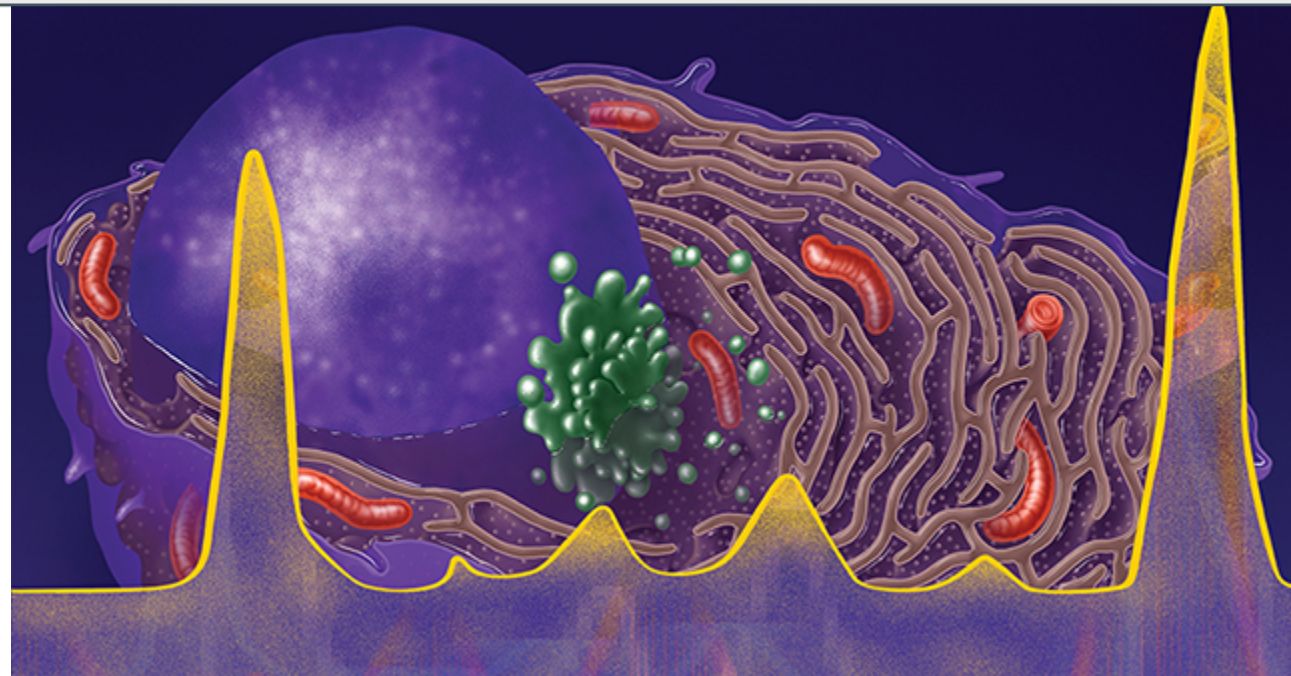
INTERNATIONAL MYELOMA FOUNDATION

CCO LIVE

Advances in the Optimal Choice of Therapeutic Strategies for Patients With R/R Myeloma

Faculty Presenters:

Jesús F. San-Miguel, MD, PhD



This activity is supported by educational grants from AbbVie; Amgen; Bristol-Myers Squibb; Celgene Corporation; Janssen Biotech, Inc., administered by Janssen Scientific Affairs, LLC; and Takeda Oncology.

Program Faculty

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Director of Clinical and Translational Medicine
Universidad de Navarra
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Jesús F. San-Miguel, MD, PhD, has disclosed that he has received consulting fees from Amgen, Bristol-Myers Squibb, Celgene, Janssen, MSD, Novartis, Roche, Sanofi, and Takeda.

Therapeutic Strategies at Relapse in Multiple Myeloma

Jesus San-Miguel
Universidad Navarra

Patient Case Example

- A 66-year-old woman was diagnosed with IgG lambda **R-ISS stage II** myeloma
  - BM showed 60% PC with **1q gain plus t(4;14)**
  - MC: 43 g/L; **Hb: 10.3 g/dL**, creatinine: 1.2 mg/dL; calcium: 9.2 mg/dL
  - She had extensive **bony disease**
- She was treated **VTD + ASCT + lenalidomide for 2 years** and achieved **sCR**
- **After 4 years,** she relapsed

How would you treat this patient?

Faculty	Recommendation
Brian G.M. Durie, MD	Daratumumab/bortezomib/dexamethasone
Shaji Kumar, MD	Daratumumab/lenalidomide/dexamethasone
Philippe Moreau, MD	Daratumumab/lenalidomide/dexamethasone
S. Vincent Rajkumar, MD	Daratumumab/lenalidomide/dexamethasone
Jesús F. San-Miguel, MD, PhD	Rescue treatment followed by second ASCT

66-Year-Old Man Relapsing After VTD + ASCT + Len x 2-Yrs: How to Make the RIGHT CHOICE?

Decisions based on the **duration of the previous response**

Late relapse (> 3-4 years post ASCT)

- ***Aggressive relapse:*** **Reinduction (VRD/KRD +/- Dara) + 2nd ASCT**
- ***Biochemical relapse*****: Repeat the initial approach or same as above**

Early relapse (< 1 year post ASCT)

"Overcome drug resistance"

Combination of non cross-resistant agents

VRD (KRD)-PACE + Dara → RIC-Allo/CAR-T

Intermediate relapse (1-3 years post ASCT)

"Prolong survival until curative treatments are developed"

Sequential novel agent combinations: Dara + PomDex.....KRD...

Patient Case Example, Continued

- A 66-year-old woman was diagnosed with IgG lambda R-ISS stage II MM
  - BM showed **1q gain plus t(4;14)** with extensive **bony disease**
  - She was treated VTD + ASCT + lenalidomide for 2 years and achieved sCR
  - After 4 years, she relapsed
- She refused 2nd ASCT (70 years, with hypertension) and was treated with **VCD x 8 cycles** and **achieved CR**
- **She relapsed 10 months later**

How would you treat this patient?

