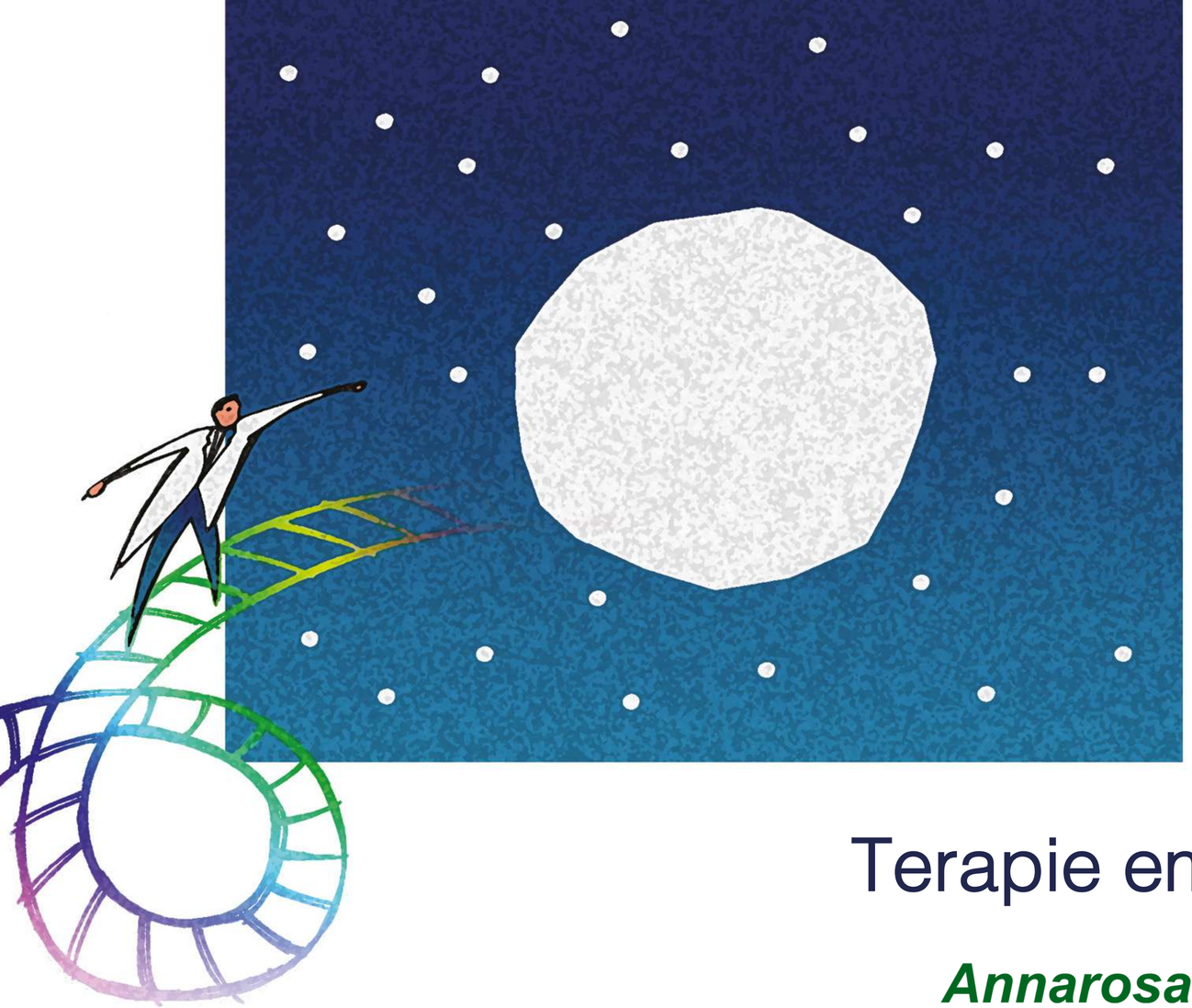


The young side of
LYMPHOMA

gli under 40 a confronto

Verona, Centro Congressi Camera di Commercio
26-27 settembre 2025

Terapie emergenti nel HL

Annarosa Cuccaro

Istituto Nazionale Tumori, IRCCS Fondazione G. Pascale,
UOC Ematologia Oncologica, Napoli

Disclosures of Name Surname

Company name	Research support	Employee	Consultant	Stockholder	Speakers bureau	Advisory board	Other
Takeda						x	

- LH SN II, Stadio IIB

Aprile 2014

Maggio-Ottobre 2014

ABVD x 6

RP

Gennaio-Aprile 2015

IGEV + ASCT

SD

Aprile 2015-Aprile 2016

BV x 12

PD

Aprile 2016-Novembre 2021

Nivo x 5 aa

Relapse

Cosa fareste?

a) Trapianto allogenico

b) Trial Clinico

c) Chemioterapia *platino-based*

d) *Rechallenge* BV/CPI

ARTICLE OPEN

Check for updates

Outcomes in patients with classic Hodgkin lymphoma refractory or intolerant to brentuximab vedotin and anti-PD-1 therapy: a real world analysis from 15 U.S. academic centers

Timothy J. Voorhees[1✉], Eric M. McLaughlin[2], Pallawi Torka[3], Jorge Florindez[4], Na Hyun Kim[5], Tamara K. Moyo[6], Heather Reves[7], Nuttavut Sumransub[8], Saarang Deshpande[9], Ashley Rose[10], Cassandra Duarte[11], Muhammad Salman Faisal[12], Showkat Hamid[12], Suki Subbiah[13], Sabarish Ayyappan[14], Lauren Shea[15], Matt Cortese[12], Krish Patel[16], Ajay Major[11], Hayder Saeed[10], Jakub Svoboda[9], Sanjal Desai[8], Praveen Ramakrishnan Geethakumari[7], Mehdi Hamadani[5], Natalie Grover[4] and Narendranath Epperla[1,17]

Table 3. Double Refractory Characteristics.

	All patients $n = 173$
Age At DR/INT, (median, range)	40 (19–90)
DR/INT	
Double Refractory	127 (73.4)
Intolerant	46 (26.6)
Years From Diagnosis, (median, range)	3.4 (0.5–23.1)
Prior LOT (median, range)	5 (1–13)
Prior ASCT at DR/INT	
Yes	86 (49.7)
No	87 (50.3)
Treatment After DR/INT	
Yes	153 (88.4)
No	20 (11.6)
BV Therapy After DR/INT	
Yes	41 (23.7)
No	132 (76.3)
Anti-PD-1 Therapy After DR/INT	
Yes	63 (36.4)
No	110 (63.6)

Table 1. Patient Characteristics.

	All Patients $n = 173$	Refractory (DR) $n = 127$	Intolerant (INT) $n = 46$
Age at Diagnosis, median (range)	34 (16–89)	33 (16-89)	40 (18-87)
Sex, *n* (%)			
Male	105 (60.7)	76 (59.8)	29 (63.0)
Female	68 (39.3)	51 (40.2)	17 (37.0)
Race, *n* (%)			
Caucasian	136 (78.6)	100 (78.7)	36 (78.3)
African American	25 (14.5)	18 (14.2)	7 (15.2)
Hispanic	6 (3.5)	5 (3.9)	1 (2.2)
Other	6 (3.5)	5 (3.2)	2 (4.4)
cHL Subtype			
Nodular Sclerosing	118 (68.2)	86 (67.7)	32 (69.6)
Mixed Cellularity	18 (10.4)	10 (7.9)	8 (17.4)
Lymphocyte Rich	1 (0.6)	1 (0.8)	0 (0)
Lymphocyte Deplete	6 (3.5)	6 (4.7)	0 (0)
Unknown	30 (17.3)	24 (18.9)	6 (13.0)
Stage At Diagnosis			
Limited (I-II)	36 (20.8)	29 (22.8)	7 (15.2)
Advanced (III-IV)	132 (76.3)	97 (76.4)	35 (76.1)
Unknown	5 (2.9)	1 (0.8)	4 (8.7)
Front-line Therapy			
ABVD/AVD	134 (77.5)	96 (75.6)	38 (82.6)
BV-AVD	15 (8.7)	12 (9.5)	3 (6.5)
BV + Nivo	5 (2.9)	5 (3.9)	0 (0)
BV +/- Chemo	10 (5.8)	7 (5.5)	3 (6.5)
Other	9 (5.2)	7 (5.5)	2 (4.4)
Front-line CR			
Yes	63 (36.4)	44 (34.7)	19 (41.3)
No	105 (60.7)	81 (63.8)	24 (52.2)
Unknown	5 (2.9)	2 (1.6)	3 (6.5)
Front-line Refractory			
Yes	100 (57.8)	77 (60.6)	23 (50.0)
No	71 (41.0)	49 (38.6)	22 (47.8)
Unknown	2 (1.2)	1 (0.8)	1 (2.2)

Blood Cancer Journal, 2025

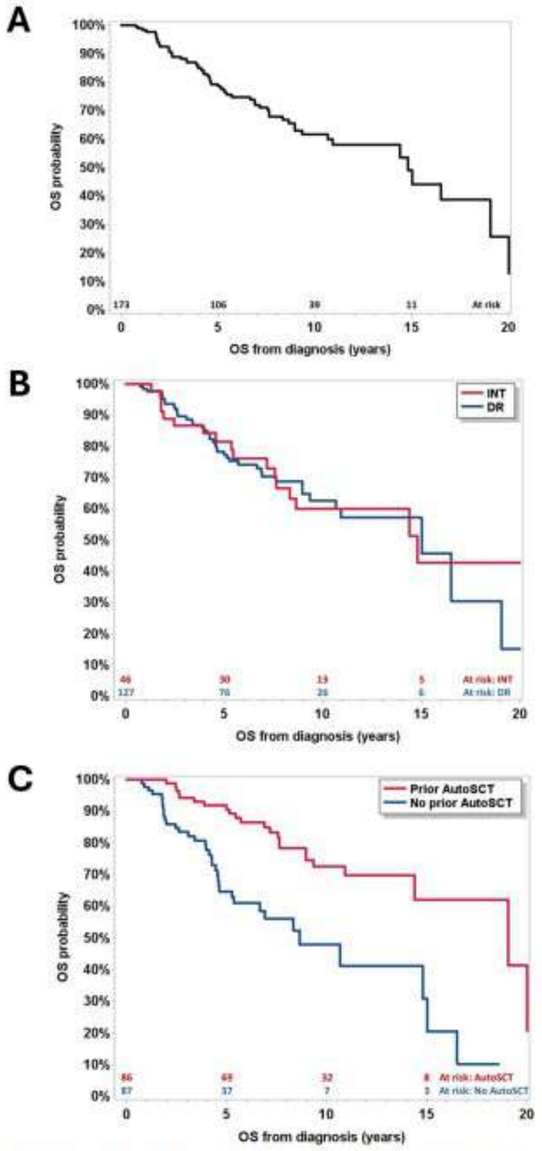


Fig. 1 Overall survival from diagnosis (OS-1). A the full cohort, (**B**) cohort stratified by INT (red) or DR (blue), (**C**) cohort stratified by prior ASCT (red) or no prior ASCT (blue) at the time of DR/INT.

