Presentation skills

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Objectives

- Understand why presenting can be fun and beneficial to your career
- Become familiar with key concepts for good presentations
- Learn what to avoid as you develop talks/ presentations

Why give a talk?

- Share what you have learned/discovered
- Get credit for your hard work
- Advance the field
- Prompt feedback and dialogue
- Advance your career
- Gain experience
- Networking

Pitfalls in giving a talk

- Inadequate preparation
- Not knowing your audience
- Not knowing your topic
- Too many slides/going over time
- Bad slides
- Overstating your conclusions – “It is what it is”
- Not anticipating questions that will come

Things you don't have to do

- Be funny
- Know everything
- Have the answer to every question
- Cram in everything

Things you should do

- Take your time but be on time
- Make sure your main messages are clear
- Make sure the main message of each slide is clear
- Tell a story
- Acknowledge those who contributed to the work
- Acknowledge those who did work in the area before you
- Leave with some ideas about future questions

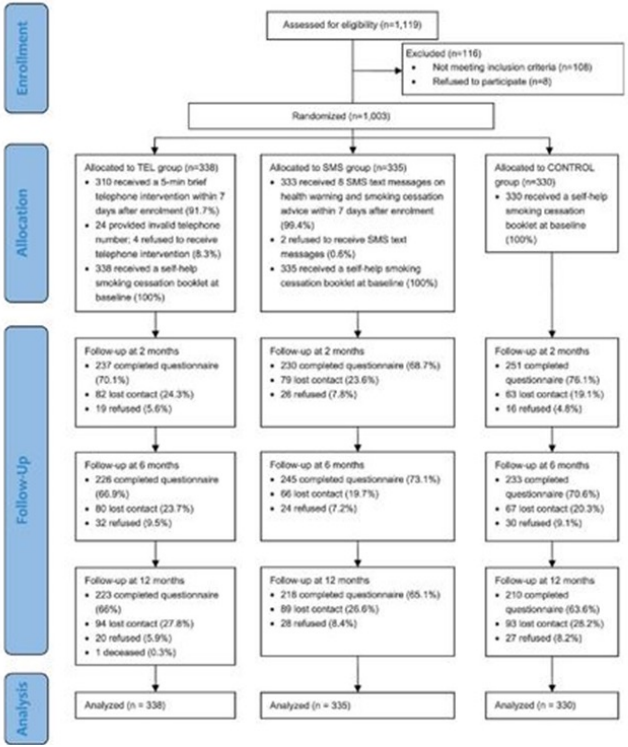
With apologies to Led Zeppelin

Good slides, bad slides, you know I've had my share….

“I know you can’t read this slide”

“I know this is a busy slide”

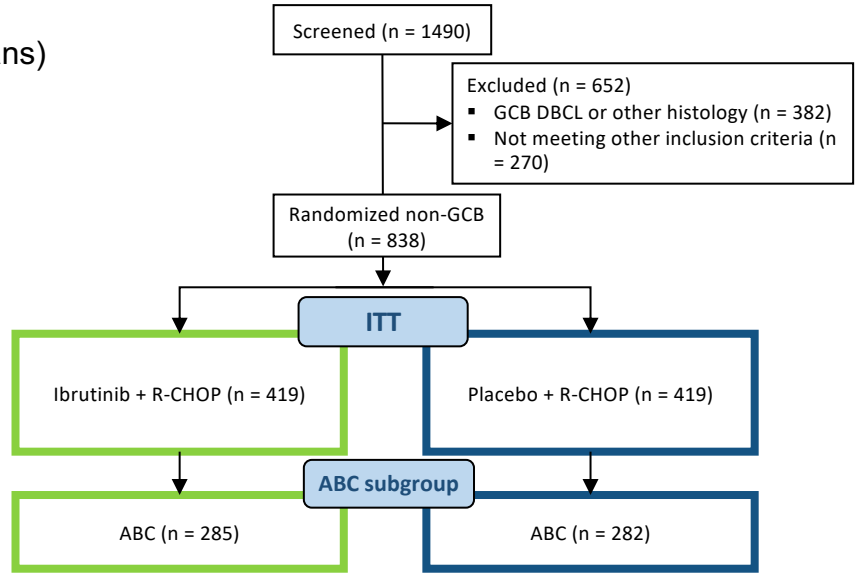
CONSORT Diagram



PHOENIX: R-CHOP ± Ibrutinib

Key eligibility criteria

- Untreated non-GCB DLBCL (Hans)
- Stage II to IV disease
- R-IPI ≥ 1
- ECOG performance status ≤ 2



