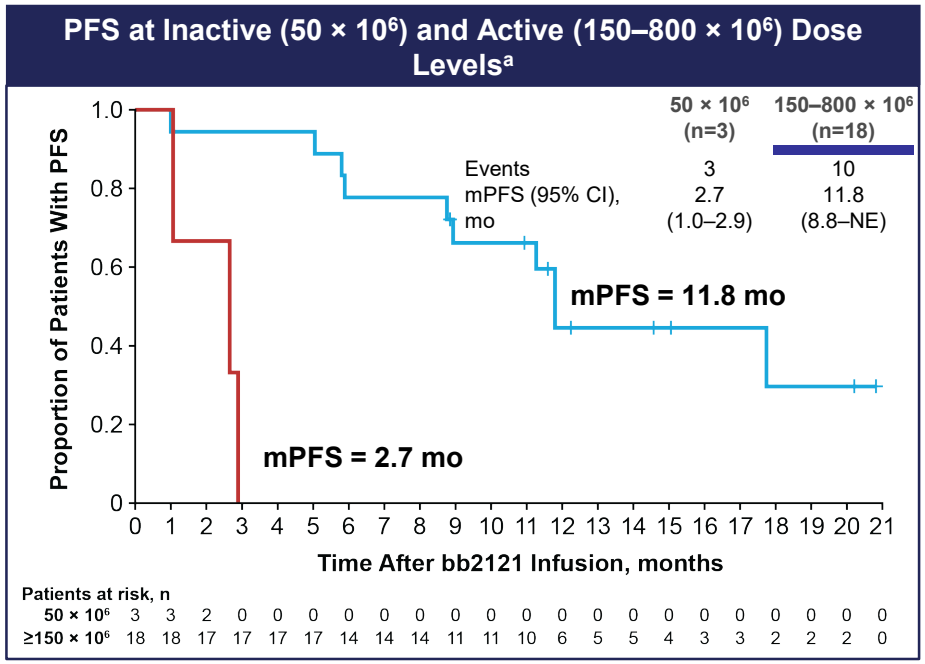
# bb2121 Anti-BCMA CAR T Cell Therapy in Patients With Relapsed/Refractory Multiple Myeloma: Updated Results From a Multicenter Phase I Study

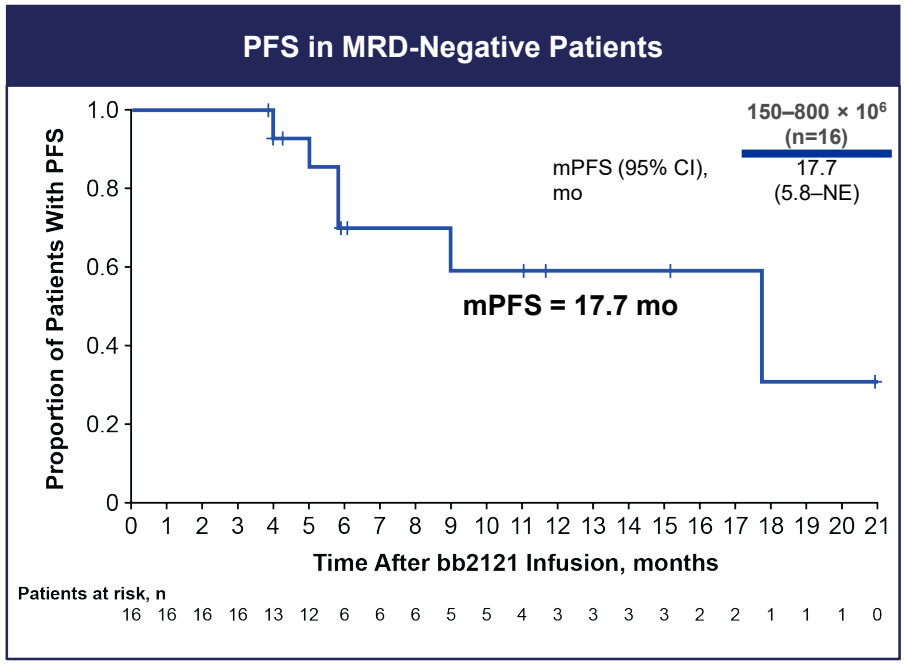
Noopur Raje, MD,[1] Jesus Berdeja, MD,[2] Yi Lin, MD, PhD,[3] Nikhil Munshi, MD,[4] David Siegel, MD, PhD,[5] Michaela Liedtke, MD,[6] Sundar Jagannath, MD,[7] Deepu Madduri, MD,[7] Jacalyn Rosenblatt, MD,[8] Marcela Maus, MD, PhD,[1] Ashley Turka,[9] Lyh Ping Lam, PharmD,[9] Richard A. Morgan, PhD,[9] M. Travis Quigley,[9] Monica Massaro, MPH,[9] Kristen Hege, MD,[10] Fabio Petrocca, MD,[9] and James N. Kochenderfer, MD[11]