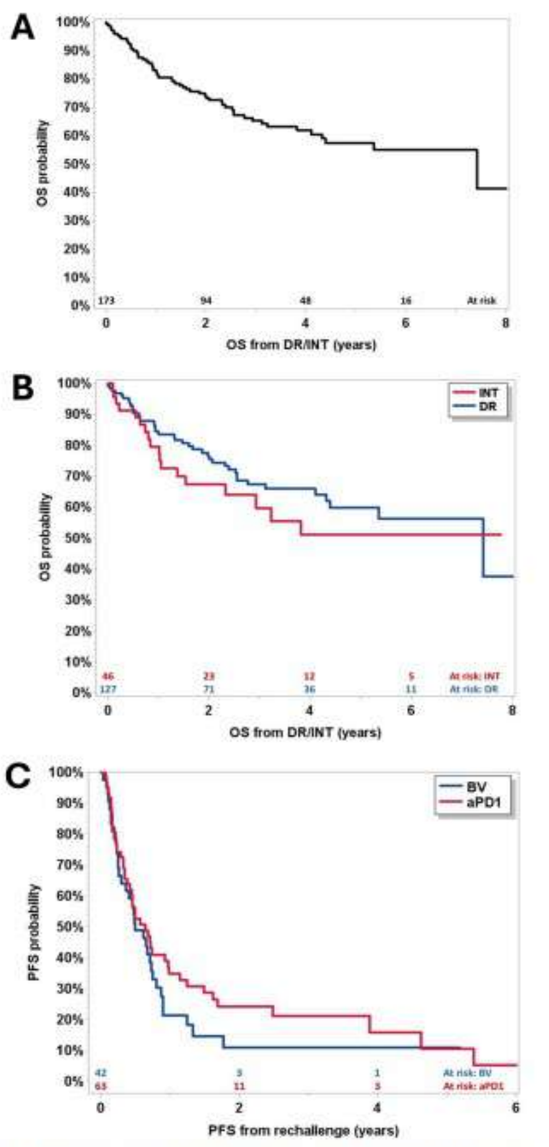


Fig. 2 Overall survival from the time of DR/INT (OS-2). A the full cohort, (**B**) cohort stratified by INT (red) or DR (blue). **C** Progression free survival (PFS) for patients rechallenged with anti-PD-1 based therapy (red) or BV based therapy (blue).

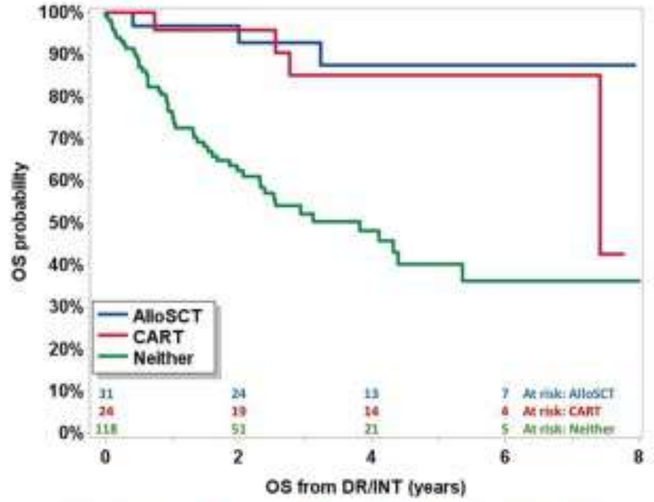
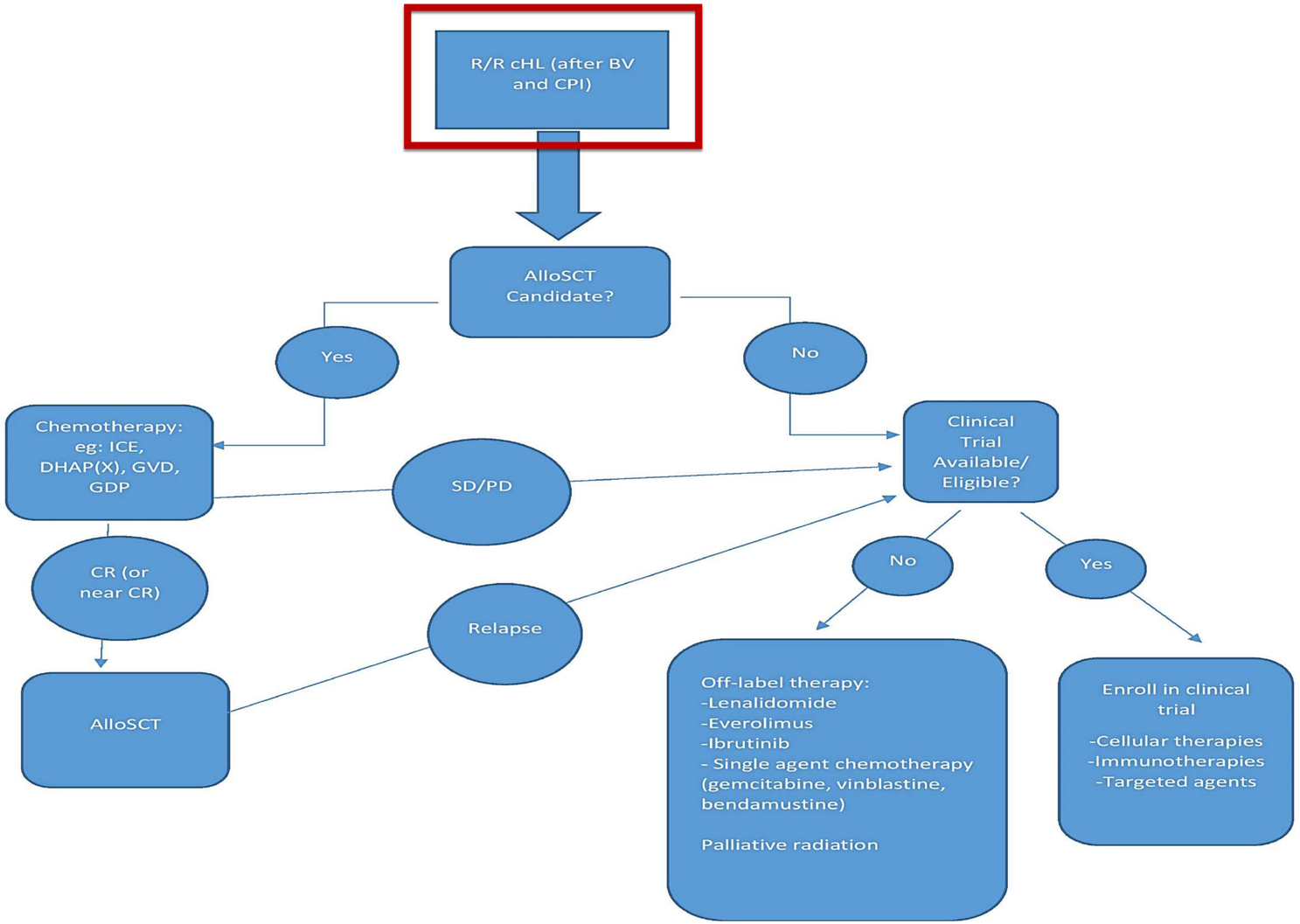
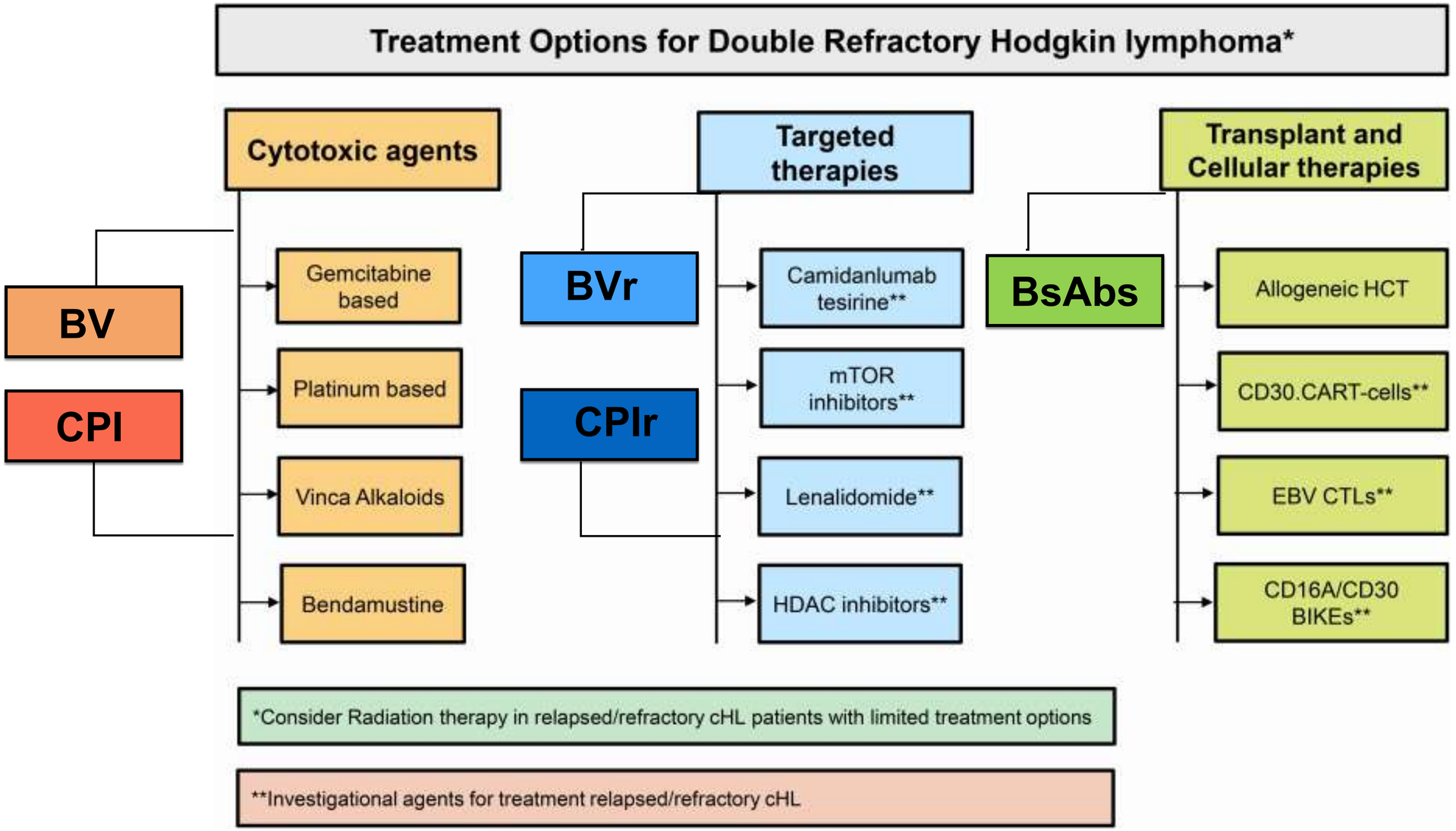


Fig. 3 Overall survival from the time of DR/INT (OS-2) stratified by subsequent AlloSCT (blue), CD30.CAR-T cell therapy (red), or neither therapy (green).