Younes, et al. *J Clin Oncol*. 2019

WHO Lymphoma Classification 2016

Table 1. 2016 WHO classification of mature lymphoid, histiocytic, and dendritic neoplasms

Mature B-cell neoplasms
Chronic lymphocytic leukemia/small lymphocytic lymphoma
Monoclonal B-cell lymphocytosis*
B-cell prolymphocytic leukemia
Splenic marginal zone lymphoma
Hairy cell leukemia
Splenic B-cell lymphoma/leukemia, unclassifiable
Splenic diffuse red pulp small B-cell lymphoma
Hairy cell leukemia-variant
Lymphoplasmacytic lymphoma
Waldenström macroglobulinemia
Monoclonal gammopathy of undetermined significance (MGUS), IgM*
μ heavy-chain disease
γ heavy-chain disease
α heavy-chain disease
Monoclonal gammopathy of undetermined significance (MGUS), IgG/A*
Plasma cell myeloma
Solitary plasmacytoma of bone
Extraosseous plasmacytoma
Monoclonal immunoglobulin deposition diseases*
Extranodal marginal zone lymphoma of mucosa-associated lymphoid tissue (MALT lymphoma)
Nodal marginal zone lymphoma
Pediatric nodal marginal zone lymphoma
Follicular lymphoma
In situ follicular neoplasia*
Duodenal-type follicular lymphoma*
Pediatric-type follicular lymphoma*
*Large B-cell lymphoma with IRF4 rearrangement**
Primary cutaneous follicle center lymphoma
Mantle cell lymphoma
In situ mantle cell neoplasia*
Diffuse large B-cell lymphoma (DLBCL), NOS
Germinal center B-cell type*
Activated B-cell type*
T-cell/histiocyte-rich large B-cell lymphoma
Primary DLBCL of the central nervous system (CNS)
Primary cutaneous DLBCL, leg type
EBV$^+$ DLBCL, NOS*
*EBV$^+$ mucocutaneous ulcer**
DLBCL associated with chronic inflammation
Lymphomatoid granulomatosis
Primary mediastinal (thymic) large B-cell lymphoma
Intravascular large B-cell lymphoma
ALK$^+$ large B-cell lymphoma
Plasmablastic lymphoma
Primary effusion lymphoma
*HHV8$^+$ DLBCL, NOS**
Burkitt lymphoma
*Burkitt-like lymphoma with 11q aberration**
High-grade B-cell lymphoma, with *MYC* and *BCL2* and/or *BCL6* rearrangements*
High-grade B-cell lymphoma, NOS*
B-cell lymphoma, unclassifiable, with features intermediate between DLBCL and classical Hodgkin lymphoma
Mature T and NK neoplasms
T-cell prolymphocytic leukemia
T-cell large granular lymphocytic leukemia
Chronic lymphoproliferative disorder of NK cells
Aggressive NK-cell leukemia
Systemic EBV$^+$ T-cell lymphoma of childhood*
Hydroa vacciniforme–like lymphoproliferative disorder*
Adult T-cell leukemia/lymphoma
Extranodal NK/T-cell lymphoma, nasal type
Enteropathy-associated T-cell lymphoma

Table 1. (continued)

Monomorphic epitheliotropic intestinal T-cell lymphoma*
*Indolent T-cell lymphoproliferative disorder of the GI tract**
Hepatosplenic T-cell lymphoma
Subcutaneous panniculitis-like T-cell lymphoma
Mycosis fungoides
Sézary syndrome
Primary cutaneous CD30$^+$ T-cell lymphoproliferative disorders
Lymphomatoid papulosis
Primary cutaneous anaplastic large cell lymphoma
Primary cutaneous γδ T-cell lymphoma
Primary cutaneous CD8$^+$ aggressive epidermotropic cytotoxic T-cell lymphoma
*Primary cutaneous acral CD8$^+$ T-cell lymphoma**
*Primary cutaneous CD4$^+$ small/medium T-cell lymphoproliferative disorder**
Peripheral T-cell lymphoma, NOS
Angioimmunoblastic T-cell lymphoma
*Follicular T-cell lymphoma**
*Nodal peripheral T-cell lymphoma with TFH phenotype**
Anaplastic large-cell lymphoma, ALK$^+$
Anaplastic large-cell lymphoma, ALK$^-$ *
*Breast implant–associated anaplastic large-cell lymphoma**
Hodgkin lymphoma
Nodular lymphocyte predominant Hodgkin lymphoma
Classical Hodgkin lymphoma
Nodular sclerosis classical Hodgkin lymphoma
Lymphocyte-rich classical Hodgkin lymphoma
Mixed cellularity classical Hodgkin lymphoma
Lymphocyte-depleted classical Hodgkin lymphoma
Posttransplant lymphoproliferative disorders (PTLD)
Plasmacytic hyperplasia PTLD
Infectious mononucleosis PTLD
Florid follicular hyperplasia PTLD*
Polymorphic PTLD
Monomorphic PTLD (B- and T-/NK-cell types)
Classical Hodgkin lymphoma PTLD
Histiocytic and dendritic cell neoplasms
Histiocytic sarcoma
Langerhans cell histiocytosis
Langerhans cell sarcoma
Indeterminate dendritic cell tumor
Interdigitating dendritic cell sarcoma
Follicular dendritic cell sarcoma
Fibroblastic reticular cell tumor
Disseminated juvenile xanthogranuloma
Erdheim-Chester disease*