Faculty	Recommendation
Brian G.M. Durie, MD	Daratumumab/lenalidomide/dexamethasone
Shaji Kumar, MD	Daratumumab/lenalidomide/dexamethasone
Philippe Moreau, MD	Daratumumab/lenalidomide/dexamethasone
S. Vincent Rajkumar, MD	Daratumumab/lenalidomide/dexamethasone
Jesús F. San-Miguel, MD, PhD	Daratumumab/lenalidomide/dexamethasone

Lenalidomide-Based Regimens: Efficacy

Efficacy	POLLUX (n=569) DaraRd vs Rd [1-3]	ASPIRE (n=792) KRd vs Rd [4,5]	ELOQUENT-2 (n=646) ERd vs Rd [6]	TOURMALINE-MM1 (n=722) IRd vs Rd [7]
PFS HR (▲m)	**0.44** (▲ 27) **44.5** vs 17.5 m	**0.67**(▲ 8.7 m) **26.3** vs 17.6 m	**0.71** (▲ 4.5 m) **19.4** vs 14.9 m	**0.74**(▲ 5.9 m) **20.6** vs 14.7 m
ORR, %	93	87	79	78
≥ CR, %	**51**	**32**	**5**	**14**
OS HR (95% CI)	**0.63**	**0.79** (▲ 8 m) 48 vs 40 m	**0.78** (▲ 4.1 m) 43.7 vs 39.6 m	**NE**
High Risk: m (HR)	**22.6** (0.64)	**23** (0.70)	**19** (0.60)	**21** (0.54)

This table is provided for ease of viewing information from multiple trials with different patient populations. Direct comparison across trials is not intended and should not be inferred.
DOR, duration of response; NE, not evaluated.

1. Bahlis NJ, et al. ASH 2018; abstract 1996. 2. Dimopoulos M, et al. Poster presented at EHA 2017; abstract P334.
3. Usmani SZ, et al. Oral presentation at ASH 2016; abstract 1151. 4. Siegel DS, et al. Poster presented at EHA 2017; abstract P333.
5. Stewart AK, et al. N Engl J Med. 2015;372:142-52. 6. Lonial S, et al. NEJM 2015,373:621-31; Oral presentation at ASCO 2017; abstract 8028.
7. Moreau P, et al. N Engl J Med. 2016;374:1621-34. 8. Dimopoulos MA, et al. N Engl J Med. 2016;375:1319-31.
9. Dimopoulos MA, et al. Br J Haematol. 2017;178:896-905.

Patient Case Example

- A 66-year-old woman was diagnosed with IgG lambda R-ISS stage II MM
  - BM showed **1q gain plus t(4;14)** with extensive **bony disease**
  - She was treated VTD + ASCT + lenalidomide for 2 years and achieved sCR
  - After 4 years, she relapsed
- She refused 2nd ASCT (70 years, with hypertension) and was treated with VCD x 8 cycles and achieved CR
- She relapsed 10 months later
- She began tx with **lenalidomide/dexamethasone** until progression
  - On cycle 5, she was already in VGPR and maintained her response for 15 months before relapse

How would you treat this patient?

Faculty	Recommendation
Brian G.M. Durie, MD	Elotuzumab/pomalidomide/dexamethasone
Shaji Kumar, MD	Daratumumab/bortezomib/dexamethasone
Philippe Moreau, MD	Daratumumab/bortezomib/dexamethasone
S. Vincent Rajkumar, MD	Daratumumab/bortezomib/dexamethasone
Jesús F. San-Miguel, MD, PhD	Daratumumab/bortezomib/dexamethasone

70-Yr-Old Woman 1st Relapse Following Continuous Lenalidomide-Dex

Proteasome Inhibitors-Based Regimens: Efficacy

Efficacy	ENDEAVOR (n=929) **Kd vs Vd** [3]	CASTOR (n=499) **DaraVd vs Vd** [1-2]	OPTIMISMM (n=559) **PVd vs Vd** [4]	PANORAMA-1 (n=768) **PanoVd vs Vd** [5]
PFS HR	**0.53** (▲ 9.3 m) **18.7** vs 9.4 m	**0.32** (▲ 9.6 m) **16.7** vs 7.1 m	**0.61** (▲ 4.1 m) * **11.2** vs 7.1 m	**0.63** (▲ 4 m) * **12** vs 8 m
ORR, %	77	85	82.2	60.7
≥ CR, %	**13**	**30**	**15.7**	**27.6**
OS HR (95% CI)	**0.79** (▲ 7.6 m) **47.6 vs 40 m**	--	--	--
Len Refract	24% (8.6m)	18% (9.3m)	71% (9.5m)	<10%
High Risk: m (HR)	**8.8** (0.73)	**11.2** (0.45)	**8.4** (0.56)	NA

1. Palumbo et al. NEJM 2016. 2. Spencer A et al. ASH 2017. Poster presentation. Abstract 3145 ; 3. Dimopoulos MA et al, Lancet Oncology 2016; 4. Richardson P, et al. ASCO 2018 5. San Miguel et al . Lancet Oncology 2014

Patient Case Example

- A 66-year-old woman was diagnosed with IgG lambda R-ISS stage II MM
  - BM showed **1q gain plus t(4;14)** with extensive **bony disease**
  - She was treated VTD + ASCT + lenalidomide for 2 years and achieved sCR but relapsed after 4 years
- She refused 2nd ASCT (70 years, with hypertension) and was treated with VCD x 8 cycles and achieved CR
  - She relapsed 10 months later
- She began tx with lenalidomide/dexamethasone until progression
  - She achieved VGPR but relapsed 15 months later
- **She received Dara-Vd and achieved PR on C2 but progressed with extramedullary disease on C8**

Let’s Vote!