T.J. Voorhees, Blood Cancer Journal ,2025

Management of relapsed and refractory HL post-BV & post-CPI




Treatment Options for Double Refractory Hodgkin lymphoma*
Cytotoxic agents
Targeted therapies
Transplant and Cellular therapies
BV
CPI
BVr
CPIr
BsAbs
Gemcitabine based
Platinum based
Vinca Alkaloids
Bendamustine
Camidanlumab tesirine**
mTOR inhibitors**
Lenalidomide**
HDAC inhibitors**
Allogeneic HCT
CD30.CART-cells**
EBV CTLs**
CD16A/CD30 BIKEs**
*Consider Radiation therapy in relapsed/refractory cHL patients with limited treatment options
**Investigational agents for treatment relapsed/refractory cHL

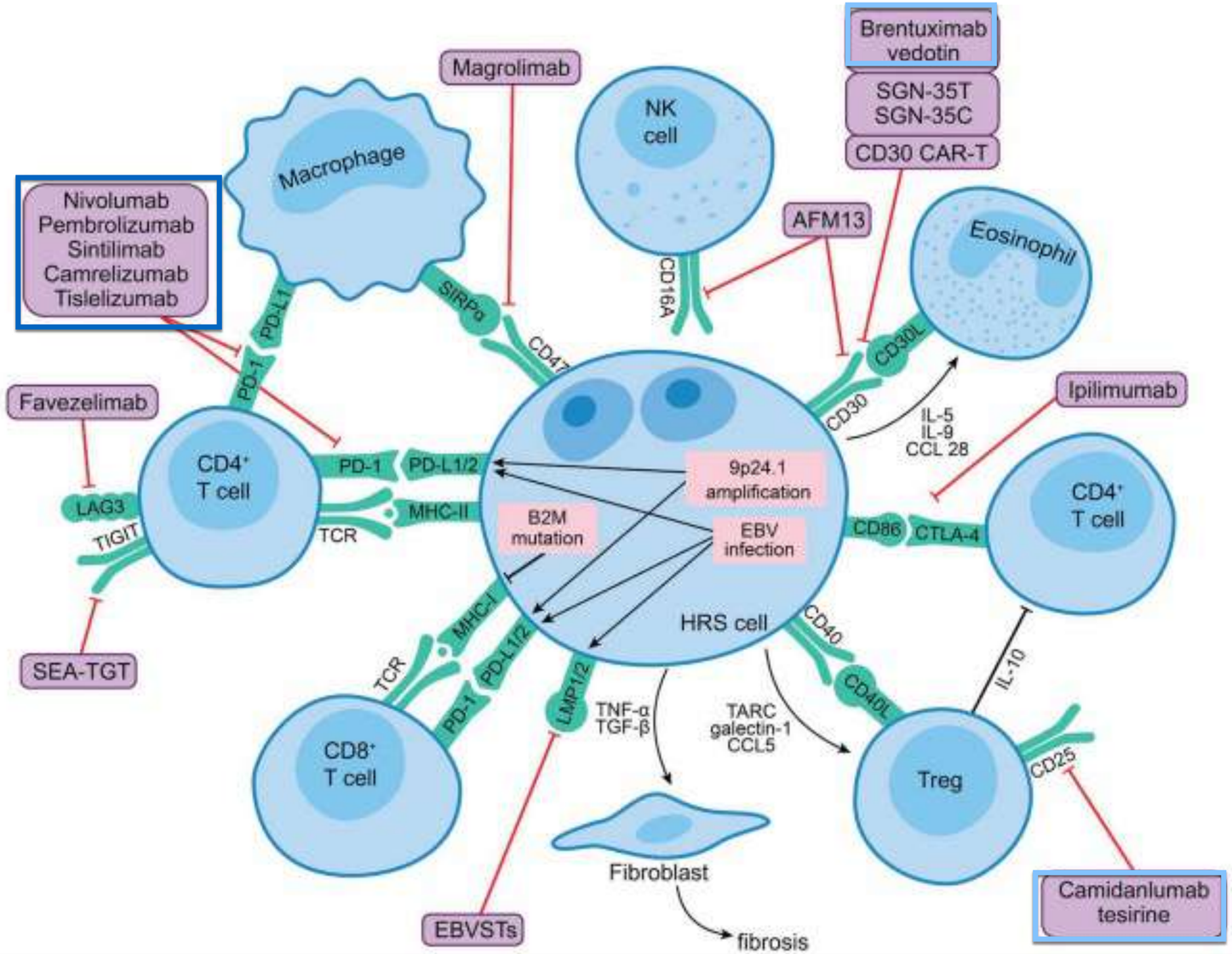
Cytotoxic agent HL

	N	ORR (%)	CR (%)	PFS	OS	Citation
GDP	23	69	17	NR	NR	Baetz et al.[11]
GVD*	41	62	20	4-y EFS: 52%	4-y OS: 70%	Bartlett et al.[12]
ICE	65	88	26	58% at 43 mo	NR	Moskowitz et al.[13]
DHAP	102	89	21	NR	NR	Josting et al.[14]
Gemcitabine	23	39	9	6.7 mo	10.7 mo	Santoro et al.[19]
Vinblastine	17	59	12	8.3 mo	38.8 mo	Little et al.[20]
Bendamustine	35	53	33	5.2 mo	NR	Moskowitz et al.[21]

Cytotoxic agent HL + BV/CPI

Regimen	Phase	No.	Primary Refractory, No.	Relapsed, No.	CMR, %	PFS (all patients)	PFS (ASCT cohort)	Reference
BV-based salvage regimens								
BV→augmented ICE	II	45	25	20	76[1]	82% at 3 years	82% at 3 years	Moskowitz et al[19]
BV→salvage therapy	II	57	35	22	74[2]	71% at 2 years	71% at 2 years	Herrera et al[17]
BV + ICE	II	45	29	16	74	80% at 2 years	Not reached	Lynch et al[48]
BV + ESHAP	I/II	66	40	26	70	71% at 30 months	Not reached	Garcia-Sanz et al[49]
BV + DHAP	II	61	23	38	79	76% at 2 years	Not reached	Hagenbeek et al[50]
BV + gemcitabine	I/II	45[4]	29	16	67	Not reached	Not reached	Cole et al[51]
BV + bendamustine	I/II	55	28	27	74	62.6% at 2 years	70% at 2 years	LaCasce et al[52]
Anti–PD-1–based salvage regimens								
N ± ICE[5]	II	39	18	21	91	72% at 2 years	94% at 2 years	Mei et al[18]
Pem-ICE	II	42	16	26	86.5	87.2% at 2 years	Not reached	Bryan et al[21]
Pem-GVD[7]	II	39	16	23	95	100% at 13.5 months	100% at 13.5 months	Moskowitz et al[20]
BV + nivolumab	I/II	91	38	53	67	78% at 2 years	91% at 3 years	Herrera et al, Moskowitz et al[16,53]

TME & Immunotherapeutic targets in cHL



Michael A. Spinner & Ranjana H. Advani,
EXPERT OPINION ON EMERGING DRUGS, 2024

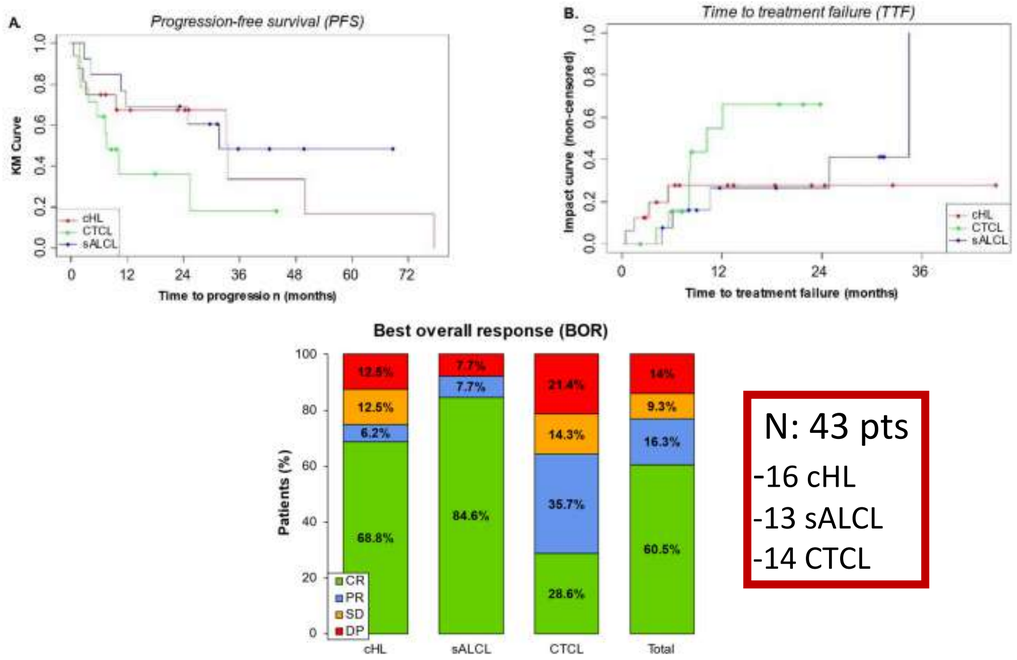
BV & CPI *rechallenge*

cancers
MDPI

Article

Retreatment with Brentuximab Vedotin in Patients with Relapsed/Refractory CD30+ Malignancies: A Retrospective Medical Chart Review Study in Spain-The BELIEVE Study

Anna Sureda [1], Ramón García-Sanz [2,*], Eva Domingo-Domenech [1], Francisco Javier Capote [3], Antonio Gutiérrez [4], Antonia Rodriguez [5], David Aguiar [6], Pilar Giraldo [7], María Stefanía Infante [8], Javier López-Jiménez [9], Carmen Martínez [10], Blanca Sánchez-González [11], Pablo L. Ortiz-Romero [12], Marta Grande [13,14] and Lourdes Baeza-Montañez [13]



N: 43 pts
-16 cHL
-13 sALCL
-14 CTCL

SYSTEMATIC REVIEW
blood neoplasia

Outcomes of immune checkpoint inhibitor rechallenge in relapsed/refractory Hodgkin lymphoma

Yoshito Nishimura,[1,*] Yiqun Han,[2,*] Nadia Toumeh,[3] Guneet Janda,[3] Yu Fujiwara,[4] Urshila Durani,[1] Grzegorz S. Nowakowski,[1] Jonas Paludo,[1] Stephen M. Ansell,[1] and Jithma P. Abeykoon[1]

Key Points

- ICI rechallenge in R/R classic HL can lead to high response rates comparable with uninterrupted ICI therapy.
- ICI rechallenge had acceptable safety profiles with grade 3 to 5 AEs occurring in 31%.