Provisional entities are listed in italics.
*Changes from the 2008 classification.

small population, but in others associated with a lymphocytosis.[4] Whereas in 2008 it was unknown whether MBL was a precursor of CLL, we now know that MBL precedes virtually all cases of **CLL/small lymphocytic lymphoma** (SLL).[5] The updated WHO will retain the current criteria for MBL, but will emphasize that "low-count" MBL, defined as a PB CLL count of $<0.5 \times 10^9$/L, must be distinguished from "high-count" MBL because low count MBL has significant differences from CLL, an extremely limited, if any, chance of progression, and, until new evidence is provided, does not require routine follow-up outside of standard medical care.[6,7] In contrast, high-count MBL requires routine/yearly follow-up, and has very similar phenotypic and genetic/molecular features as Rai stage 0 CLL, although immunoglobulin heavy chain variable region (IGHV)-mutated cases are more frequent in MBL.[8] Also impacting our diagnostic criteria, the revision will eliminate the option to diagnose CLL with $<5 \times 10^9$/L PB CLL cells in the absence of extramedullary

100 + entities

Swerdlow, et al. *Blood.* 2016

Lymphoma Classification 1982-1994

National Cancer Institute Sponsored Study of Classifications of Non-Hodgkin's Lymphomas

Summary and Description of a Working Formulation for Clinical Usage

THE NON-HODGKIN'S LYMPHOMA PATHOLOGIC CLASSIFICATION PROJECT*

Low Grade	Intermediate Grade	High Grade
Small lymphocytic	Follicular large cell	Large cell immunoblastic
Follicular small-cleaved cell	Diffuse small cleaved cell	Lymphoblastic
Follicular mixed small-cleaved and large cell	Diffuse mixed small and large cell	Small non-cleaved cell (Burkitt and non-Burkitt type)
	Diffuse large cell	