*[1]Massachusetts General Hospital Cancer Center, Boston, MA; [2]Sarah Cannon Research Institute and Tennessee Oncology, Nashville, TN; [3]Mayo Clinic, Rochester, MN; [4]Dana-Farber Cancer Institute, Boston, MA; [5]Hackensack University Medical Center, Hackensack, NJ; [6]Stanford University Medical Center, Palo Alto, CA; [7]Mount Sinai Medical Center, New York, NY; [8]Beth Israel Deaconess Medical Center, Boston, MA; [9]bluebird bio, Inc, Cambridge, MA; [10]Celgene Corporation, San Francisco, CA; [11]Experimental Transplantation and Immunology Branch, National Cancer Institute/National Institutes of Health, Bethesda, MD*

# PROGRESSION-FREE SURVIVAL

- mPFS of 11.8 months at active doses (≥150 × $10^6$ CAR+ T cells) in 18 subjects in dose escalation phase
- mPFS of 17.7 months in 16 responding subjects who are MRD-negative




Data cutoff: March 29, 2018. Median and 95% CI from Kaplan-Meier estimate. NE, not estimable. [a]PFS in dose escalation cohort.

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# OVERALL SUMMARY

**bb2121 at active doses (≥150 × $10^6$ CAR+ T cells) induces deep and durable responses in a heavily pretreated population with R/R MM**

- Median PFS of 11.8 months for patients in the dose escalation cohort
- MRD-negative results in 100% of 16 evaluable responding patients; median PFS of 17.7 months
- Comparable ORR in patients with low and high BCMA-expressing MM
- Dose response relationship observed across the active dose ranges
- Higher peak CAR T expansion in responders versus nonresponders

**To date, the safety profile of bb2121 has been manageable at doses as high as 800 × $10^6$ CAR+ T cells**

- Mostly grade 1/2 CRS observed with infrequent tocilizumab and corticosteroid use
- The 2 events of grade 3 CRS resolved within 24 hours
- 1 case of reversible grade 4 neurotoxicity without additional events during expansion