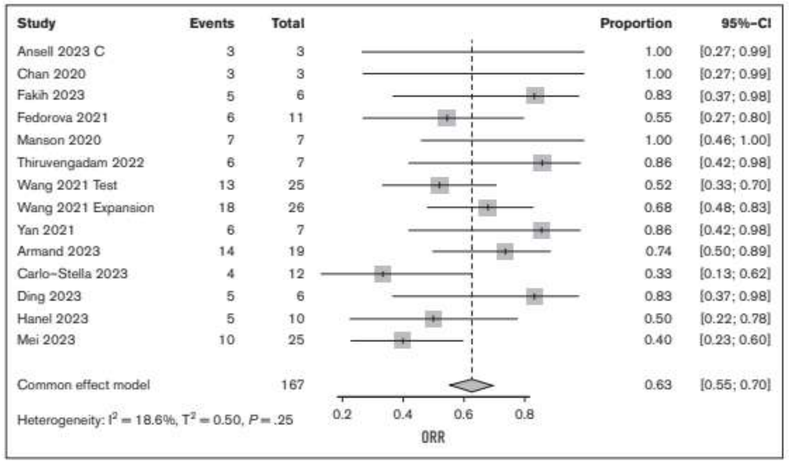


Figure 1. **The pooled ORRs after rechallenge across all studies.**

Cancers, 2025
Blood Neoplasia, 2025

Immunotherapies and small molecules in development for cHL.1

Compound	Structure	Phase	Combinations	Mechanism of action
Immune checkpoint inhibitors				
Sintilimab	Anti-PD-1 mAb	*Phase 2*	ICE	✓ Enhance CD4+ Tcell response, withrawal of prosurvival signals, possible NK cell activation
Camrelizumab	Anti-PD-1 mAb	*Phase 2*	Dacarbazine,AVD	
Tislelizumab	Anti-PD-1 mAb	*Phase 2*	Gem/Ox, AVD	
Avelumab	Anti PD-L1 mAb	*Phase 1b*	/	
Ipilimumab	Anti-CTLA-4 mAb	*Phase 2*	Nivolumab,BV	✓ Potentiare T-cell responses
Favezelimab	Anti-LAG 3 mAb	*Phase 2*	Pembrolizumab	✓ Overcome T-cell exhaustion
MK-4280°	Coformulated anti-PD-1+LAG3 mAb	*Phase 3*	/	✓ OvercomeT-cell exhaustion, Potentiare T-cell responses
Magrolimab	Anti-CD47 mAb	*Phase 2*	Pembrolizumab	✓ Enhance phagocitosis, increase antigen presentation
IMM01	SIRPα-fc fusion	*Phase 2*	Tislelizumab	✓ Enhance phagocitosis, increase antigen presentation
SEA-TST	Anti-TIGIT mAb	*Phase 1*	BV	✓ Enhance T & NK cell response
Immunomodulatory agents & small molecules				
Lenalidomide	CELMoD	*Phase 2*	Nivolumab,temserolimus	✓ Increase IL-2 production,enhance T & NK cell responses
Ruxolitinib	JAK1/2 inhibitor	*Phase 2*	Nivolumab	✓ Inhibit JAK/STAT signaling synergize with PD-1 inhibitors
Ibrutinib	BTK/ITK inhibitor	*Phase 2*	Nivolumab	✓ Enhance TH1 responses
Epigenetic modifying therapies				
Vorinostat	HDAC inhibitor	*Phase 1*	Pembrolizumab	✓ Enhance antigen presentation, t-cell recruitment & function
Entinostat	HDAC inhibitor	*Phase 2*	Pembrolizumab	
Decitabine	Hypomethylating agent	*Phase 2*	Camrelizumab	✓ Increase tumor immunogenity, T cell infiltration & overcome resistance to PD-1 inhibitors
Decitabine/cedazundine	Hypomethylating agent	*Phase 1*	Nivolumab	
Azacitidine	Hypomethylating agent	*Phase 1*	Nivolumab	

Adapted by Michael A. Spinner & Ranjana H. Advani,
EXPERT OPINION ON EMERGING DRUGS, 2024

Immunotherapies and small molecules in development for cHL.2

Compound	Structure	Phase	Combinations	Mechanism of action
Antibody-drug conjugates (ADCs)				
Camidanlumab tesirine	Anti-CD25 ADC	*Phase 2*	/	✓ Deplete regulatory T-cells, target CD25+ HRS cells
SGN-35T	Anti-CD30 ADC	*Phase 1*	/	✓ Target CD30+ HRS cells with novel linker and MMRE (35T) or camptotethecin payload (35C)
SGN-35C	Anti-CD30 ADC	*Phase 1*	/	
Bispecific antibodies (BsAbs)				
AFM13	CD30/CD16A BsAb	*Phase 2*	Pembrolizumab,AB-101	✓ Recruit NK cells to target CD30+ HRS cells
GEN3017	CD30/CD3 BsAb	*Phase 1/2*	/	✓ Recruit T cells to target
AZD7789	PD-1/TIM-3 BsAb	*Phase 1/2*	/	✓ Overcome T-cell exhaustion, potentiate T.cell responses
IBI322	CD47/PD-L1 BsAb	*Phase 2*	/	✓ Enhance phagocytosis, increase antigen presentation, potentiate T-cell responses
Cellular therapies				
Autologus CD30/CAR-T	Anti-CD30 CAR-T	*Phase 2*	/	✓ Engineeered autologous or allogenic T cells targeting CD30+ HRS-cells
Allogenic CD30 CAR. EBVSTs	Anti-CD30 CAR-T	*Phase 1*	/	
Allogenic UCB-derived NK cells	Cytochine-enhanced NK cells	*Phase 2*	AFM13	✓ Allogenic NK cell-mediiated cytotoxicity enhanced ADCC

Adapted by Michael A. Spinner & Ranjana H. Advani,
EXPERT OPINION ON EMERGING DRUGS, 2024

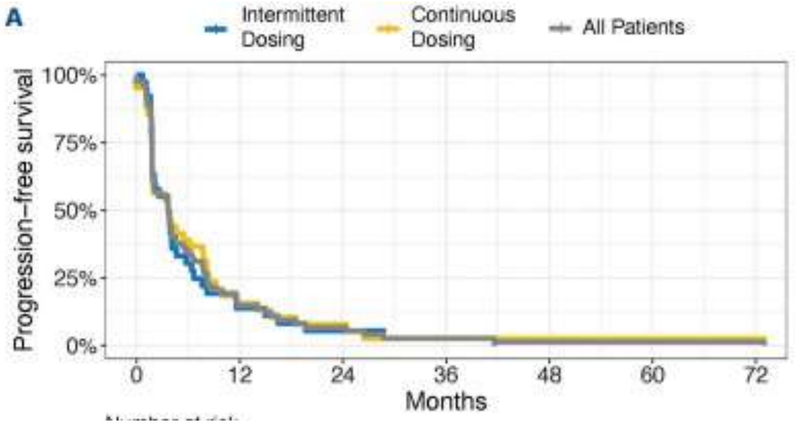
Immunomodulatory agents & small molecules.1

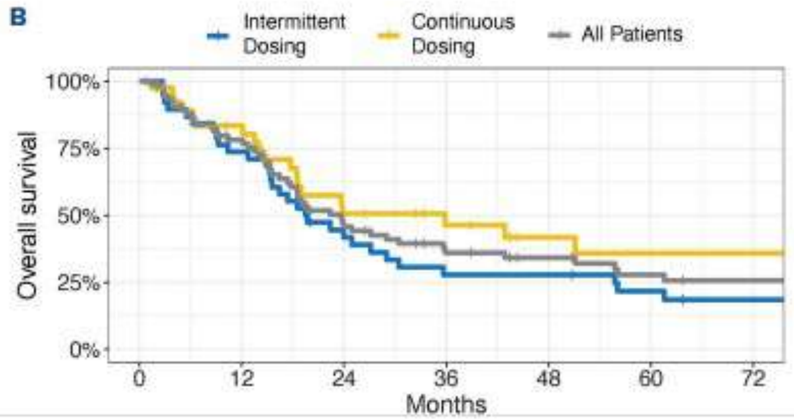
❑ Lenalidomide

LETTER TO THE EDITOR

A phase II study of interrupted and continuous dose lenalidomide in relapsed/refractory Hodgkin lymphoma

Type of response	Entire cohort			Response-evaluable patients		
	Interrupted N=38	Continuous N=42	Combined N=80	Interrupted N=36	Continuous N=36	Combined N=72
CR*, N (%)	1 (2.6)	3 (7.2)	4 (5.0)	1 (2.8)	3 (8.3)	4 (5.5)
PR*, N (%)	6 (15.7)	9 (21.4)	15 (18.8)	6 (16.6)	9 (25.0)	15 (20.8)
SD ≥6 months*, N (%)	6 (13.2)	6 (14.3)	12 (15.0)	6 (16.6)	6 (16.7)	12 (16.7)
ORR (CR+PR)**, N (%)	7 (13.2)	12 (28.6)	19 (23.8)	7 (19.4)	12 (32.4)	19 (26.4)
Disease control rate** (CR+PR+SD ≥6 months), N (%)	13 (34.2)	18 (42.9)	31 (38.5)	13 (36.1)	18 (48.6)	31 (43.1)