Morphology → Phenotype → Genetic → Molecular

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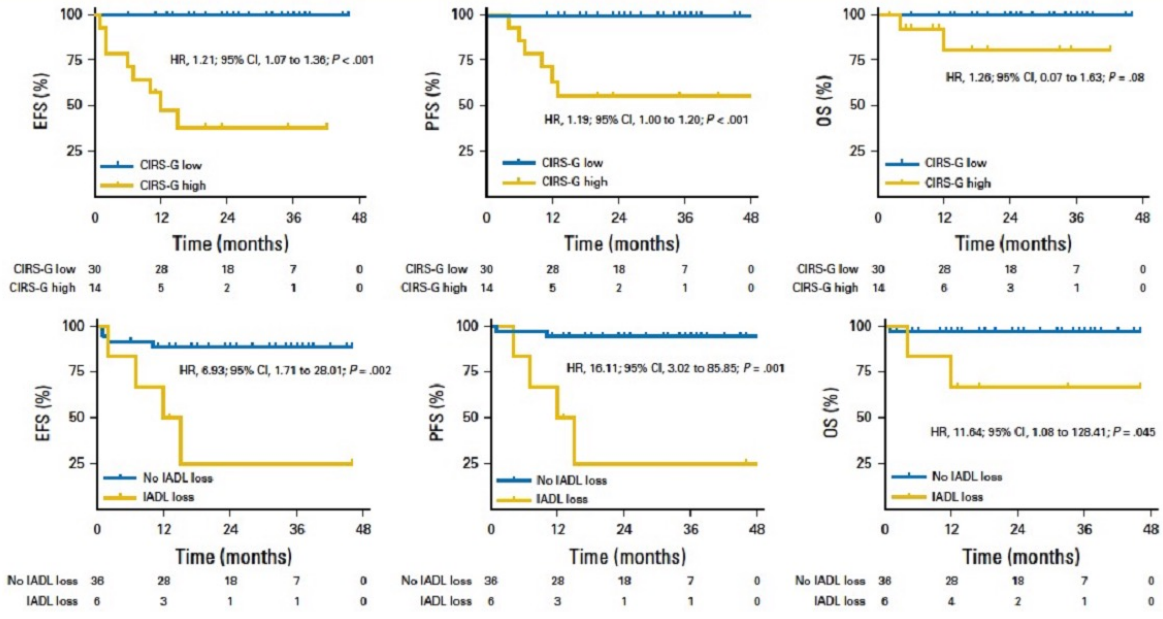
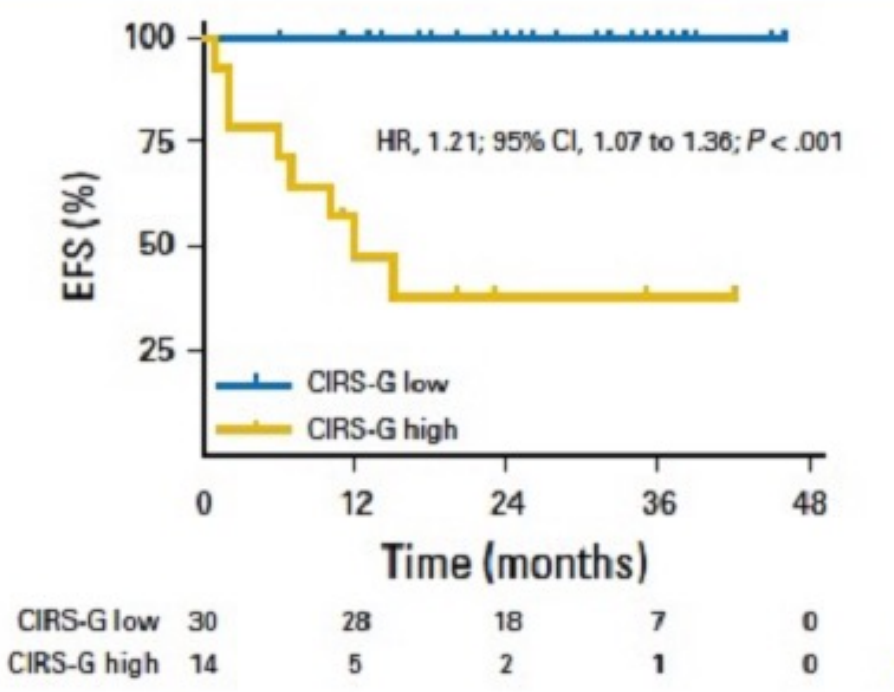
100 + entities

Swerdlow, et al. *Blood.* 2016

Was “functional status” prognostic of outcome?