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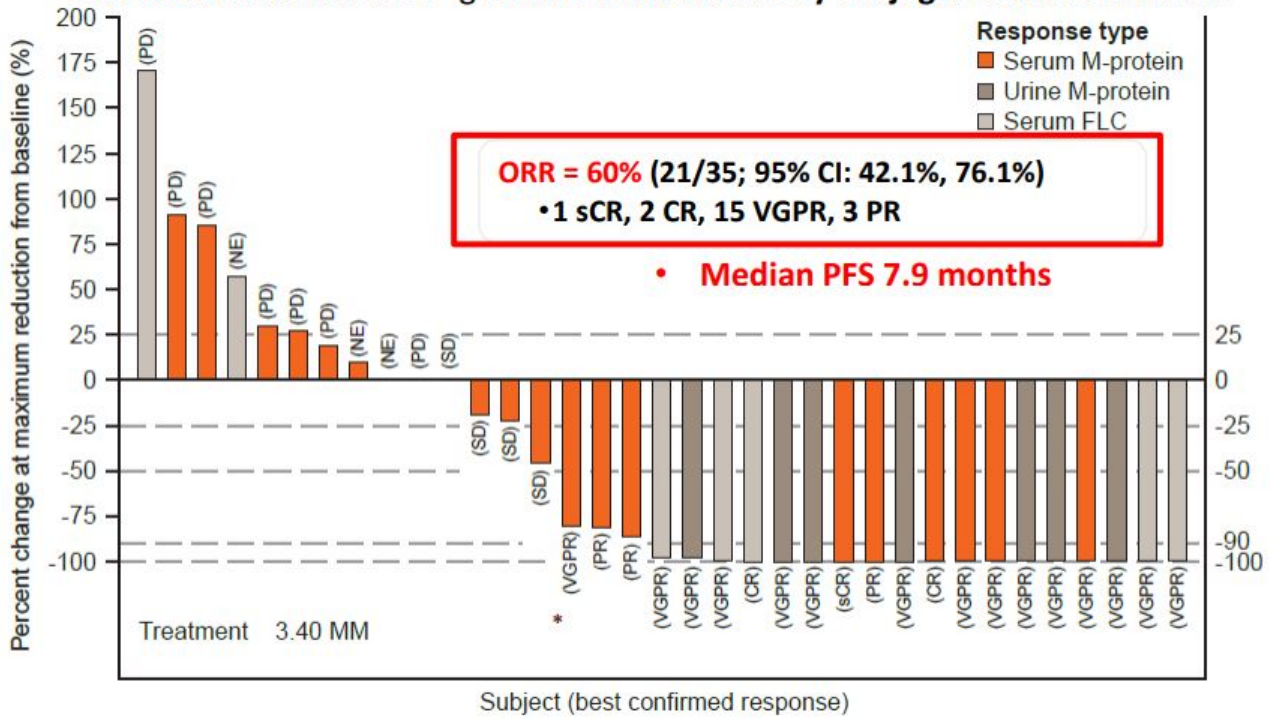
# GSK 7916 – Another way to target BCMA

▪**GSK2857916:** humanized IgG1 **anti-BCMA antibody conjugated with Auristatin**




*One patient with a VGPR had a <90% reduction in serum M-protein due to missing laboratory data, which was confirmed by investigators as too small to quantify after the data cut-off

CI, confidence interval; CR, complete response; FLC, free light chain; M-protein, myeloma protein; ORR, overall response rate; PD, progressive disease; PR, partial response; sCR, stringent complete response; SD, stable disease; VGPR, very good partial response

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# Clinical Trials

- Information available at:

  www.clinicaltrials.gov

- Currently 2400 listed under myeloma!
  - 448 are currently accruing
    - 292 in the US
    - 33 in Canada

# IMF

IMF Website for clinical trials

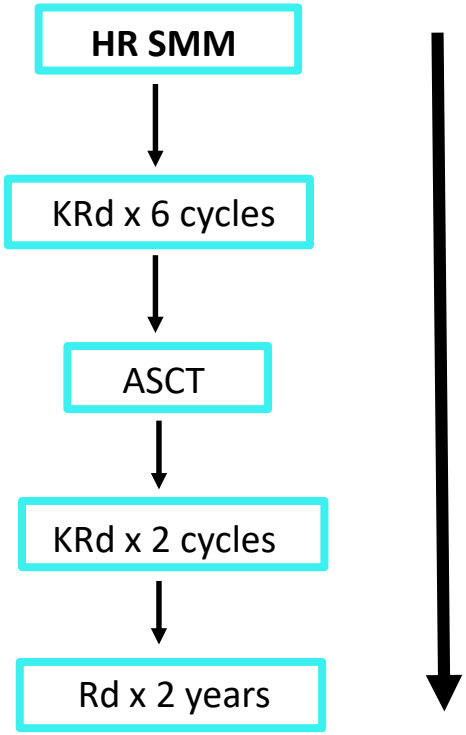
www.myeloma.org

- Also note the Clinical Trial “Fact Sheets”