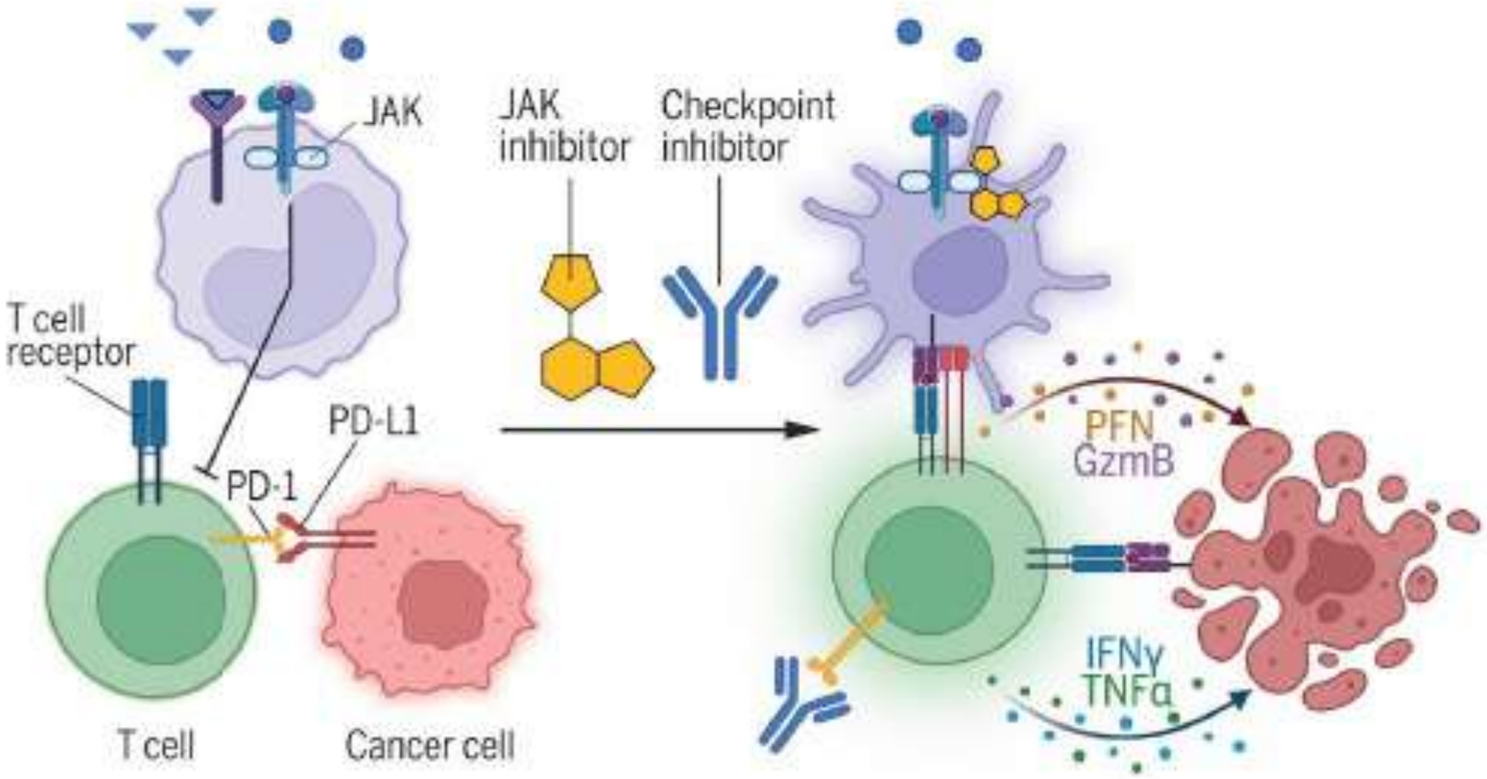


T.A. FehnigerJ et al, Haematologica 2024

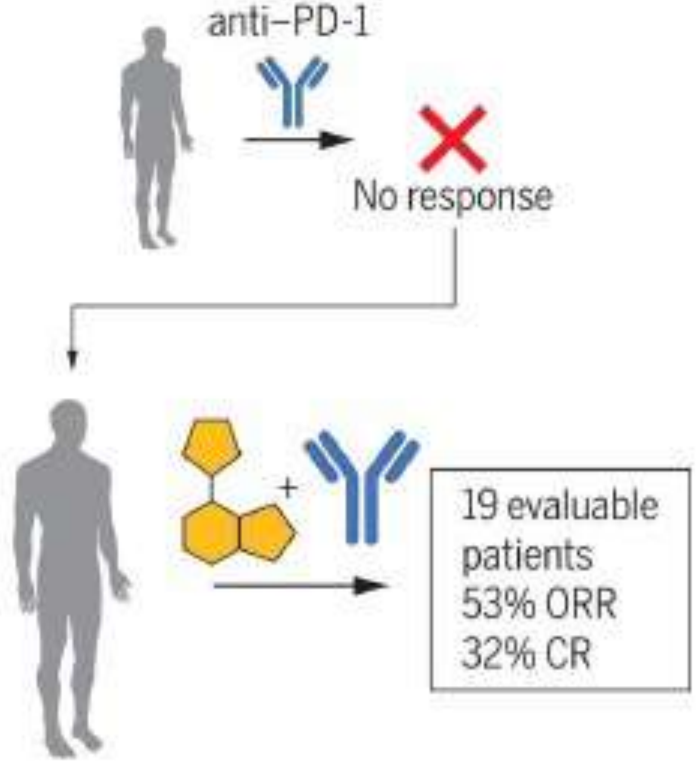
Immunomodulatory agents & small molecules.1

❑ Ruxolitinib + anti-PD-1

JAK inhibition with checkpoint inhibitors reprograms myeloid cells from a suppressive to an immunostimulatory state



Ruxolitinib with nivolumab in anti–PD-1 relapsed or refractory Classical Hodgkin lymphoma



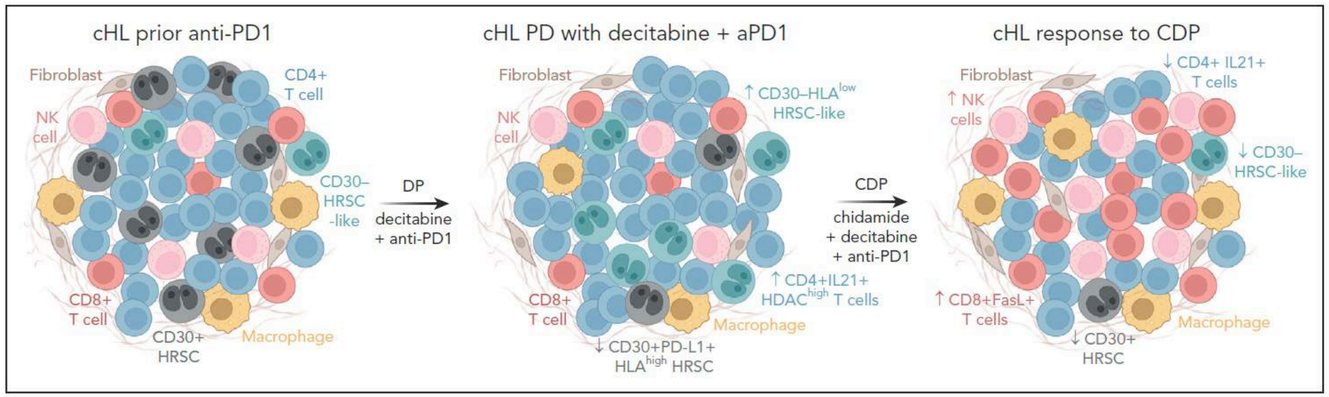
Jak J et al. Science 2024

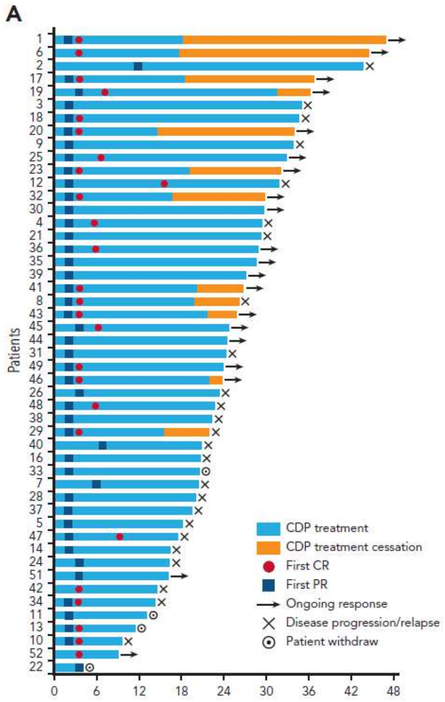
Epigenetic modifying therapies

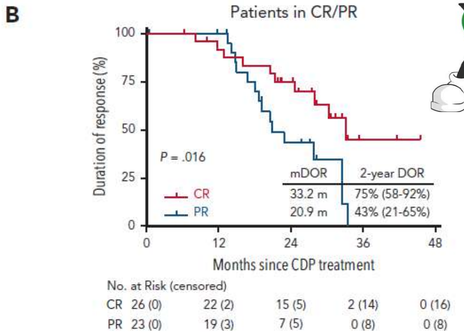
- HDACi+: chidamide+ decitabine+ anti–PD-1 camrelizumab

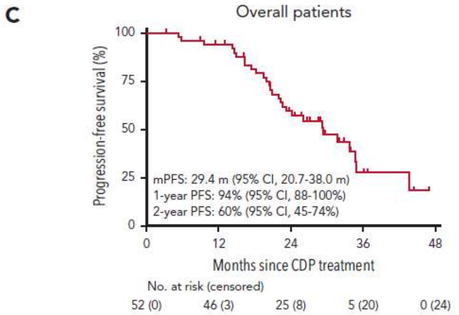
IMMUNOBIOLOGY AND IMMUNOTHERAPY

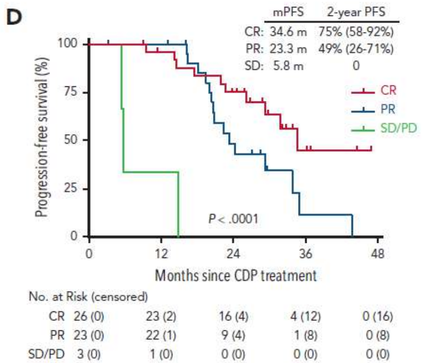
Epigenetic agents plus anti-PD-1 reprogram the tumor microenvironment and restore antitumor efficacy in Hodgkin lymphoma

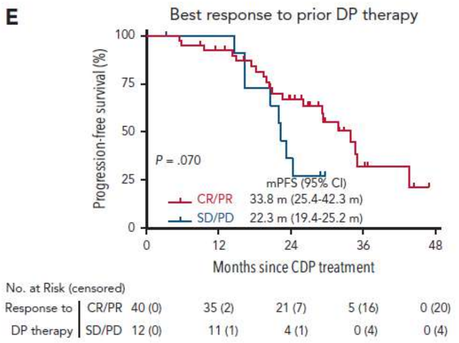












Blood (2024) 144 (18): 1936–1950

Epigenetic modifying therapies

❑ HDACi+: Vorinostat+ Bendamustine *(fusion molecule)*

Safety and Efficacy of Tinostamustine in a Subpopulation of Patients With Relapsed/Refractory Hodgkin Lymphoma From a Phase I Trial

Anna Sureda[1] | Antonio Pinto[2] | Hervé Ghesquières[3] | Franck Morschhauser[4] | Olivier Tournilhac[5] | Pim Mutsaers[6] | Josée M. Zijlstra[7] | Rosaria De Filippi[8] | Kasia Hilgier[9] | Nick Manamley[10] | Tomas Janik[9] | Pier Luigi Zinzani[11,12]

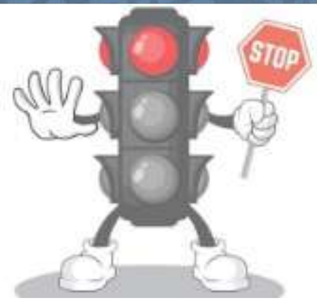
# Previous lines of therapy	Prior checkpoint inhibitor?	Checkpoint inhibitor as last line of therapy?	C1	C2	C3	C4	C5	C6	C7	C8	C9	C10	C11	C12	Dosing
3	N	N	SD												40/60
4	N	N	PD												100/60
5	Y	Y	PR												120/60
5	Y	Y	PR												120/60
4	Y	Y	PR					CR							120/60
6	Y	Y	SD					PD							80/45
5	Y	N	PD												80/45
6	Y	Y	PR											SD	60/30
3	N	N	PR												60/30
4	Y	Y	PR				SD								60/30

Complete response | Partial response
Stable disease | Progressive disease

First in class

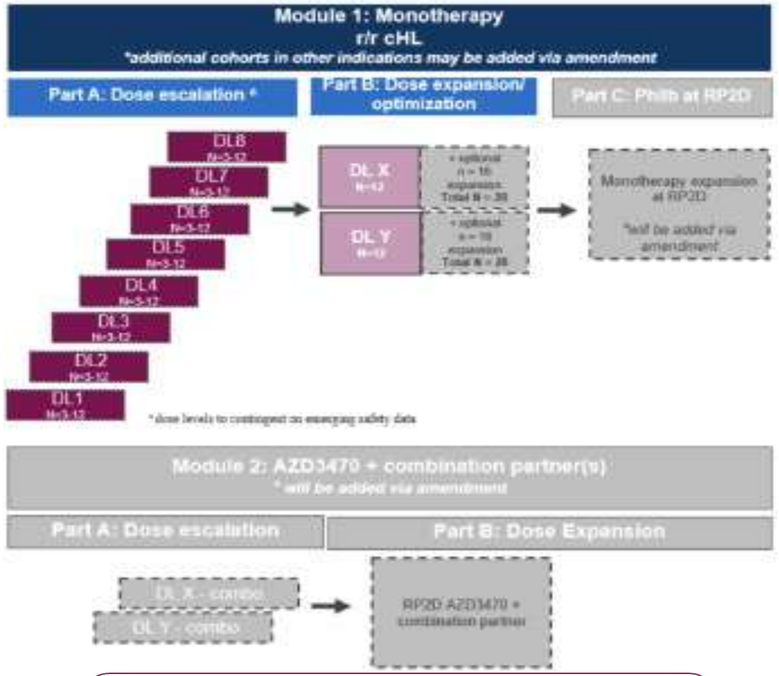
Hematological Oncology, 2025; 43:e70000
https://doi.org/10.1002/hon.70000