INTERNATIONAL
MYELOMA
FOUNDATION

How would you treat this patient now?

Faculty	Recommendation
Brian G.M. Durie, MD	Carfilzomib/pomalidomide/dexamethasone
Shaji Kumar, MD	Elotuzumab/pomalidomide/dexamethasone
Philippe Moreau, MD	Clinical trial with BCMA CAR T-cell therapy
S. Vincent Rajkumar, MD	Carfilzomib/pomalidomide/dexamethasone
Jesús F. San-Miguel, MD, PhD	Elotuzumab/pomalidomide/dexamethasone

Treatment at 3rd/subsequent relapses

Poma – Dexa[1] *(backbone)*
ORR: 31%; PFS 4 m; OS: 13.1 m

PCyDex (ORR 65%; PFS 9.5m)[2]
EloPom Dex (ORR:53%, PFS 10.2m)[3]

Daratumumab[5]
ORR: 31%; PFS 4m; OS 20.1 m

DaraCfzDex *

DaraPomDex (ORR 60%, PFS 8.8m)
IxaPomDex (ORR 55%)[4]

CfzPomDex **n=(60) EMN011**[7]
- **ORR: 87% (31%CR)**
- **PFS: 18m**

*DaraCfzDex **n=85 (60% Len Ref)**[6]
- **ORR: 86% (81%)**
- **PFS: 71% at 12m (14.1m)**

[1]San-Miguel et al Lancet Oncology 2013;14(11):1055-66; [2]*Baz et al. Blood. 2016;127(21):2561-2568;* [3]Dimopoulos NEJM2018, 379:1811-22. [4] ***Voorhees PM, ASH 2015 Abst 375;*** [5]**Usmani S, et al. Blood 2016; 6 Lonial ASH 2017 1869; 7 Sonneveld ASH 2018, (Abstr 801)**

Elotuzumab-Poma-Dexa vs Poma-Dex in RRMM: Phase 2 Randomized ELOQUENT-3 Trial – Efficacy (N = 117)

KEY INCLUSION

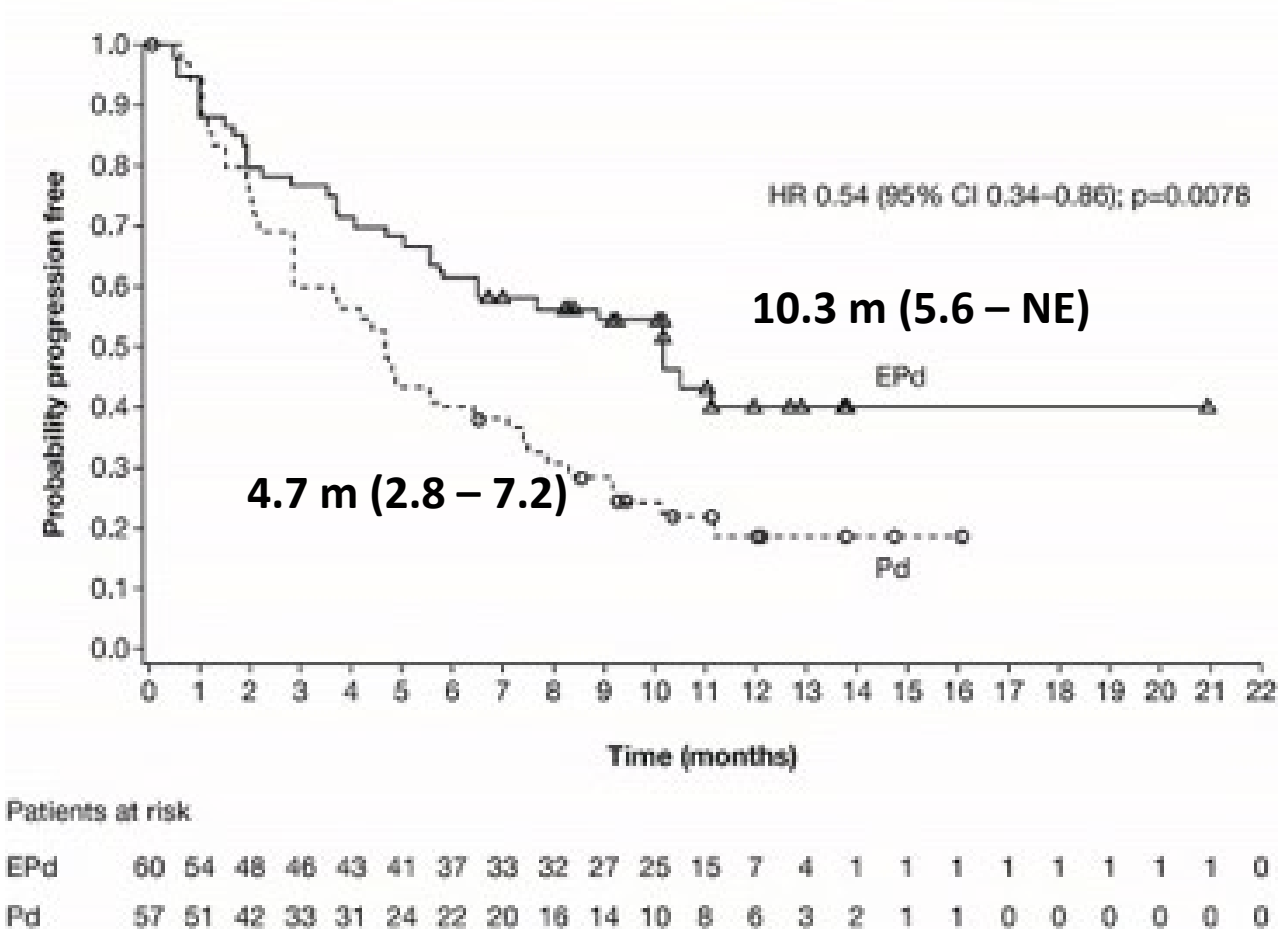
- ≥ 2 prior regimens
- Prior IMID and PI treatment
- Refractory to last line
- Refractory to Len and a PI