- The IMF now conducts trials too!
  - ASCENT in high risk smoldering disease - Europe
  - CESAR in high risk smoldering disease - USA
  - iSTOP MM in Iceland

# European "CURE" Trials: CESAR

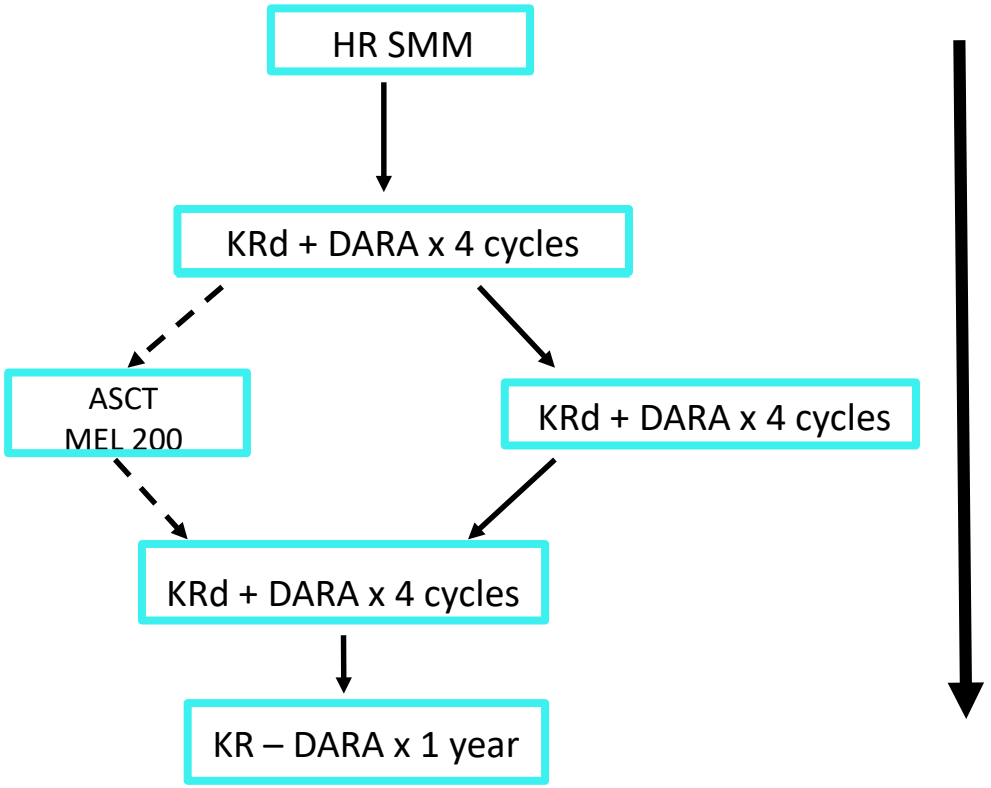
**C**urative **E**strategy **S**moldering **A**lto **R**isk




**MRD at CR**

**ASH abstract #402: 2017**

# US "CURE" Trial: ASCENT




US Sites

- Mayo
- University of Indiana
- University of Maryland
- MDAH
- Swedish Seattle
- Emory
- Chicago
- Cornell
- North Carolina
- Columbia
- Wisconsin
- Kansas

# Iceland iSTOP MM Trial

Now 2years into study….

Over 85,000 patients consented!

20-30 new MGUS, SMM and MM patients identified weekly

Early data suggests MGUS and high risk SMM more common than expected

Mass Spectrometry information will be highly informative

With genetic information available for much of population – ability to find driver mutations genuinely feasible

**Similar strategy in the US with Stand Up 2 Cancer grant JUST awarded to Ghobrial, Borello, Mikhael et al…**

# Optimizing Communication With Patients


- Basch E, et al. *JAMA.* 2017;318:197-198.

# Thank YOU!

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**Professor, Translational Genomics Research Institute (TGen)**
**City of Hope Cancer Center**

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