Bispecific antibodies



❑ PD-1/TIM-3 BsAb

Fase I/II Trial of AZD7789 *in R/R HL*



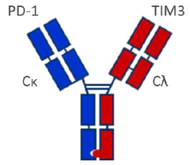
AZD7789 Structure

Drug design: DuetMab

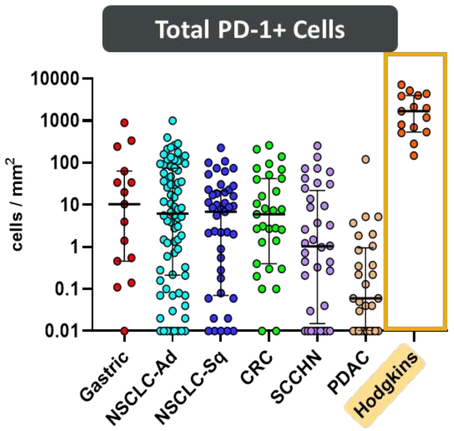
TIM-3 arm: O13 V domains

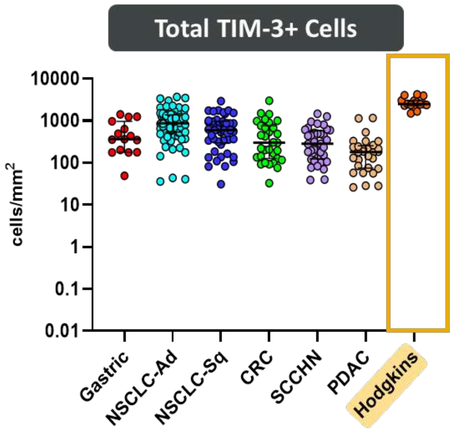
PD-1 arm: LO115 V domains

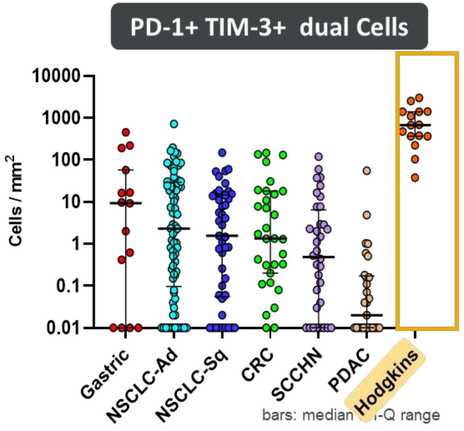
Fc isotype: IgG1-TM



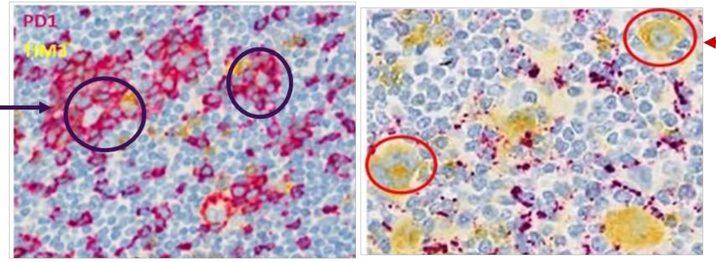
PD-1 and TIM-3 proteins highly co-expressed in HL tumor microenvironment (ME)







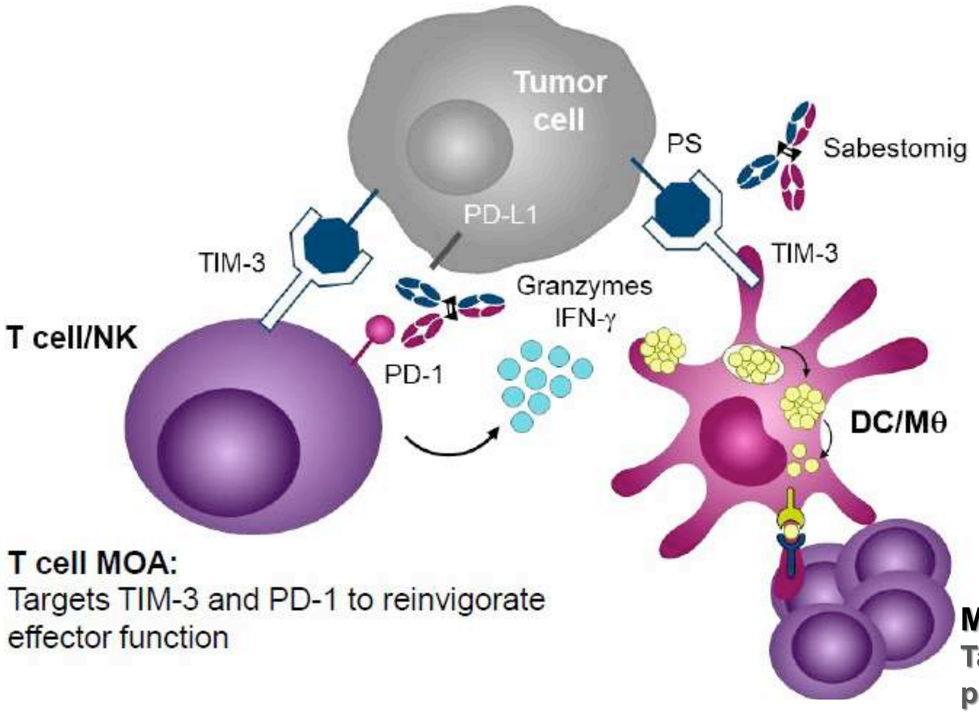
Rosette with HRS cell surrounded by PD-1+/TIM-3+ immune cells



Urosevic J et al, ASH 2023
Corrazzelli G., EHA, 2024

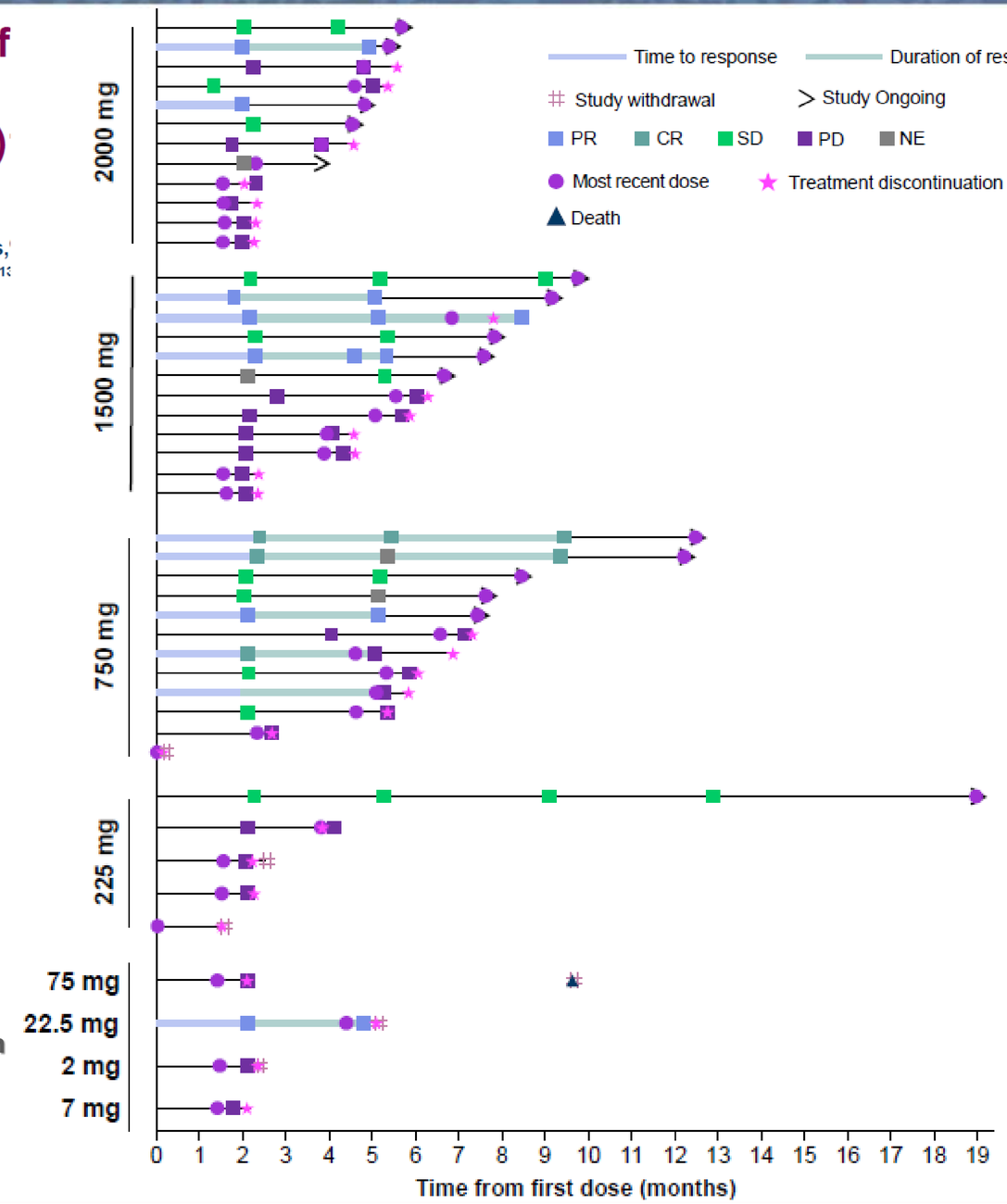
Safety and efficacy results from an open-label, Phase 1/2 trial of sabestomig (AZD7789) in patients with relapsed/refractory classical Hodgkin lymphoma previously treated with anti-PD-(L)1 therapies

Gaetano Corazzelli,[1] Franck Morschhauser,[2] Matthew Genyeh Mei,[3] Nathalie A. Johnson,[4] Craig H. Moskowitz,[5] Graham P. Collins,[6] Stephen M. Ansell,[7] Elizabeth Phillips,[8] Martin Hutchings,[9] John Kuruvilla,[10] Hun Ju Lee,[11] Alison J. Moskowitz,[12] Teresa Collins,[13] Robin Lesley,[14] George Hawkins,[13] Kaitlyn Beyfuss,[15] Richard F. Olsson,[16] Connor Hall,[17] Emma Dean,[13] Pier Luigi Zinzani[18]



T cell MOA:
Targets TIM-3 and PD-1 to reinvigorate effector function

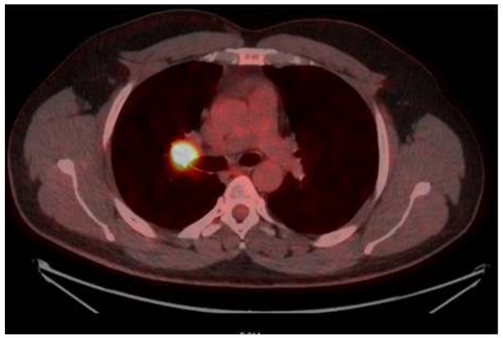
Myeloid/DC MOA:
Targets TIM-3 to increase phagocytosis, tumor antigen presentation, and antitumor T cell expansion