POM:4 mg days 1-21 ; 40 mg (20 of >75y) weekly

ELO: 10 mg/kg/w C1&C2; >C3: 20mg/kg/ 4 w

- **Median number of prior lines: 3 (2 – 8)**
- Prior exposure to: BORT (100%), CFZ (21%), LEN (99%)
- Refractory to: PI 80%, LEN 87%, double refractory (70%)

EPd vs Pd: ORR 53% vs 26% **VGPR: 20% vs 9%**



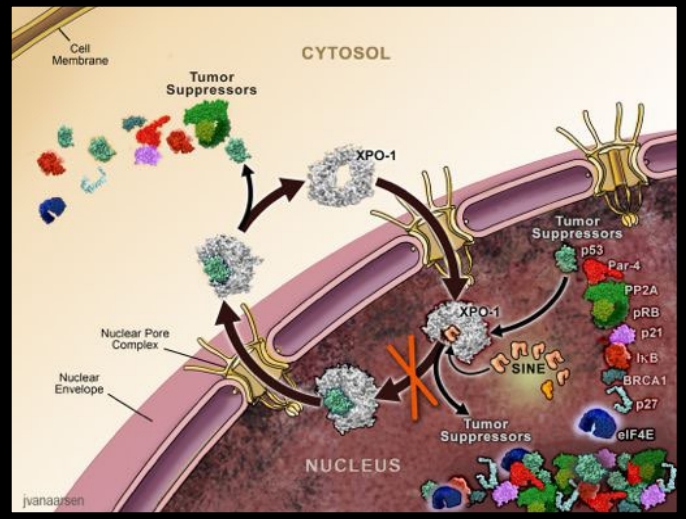
Safety Epd vs Pd : Grade 3-4 neutropenia: 13% vs 27% // Anemia: 10% vs 20% // Infections any grade: 65% vs 65%.

Safety was consistent with prior reports of ELO and POM

Dimopoulos MA et al. NEJM 2018, 379:1811-22

XPO1-Inhibitor Selinexor in RRMM. Summary of Phase I data

First-in-class, oral Selective Inhibitor of Nuclear Export (SINE) that inhibits XPO1 and activates tumor suppressor proteins & reduces oncoproteins



- Cancer cells (and MM) overexpress XPO1, causing increased export of tumor suppressors and growth regulatory proteins from the nucleus
- Selinexor inhibit XPO1 mediated nuclear-cytoplasmic transport by transiently binding to XPO1 cargo binding site.
- Accumulation of Tumor suppressors in the nucleus amplifies the natural apoptotic function in cancer cells with damaged DNA.

Tai et al. Leukemia 2014

PHASE I OF SELINEXOR PLUS/MINUS DEX IN RRMM→

- **Single agent** (oral:3-45 mg twice/ w)…. **17% MR,** *Chen et al. ASH 2014*

  Main AEs: Anorexia, nausea/vomiting, fatigue, thrombocytopenia.

- **+Dex** (n=122) (STORM)…….**26% ORR** (Pent a-Refrct) PFS: 3,7m *Vogl et al. JCO 2018; Chari ASH 2018 (Abs 598)*

  AEs: nausea 73%, vomiting 49%, anorexia 49%, thrombocytopenia 73% /59% gr 3-4)

- **+ Bortz/dex** (n=42)……. 63% (**43%** in Btz Rfct) (PFS: 9 (6,1)m)*Bahlis NJ, Blood 2018, (PH III BOSTON trial ongoing)*

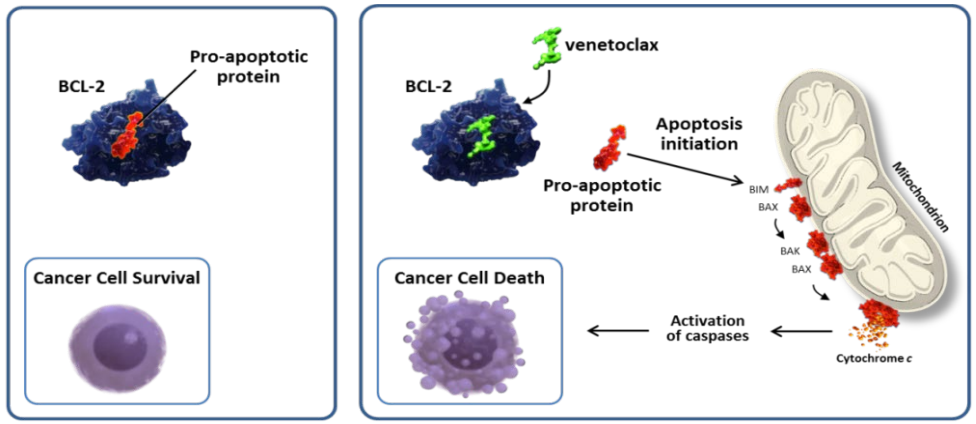
  AEs: anorexia 33%, nausea 67%, Thrombocytopenia 17%

- **+ Pom/dex** (n=24)……. 65% % in Pom Naive/Len R (**29%** in Pom/Len Rft). *Chen et al, ASH 2017)*

- **+ Dara/dex** (n=25)……. **74%** % in double Rft. *Gasparetto et al, ASH 2018, Abs 599)*

Venetoclax (bcl-2 inhibitor) in RRMM. Summary of Ph1 data

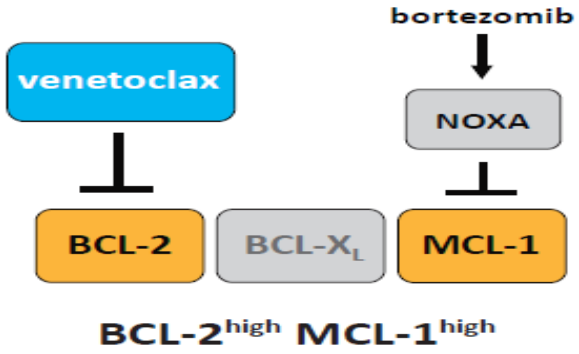
- Venetoclax is a selective, orally available small molecule BCL-2 inhibitor[1], induces cell death in multiple myeloma (MM) cell lines and primary samples, particularly those positive for the translocation t(11;14), which correlates with higher ratios of *BCL2* to *MCL1* and *BCL2* to *BCL2L1* (BCL-X_L) mRNA[1],



BCL-2 overexpression allows cancer cells to evade apoptosis by sequestering pro-apoptotic proteins.[1-3]

Venetoclax binds selectively to BCL-2, freeing pro-apoptotic proteins that initiate programmed cell death (apoptosis).[4-6]

1. Leverson JD, et al. *Sci Transl Med* 2015; 7:279ra40. 2. Czabotar, et al. *Nature Reviews* 2014;15:49-63. 3. Plati J, Bucur O, Khosravi-Far R. *Integr Biol (Camb)* 2011;3:279–296. 4. Certo M, et al. *Cancer Cell*. 2006;9(5):351-65. 5. Souers AJ, et al. *Nat Med*. 2013;19(2):202-8. 6. Del Gaizo Moore V et al. *J Clin Invest*. 2007;117(1):112-21.



1. Roberts AW et al. *NEJM* 2015
2. Punnoose E et al. *Mol Cancer Ther* 2016

- **Monotherapy** (n=66) (61% double Ref) ..... **ORR 21% (40% in t(11;14) DOR: 9.7m**

  G 3-4 AEs: thrombocytopenia (26%) & neutropenia (21%)

- **+Btz/Dex** (n=66) ..... **ORR 67% (90% in BTz sensitive & 94% in BCL2 high)**

  G3-4 AEs: Thrombocytopenia (29%), anemia (15%), neutropenia (14%),

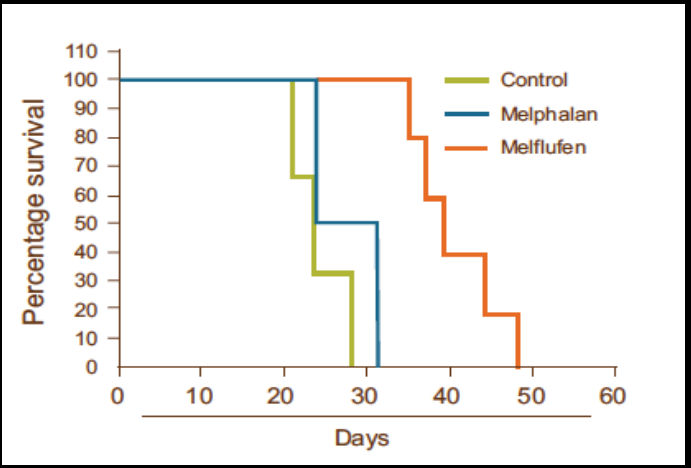
- **+Cfz/Dex** (n=42) (33% double Ref).....**ORR 78% (PFS: 5,7m. The VGPR in t(11,14): 88%)**

Kumar, et al. Blood 2017; 130: 2401-09, Moreau P, et al. Blood 2017 130:2392-4 ; Costa et al ASH 2018, Abst 303

Melflufen

- Melflufen is a highly lipophilic alkylator, belonging to the novel class of Peptidase Enhanced Compounds, consisting of melphalan + 4-fluoro-L-phenylalanine.
- Intracellular amino-peptidases that are overexpressed in most malignant cells, will rapidly cleave melflufen releasing the hydrophilic, active metabolite melphalan.
- In vitro, equimolar treatment of tumor cells with melphalan and melflufen, results in a 20-50 fold higher intracellular concentration.

Melflufen 40 mg iv every 28 days + Dex 40 mg weekly



Chauhan Clin Cancer Res 2013 & Wickström Invest New Drugs 2008

Phase II O-12-M1 trial

RRMM pts ≥ 2 lines and refr. to last line.

n = 45; 4 (2-14) lines; 64% double refr.; 53% Alkylator refr.

ORR 31% ..... 5 VGPR & 9 PR 36% in Alkylator refr.
PFS: 5,7m ; OS: 20M

G3/4 AEs: Thromboc. (58%), Neutrop(51%), Anemia: 42%

Blood 2017, 130: 3150

Phase II Horizon trial

RRMM pts ≥ 2 lines and 89% double Ref
n = 62 6 (3-11) lines; Alkylator refr. 58%;Pom & Dara Refr: 56%

ORR 32% ..... PFS: 5,7M; OS: 20,7M

G3/4 rel. TEAEs: Thromboc. (45%), Neutropenia (39%), Anemia: 21%

Richardson P. ASH 2018 (Abst 600)

Four Major Targets for Cancer Immunotherapy

Direct targeting of surface tumor antigens:

Monoclonal antibodies

Overcoming inhibitory immune suppression:

Immunomodulators: IMiDs, checkpoint inhibitors

Boosting immune effectors:

Adoptive cell therapy

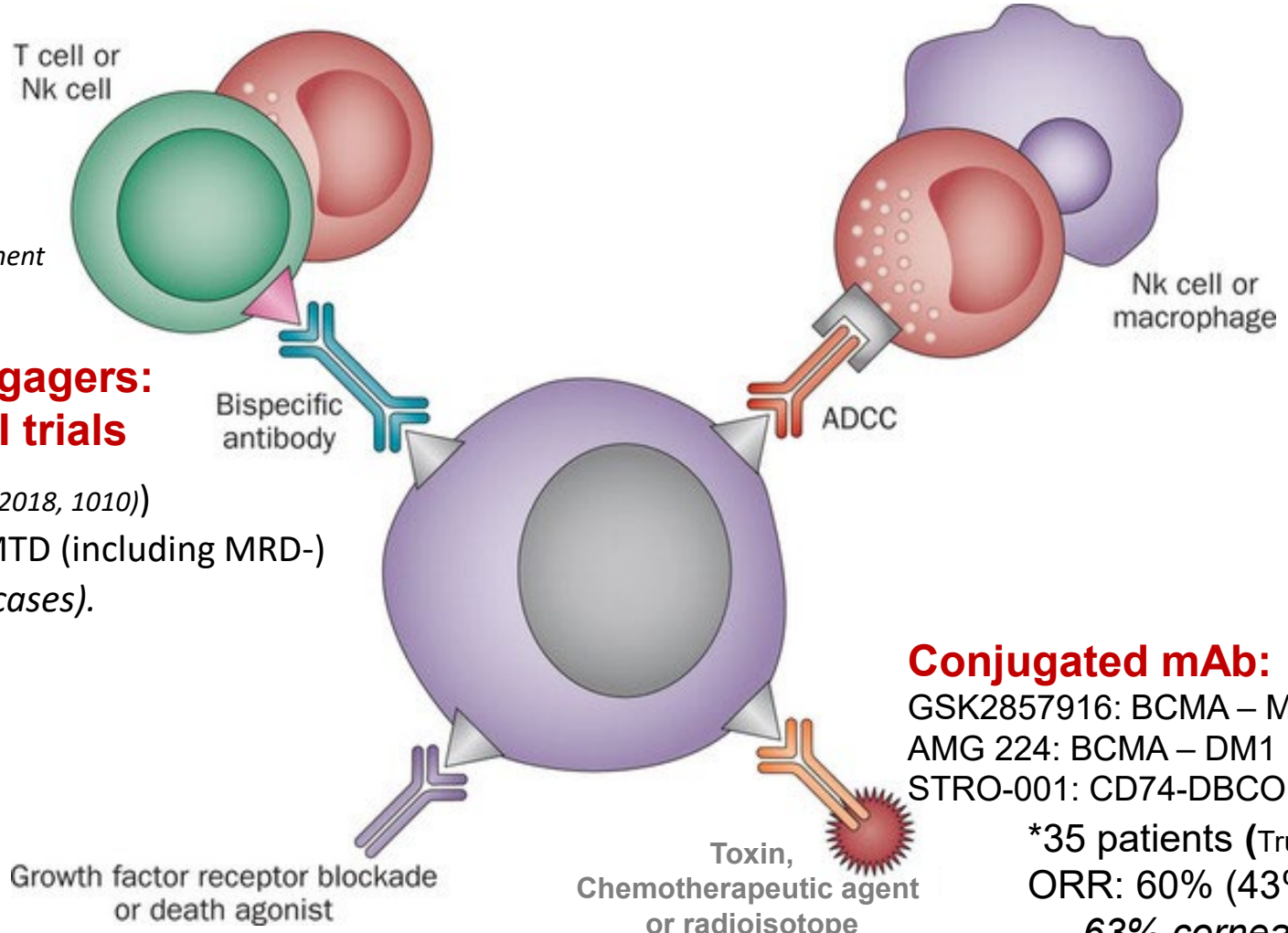
Activating tumor specific immunity:

Vaccines

IMiD, immunomodulatory drugs.

Monoclonal Antibodies: Futures Perspectives

To overcome the limitations of an immunosuppressive tumor microenvironment by linking CTLs with the tumor cell.



Bispecific T-cell engagers: BCMA–CD3 Phase I trials

- AMG 420: 35 pts: *(Topp et al ASH 2018, 1010)*)
  28% ORR (6CR). 83% ORR at MTD (including MRD-)
  SAE: 49% (infections); CRS (3 cases).

Conjugated mAb:

GSK2857916: BCMA – MMAF*
AMG 224: BCMA – DM1
STRO-001: CD74-DBCO

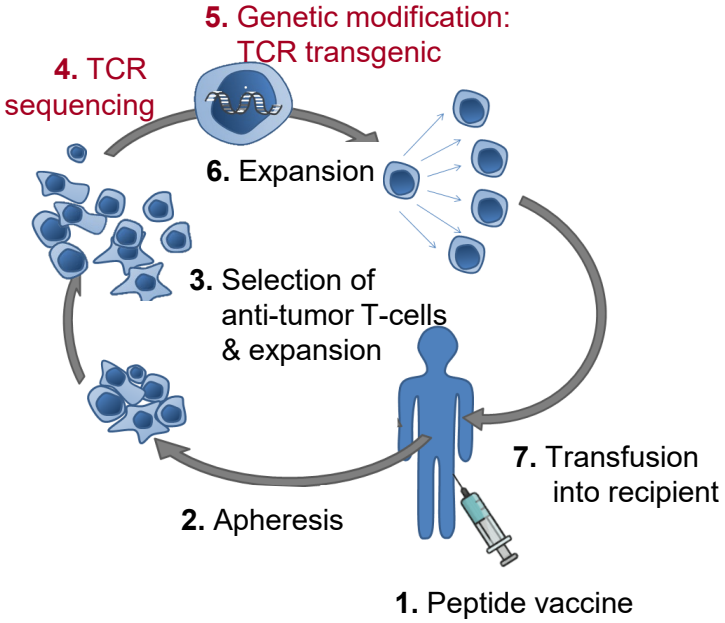
*35 patients (Trudel S, *et al. Blood* 2017;130:741)
ORR: 60% (43% previous dara) PFS: 7.9m
63% corneal events most G1-2

*

Mackall CL, *et al. Nat Rev Clin Oncol* 2014;11:693-703.