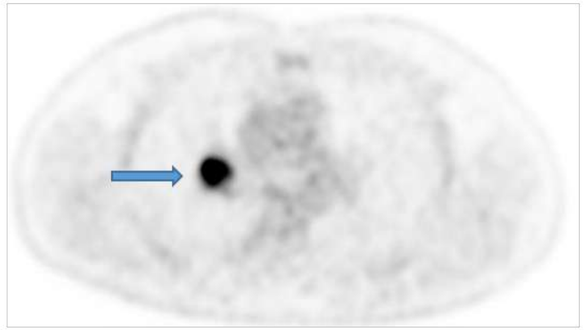


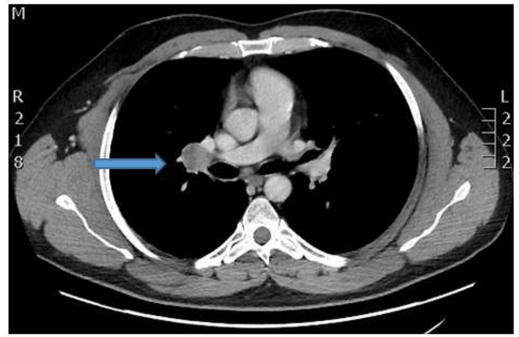
Presented at the 2024 Annual Meeting of the European Hematology Association (EHA), June 13-16, 2024

…. Be continued

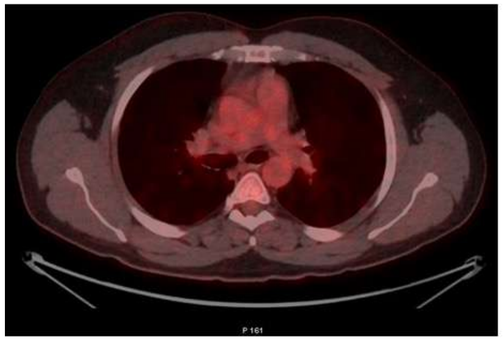
Baseline

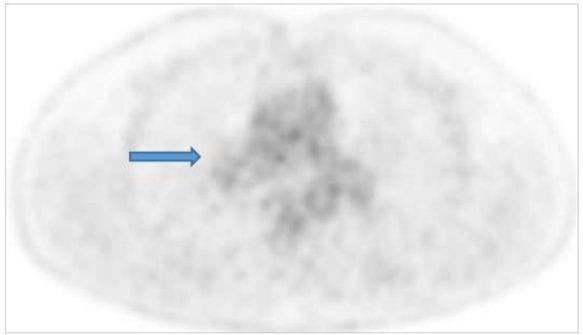


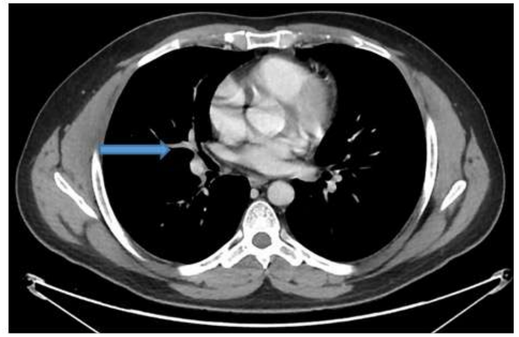




After 3 courses of AZD7789







CR 35 cycles tot
27 Sep 2025: 6 mo FU

Cosa fareste?

a) Riproporre Tipizzazione HLA

b) Riproporre Trapianto allogenico

c) Avviare a Follow-up

d) Radioterapia

Antibody-drug conjugates

❑ Ab anti CD25: Camidanlumab tesirine

Camidanlumab tesirine: updated efficacy and safety in an open-label, multicenter, phase 2 study of patients with relapsed or refractory classical Hodgkin lymphoma (R/R cHL)

Carmelo Carlo-Stella[1], Stephen Ansell[2], Pier Luigi Zinzani[3], John Radford[4], Kami Maddocks[5], Antonio Pinto[6], Graham P. Collins[7], Veronika Bachanova[8], Nancy Bartlett[9], Isabelle Bence-Bruckler[10], Mehdi Hamadani[11], Justin Kline[12], Jiri Mayer[13], Kerry J. Savage[14], Ranjana Advani[15], Paolo Caimi[16], René-Olivier Casasnovas[17], Tatyana Feldman[18], Brian Hess[19], Mariana Bastos-Oreiro[20], Sunil Iyengar[21], Sandy Eisen[22†], Yanina Negievich[22], Luqiang Wang[23], Jens Wuerthner[22], Alex F. Herrera[24*]

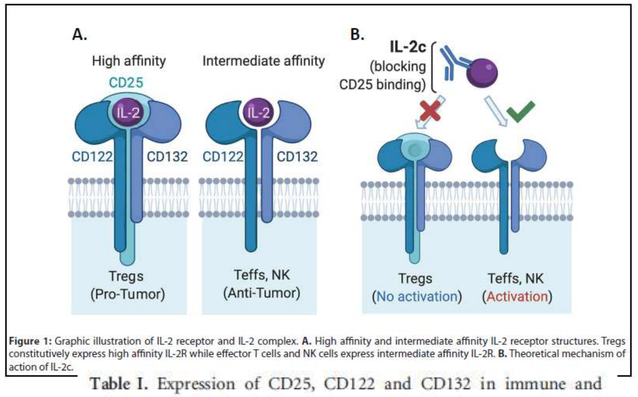
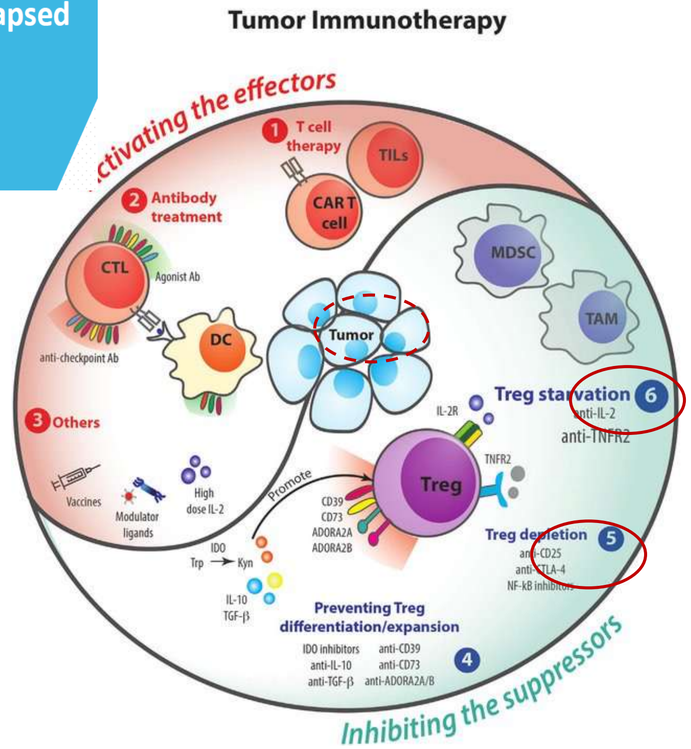


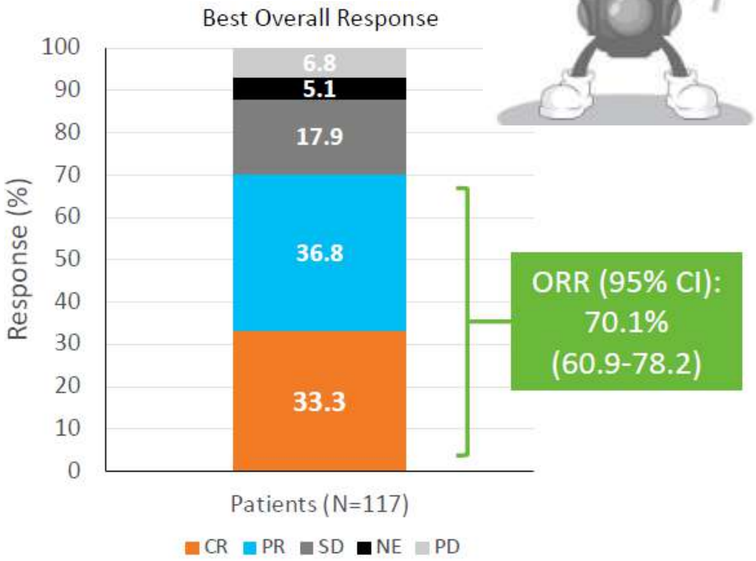
Figure 1: Graphic illustration of IL-2 receptor and IL-2 complex. **A.** High affinity and intermediate affinity IL-2 receptor structures. Tregs constitutively express high affinity IL-2R while effector T cells and NK cells express intermediate affinity IL-2R. **B.** Theoretical mechanism of action of IL-2c.

Table I. Expression of CD25, CD122 and CD132 in immune and non-immune cells.

Cell type	CD25	CD122	CD132
Thymocyte	–/+	–/+	+
Naïve T cell	–	–/+	+
Effector T cell	+++	++	+
Memory T cell	–	+/++	+
Treg	+++	+	+
Immature B cell	+	–	+
Mature B cell	–	–	+
NK cell	–	++	+
NK T cell	–/+	–/+	+
Langerhans cell	+	?	+
Endothelial cell	+	+	+



- Primary endpoint: ORR (per 2014 Lugano classification) assessed by central review
- Secondary endpoints: DoR, PFS, safety (frequency and severity of adverse events)
- As of November 1, 2021, enrollment was complete **(N=117)**



Best Overall Response in Patients with or without prior SCT

Best Overall response, n (%)[b]	BV and CHPi With Prior SCT (n=73), n (%)	BV and CHPi Without Prior SCT (n=43), n (%)
CR	30 (41.1)	8 (18.6)
PR	24 (32.9)	19 (44.2)
SD	13 (17.8)	8 (18.6)
NE[c]	3 (4.1)	3 (7.0)
PD	3 (4.1)	5 (11.6)
ORR 95% CI for ORR	54 (74.0) 62.4-83.5	27 (62.8) 46.7-77.0

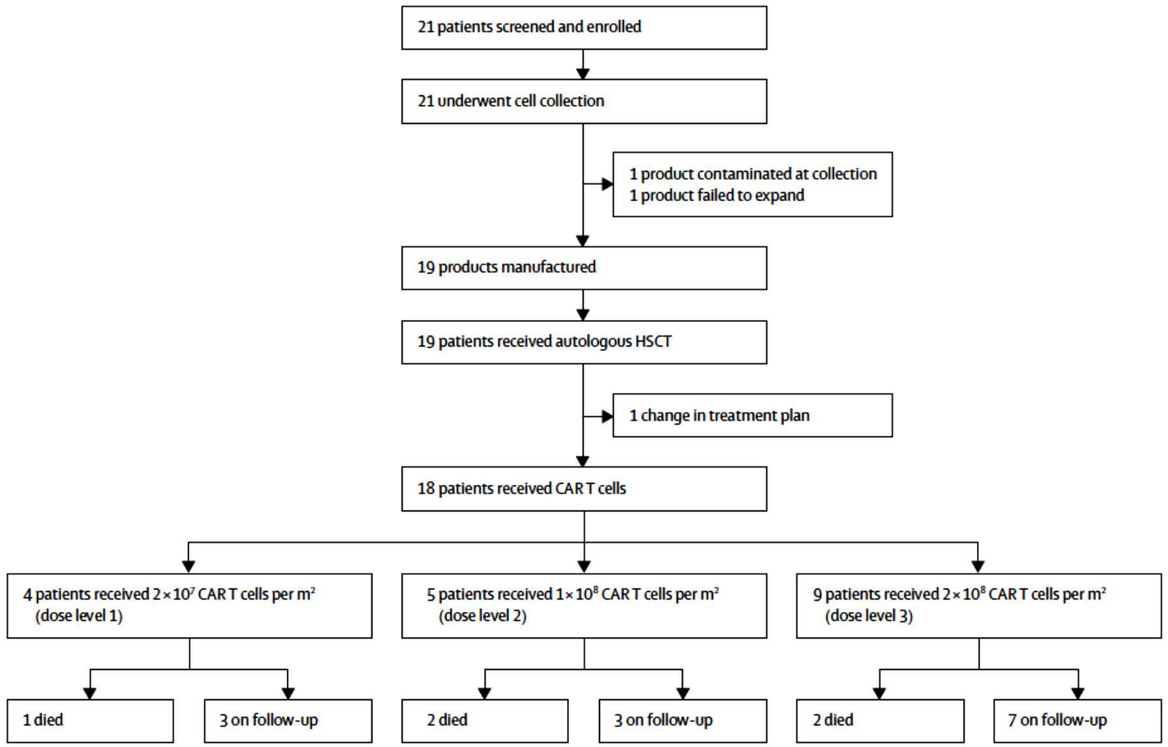
1. Sasidharan NV, et al. *Immunol Cell Biol*. 2018;96:21–33. 2. Arce Vargas F, et al. *Immunity*. 2017;46:577–86. 3. Hartley JA. *Expert Opin Investig Drugs*. 2011;20:733–44. 4. Flynn MJ, et al. *Mol Cancer Ther*. 2016;15:2709–21.
ADC, antibody drug conjugate; CD25, cluster of differentiation 25; PBD, pyrrolobenzodiazepine; Teff, effector T cell; Treg, regulatory T cell; Val-Ala, valine-alanine..