MMAF, monomethyl auristatin F; DM1, maytansinoid N(2')- deacetyl-N(2')-(3-mercapto-1-oxopropyl)-maytansine.

Adoptive Cell Therapy: Genetically Modified T-Cell Therapy



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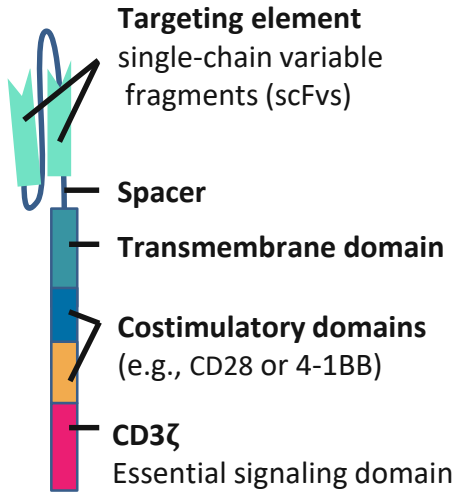
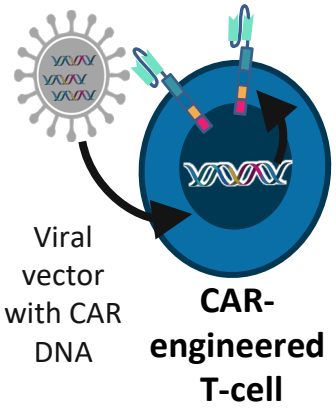




This construct is then transfected or transduced into patients' autologous T-cells

TCR engineered T-cells	CAR T-cells
HLA - restricted	Antigen recognition is independent of MHC molecule
Potential recognition of intracellular antigens	Only extracellular proteins can be recognized (like mAb)
TCR-mediated activation	Possibility to insert other genes

HLA, human leukocyte antigen; mAb, monoclonal antibody; MHC, major histocompatibility complex; TCR, T-cell receptor., Chimeric antigen receptor (CAR) T-cells
Lim WA & June CH. *Cell* 2017;168:724–40.

BCMA CAR T-Cells in MM

Trial site	ScFv	Co-s domain	Gene transfer	Conditioning therapy	T-cell dose CAR+ T-cells/kg
NCI	11D5-3	CD28	Y- retroviral	Cy 300 mg/m^2 x3 + Flu 30 mg/m^2 x3	0.3–9.0 x 10^6
Bluebird Celgene	NR, murine	4-1BB	Lentiviral	Cy 300 mg/m^2 x3 + Flu 30 mg/m^2 x3	50, 150, 450 and 800 x 10^6
University of Pennsylvania	NR, human	4-1BB	Lentiviral	None or Cy 1.5 g/m^2	10–50 x 10^6 or 100–500 x 10^6
Nanjing Legend Biotech	NR	NR	Lentiviral	Cy 300 mg/m^2 x3	1.5–7.0 × 10^6
Memorial Sloan Kettering Cancer Center	NR, human	4-1BB	Y- retroviral	Cy 3000 mg/m^2 or Cy 300 mg/m^2 x 3 + Flu 30 mg/m^2 x3	1x10^6 150, 450 and 800 x 10^6

This slide is provided for ease of viewing information from multiple trials. Direct comparison between trials is not intended and should not be inferred.
ScFv, single-chain fragment variable.

BCMA CAR T-cell Therapies for MM

	Anti-BCMA CAR[1] NCT02215967	Bb2121[2] NCT02658929	CART-BCMA[3] NCT02546167	LCAR-B38M[4] NCT03090659
Group/company	***NIH***	***Bluebird/Celgene***	***University of Pennsylvania/ Novartis***	***Nanjing Legend Biotech***
Patients	**16** patients at 9x10^6/kg dose level	22 (>150 x 10^6 cells)	**21 (3 cohorts)**: 9 (10-500 x 10^6, No Cyt) 5 (10–50 x 10^6 , Cyt) 7 (5 (100–500 x 10^6 , Cyt)	**57**
BCMA expression required?	Yes	Yes; ≥ 50% BCMA expression	No	Yes
Median prior lines of therapy	7	7	7 (3–11)	3
Reported efficacy	ORR 14/16 (**81%**) 11/14 (**79%**) MRD- EFS: **7.2 months**	**86.4%** ≥VGPR (**50%** sCR/CR) **PFS: 11.8 months**	#1**: 67%** (1 sCR, 1VGPR) #2: (**40%**) 1 PR, 1 MR both PD #3: (**83%**) 1 CR, 3 PR, 1 MR	ORR: **88%** **CR: 74%** **MRD-: 93% of CR** **PFS:15m**
Safety data	CRS all grades:100%, 37%G3-4	CRS all grades: 63% 2 events of CRS grade ≥3 resolved within 24 hours	CRS: 17 pts (grade 3: 32%) Neurotoxicity: 3 (2 grade 4) 1 death – PD candidaemia	Transient CRS (5,7% G3) No neurotoxicity