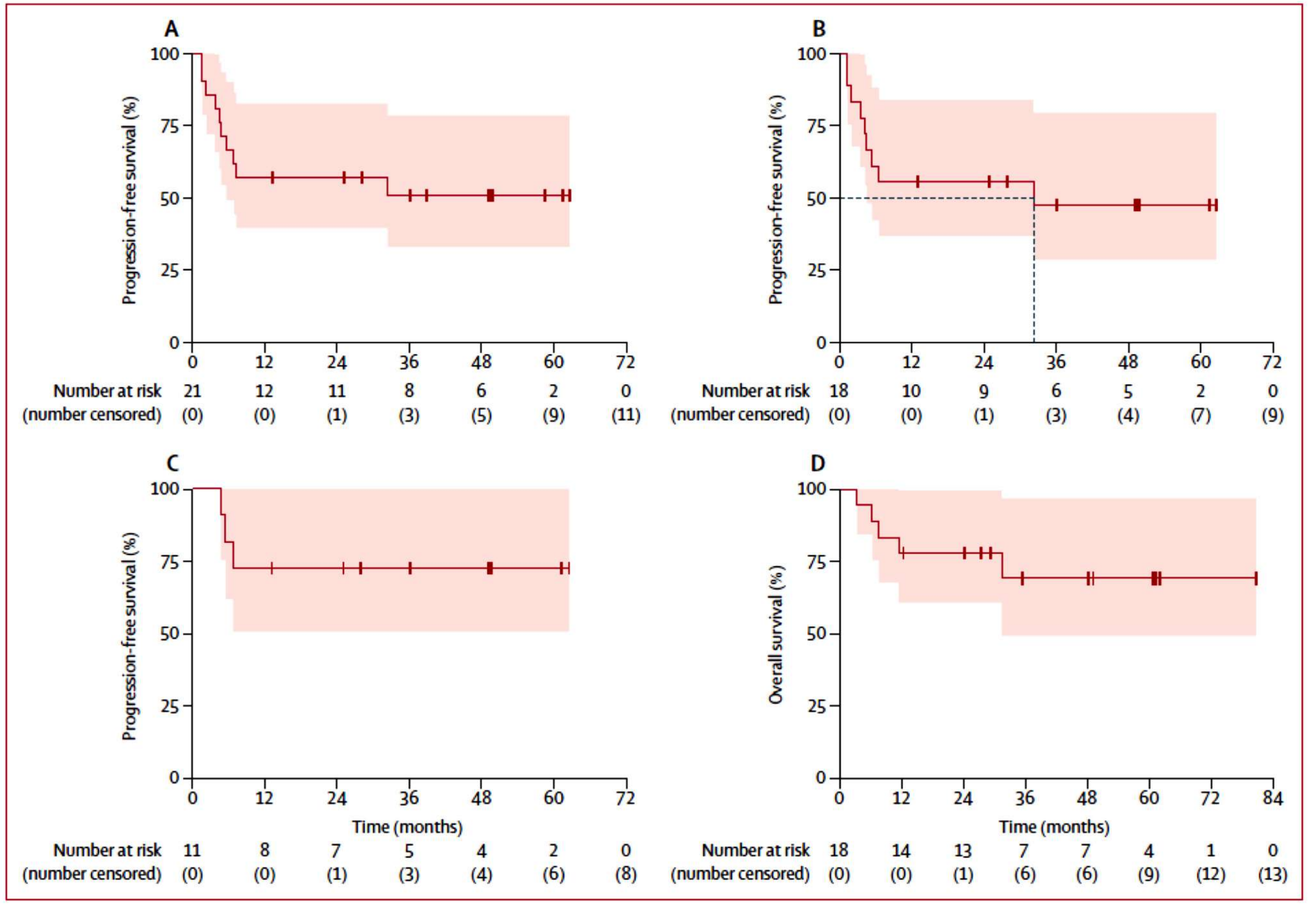
Cellular Therapy.1

❑ CAR-T anti CD30+

Anti-CD30 CAR T cells as consolidation after autologous haematopoietic stem-cell transplantation in patients with high-risk CD30⁺ lymphoma: a phase 1 study

Natalie S Grover, George Hucks, Marcie L Riches, Anastasia Ivanova, Dominic T Moore, Thomas C Shea, Mary Beth Seegars, Paul M Armistead, Kimberly A Kasow, Anne W Beaven, Christopher Dittus, James M Coghill, Katarzyna J Jamieson, Benjamin G Vincent, William A Wood, Catherine Cheng, Julia Kaitlin Morrison, John West, Tammy Cavallo, Gianpietro Dotti, Jonathan S Serody, Barbara Savoldo



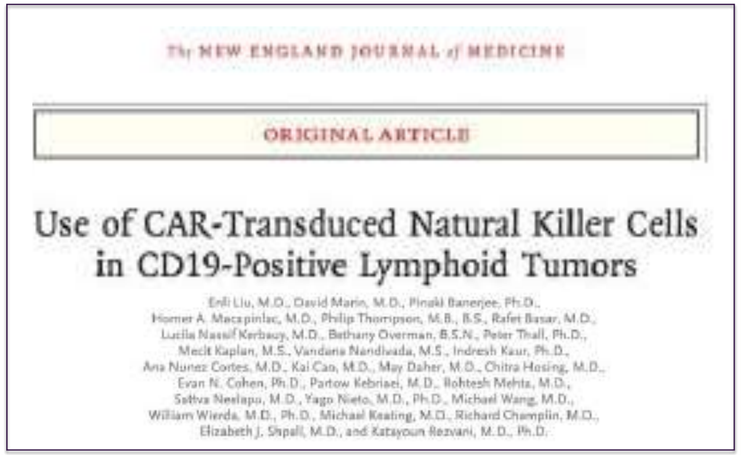


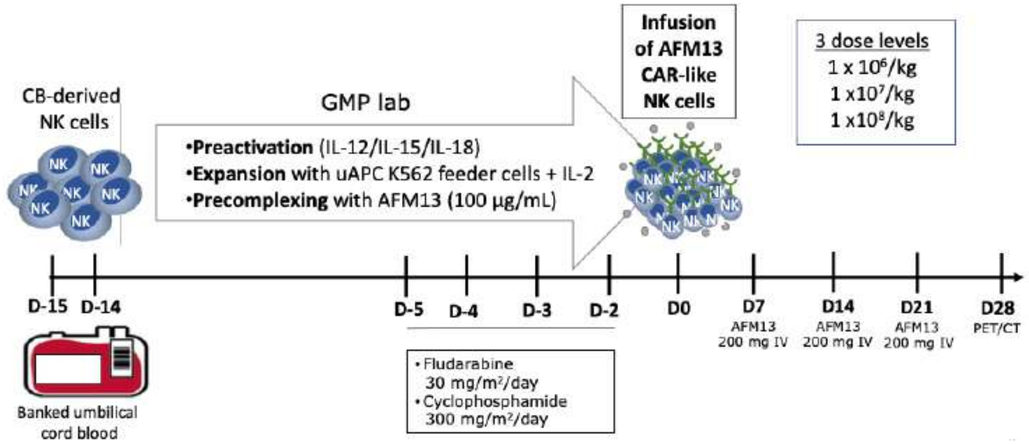
Lancet Haematology,2024

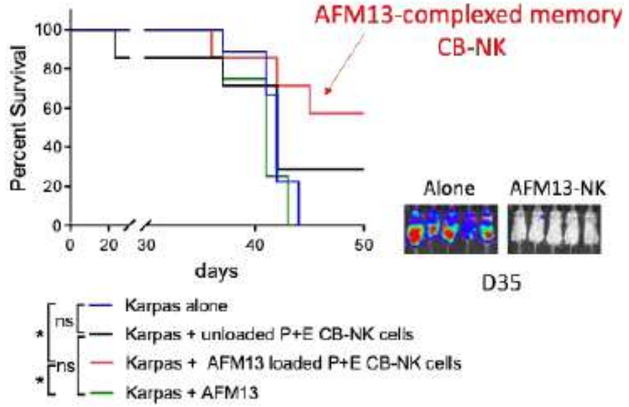
Cellular Therapy.2

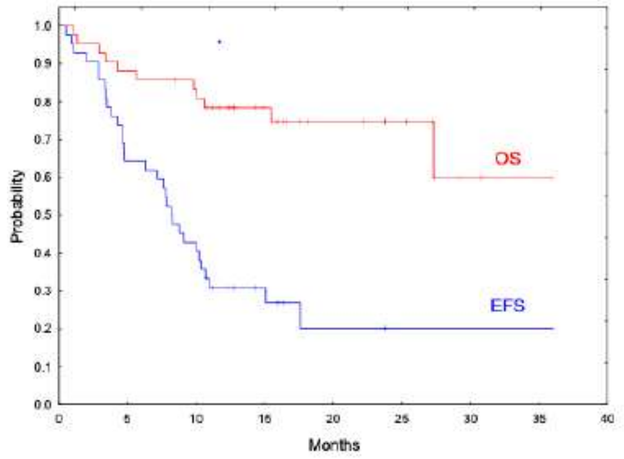
❑ AFM13-complexed CB-Derived NK cells for R/R CD30+Lymphoma NCT04074746

First In-human









	Patients treated at RP2D			
	Overall (N=36)		Hodgkin (N=32)	
	2 cycles (N=13)	4 cycles (N=23)	2 cycles (N=12)	4 cycles (N=20)
Median EFS	8 months	10 months	8 months	10 months
At 6 months	62%	70%	67%	68%
At 12 months	31%	29%	33%	27%
Median OS	NR	NR	NR	NR
At 6 months	85%	87%	92%	85%
At 12 months	85%	85%	92%	82%

Kerbauy et al, Clin Cancer Res 2021

Conclusions

- Plethora of emerging immunotherapies in R/R cHL that are poised to further improve outcomes for patients, including those who progress after BV and CPI
- While many of these therapies have demonstrated encouraging activity, rational combinations are needed to improve the durability of responses
- Advances in our understanding of cHL biology, may enable more biologically-tailored treatment approaches in the future

Grazie per l’attenzione!!!!!!!!!!!!!!!