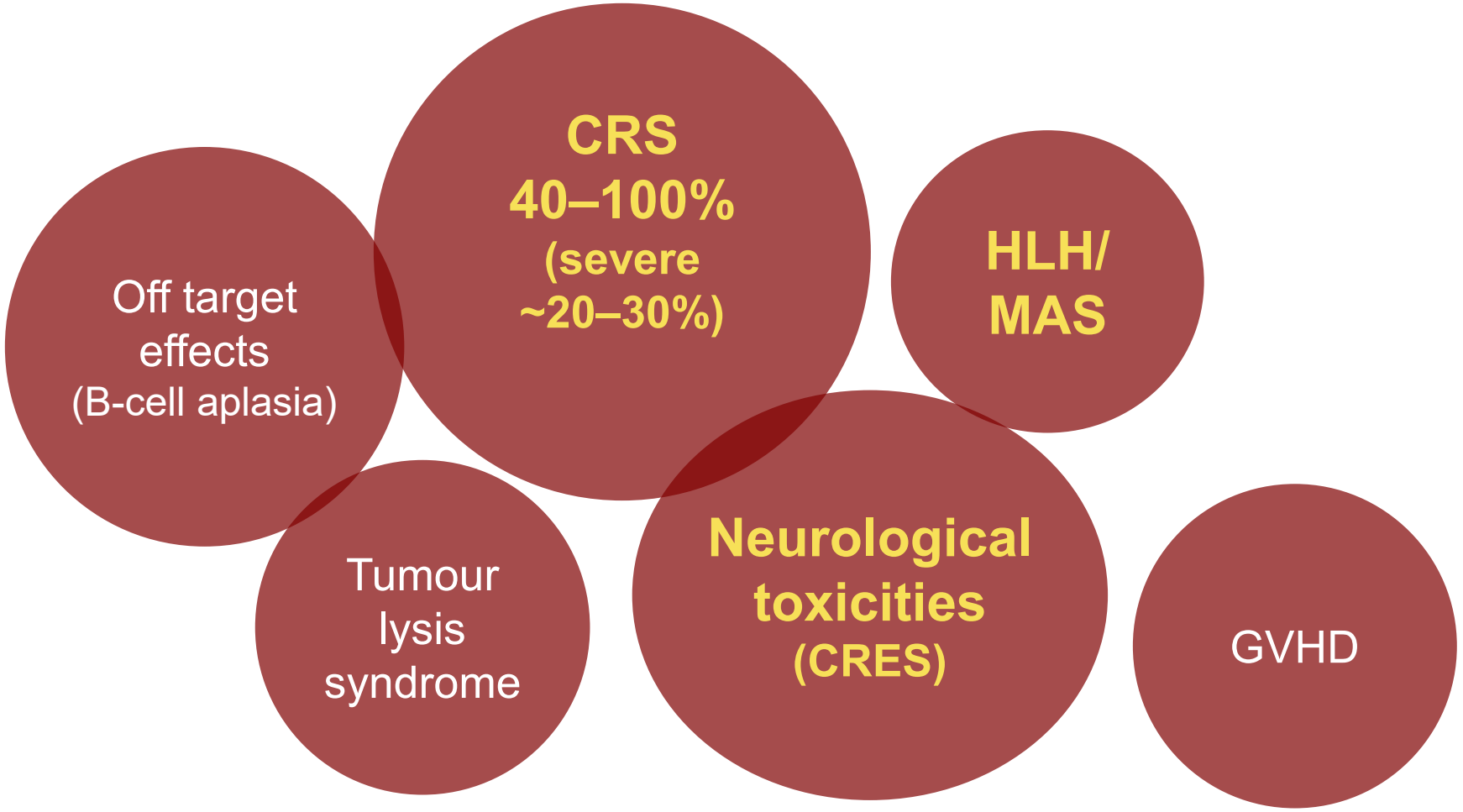
This slide is provided for ease of vie
BCMA, B-cell maturation antigen; C
partial response

1. Ali A, *et al.* Presented at ASH 2015. Abstract LBA 1; 2. Raje NS, *et al. JCO*. 2018;36:(suppl; abstr 8007); 3. Cohen AD, *et al. Blood* 2017;130:505.; 4. Zhang W, *et al.* Presented at EHA 2017. Abstract S103.

Abstracts ASH 2018: 488, 955-7, 959, 960, 1009, 1011-14

Safety Concerns Regarding CAR T-Cell Therapy

CRS is the most common toxicity triggered by the activation of T-cells and bystander immune cells → release of cytokines and chemokines: IFN-γ, soluble IL-2R, IL-6, etc



CRS, cytokine release syndrome, (Tocilizumab & Corticosteroids) CRES, CAR T-cell-related encephalopathy syndrome, GVHD, graft-versus-host disease, HLH, haemophagocytic lymphohistiocytosis, MAS, macrophage activation syndrome.

Improvements of CAR T-Cell Therapies

Limitation	Potential Improvements
Immunological rejection & safety	• **Humanised** CARs to reduce immunogenicity • **Allogeneic CAR T:** Gene editing (CRISPR/Cas9) of normal donor T-cells to remove naive TCR (to avoid GVHD) and transfection with a CAR with post-conditioning vaccination to improve memory • **Safety marker gene** to extinguish the CAR-T activity.
Immune system limitations	• Rational **combination strategies** : Checkpoint inhibitors, IMiDs, BTK inhibitors
Efficacy & antigen escape	• **Bi-specific CAR** (e.g. CD19, CD123, BCMA, SLAMF7) • Use of **specific T-cell subpopulations** (from naive to **central memory** and to terminal effector T-cells) • **APRIL** as the natural BCMA/TACI ligand instead of the Ab (anti-BCMA) • **Antibody-Coupled T-Cell Receptor (ACTR):** engages antibody to direct T-cell attack against many different Ags • **Armored CAR** (*2nd gene that generate a cytokine: i.e. IL12)*

AICD, activation-induced cell death, ScFv, single-chain fragment variable, TRAC, T-cell receptor α constant.
Brudno JN & Kochenderfer JN. *Nat Rev Clin Oncol* 2018;15:31–46.

Conclusions

- The discovery and development of new therapies addressing a variety of therapeutic targets is already changing the natural history of MM
- The understanding of the mechanisms of progression and immune-surveillance escape as well as the manipulation of autologous immune cells and gene editing are opening new frontiers in the treatment of advanced or difficult-to-treat MM
- The combination of different class of drugs with complementary immunological strategies and earlier in the natural history of the disease may offer the future possibility of long-term control or even disease eradication in some subsets of patients

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