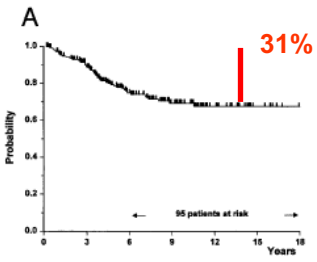


Functional status predicts outcome



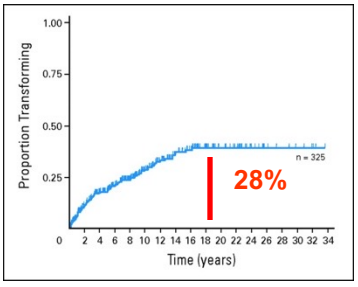
Incidence of transformed lymphoma

freedom from transformation



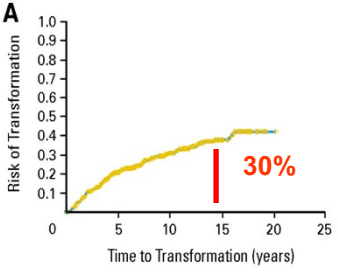
Lyon: n=220

cumulative incidence of transformation



St. Barts: n=325

actuarial risk of transformation



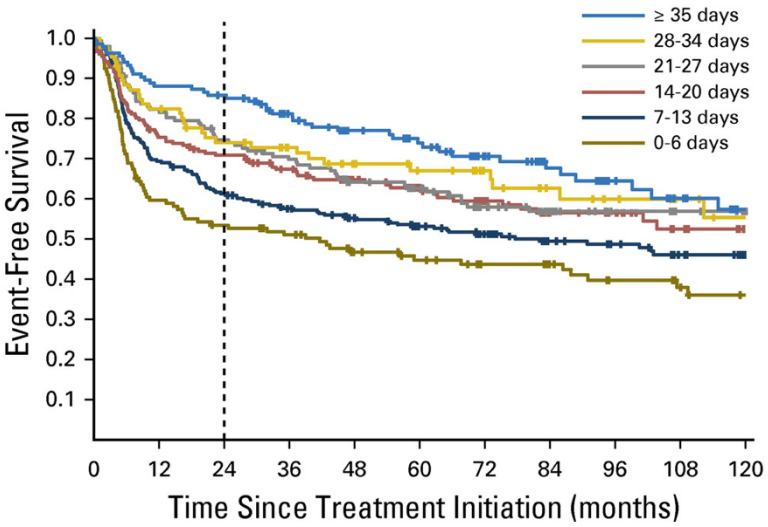
Vancouver: n=600

Transformation rate ~ 30% at 10 years

Bastion Y, et al. *J Clin Oncol*. 1997; Montoto S, et al. *J Clin Oncol*. 2007
Al-Tourah AJ, et al. *J Clin Oncol*. 2008.

Diagnosis to treatment interval (DTI) is important clinical factor in DLBCL; implications for trials

- Shorter DTI was strongly associated with adverse clinical factors
  - LDH, IPI, PS
- These patients had worse outcomes and are almost certainly underrepresented in clinical trials



Maurer, et al. *J Clin Oncol.* 2018;36:1603-1610

The Numbers Game
Truth (deception) in reporting

Misrepresentation of data info on number of pts ie not ITT in most studies

Actual results for ASCT based on a fraction of total pts in most studies who are highly selected

Author	Reference	Lymphoma Subtypes	Number of Patients	Induction Regimen	Consolidation Strategies	Survival in Patients in Complete Remission After Induction
Savage et al., 2022	36	ALCL, AITL, PTCL, NOS (mostly ALK − ALCL) in CR after induction	N=452 211 N=67(CR)	BV-CHP (n = 114) CHOP (n = 97)	Autologous SCT vs. no consolidation	BV-CHP + Auto SCT: 3-yr PFS 80.4% BV-CHP + no SCT: 3-yr PFS 54.9% CHOP + Auto SCT: 3-yr PFS 67.2% CHOP + no SCT: 3-yr PFS 54.1%
Advani et al., 2021	38	AITL	282 N=27 (CR)	Anthracycline-based w/o etoposide 65%, anthracycline-based with etoposide 16% Other 19%	Autologous SCT vs. no consolidation	Auto SCT: 5-yr PFS 79% No auto SCT: 5-yr PFS 31% Auto SCT: 5-yr OS 89% No auto SCT: 5-yr OS 52%
		A PTCL	499 N=36 (CR)	Anthracycline-based w/o etoposide 42%, anthracycline-based with etoposide 21% Other 37%	Autologous SCT vs. no consolidation	Auto SCT: 5-yr OS 87.8% No auto SCT: 5-yr OS 70.2%
			213 N=117/86 (CR)	CHOP or CHOEP	Autologous SCT vs. no consolidation	Auto SCT: 5-yr OS 82% No auto SCT: 5-yr OS 47%
		ALK − , AITL, PTC , NOS	174 N=103 (CR)	CHOP (n = 126) CHOEP (n = 16) Other (n = 32)	Autologous SCT vs. no consolidation	Auto SCT: 5-yr PFS 63% No auto SCT: 5-yr PFS 49% Auto SCT: 5-yr OS 74% No auto SCT: 5-yr OS 62%
		All PTCL	906 N=181	Heterogeneous protocols	Autologous SCT vs. no consolidation	Auto SCT: 5-yr PFS 41% * No auto SCT: 5-yr PFS 46% * Auto SCT: 5-yr OS 49% * No auto SCT: 5-yr OS 59.5% *
Ellin et al., 2014	22	All PTCL	755 N=104	CHOP or CHOEP (n = 499)	Autologous SCT vs. no consolidation	Better for the auto SCT group (estimates not given) *
Schmitz et al., 2021	55	All PTCL other than ALK ALCL	104 N=67/45 (CR)	CHOEP × 4 + DHAP × 1	Autologous SCT vs. allogeneic SCT (if donor available)	Auto SCT: 3-yr PFS 39% * Allo SCT: 3-yr PFS 43% * Auto SCT: 3-yr OS 70% * Allo SCT: 3-yr OS 57% *

* Compared groups were based on intention-to-transplant (or intent to treat in the Schmitz et al. trial) rather than based on the achievement of remission after induction. NB: All studies were retrospective other than that by Schmitz et al., 2022, which is a randomized clinical trial.

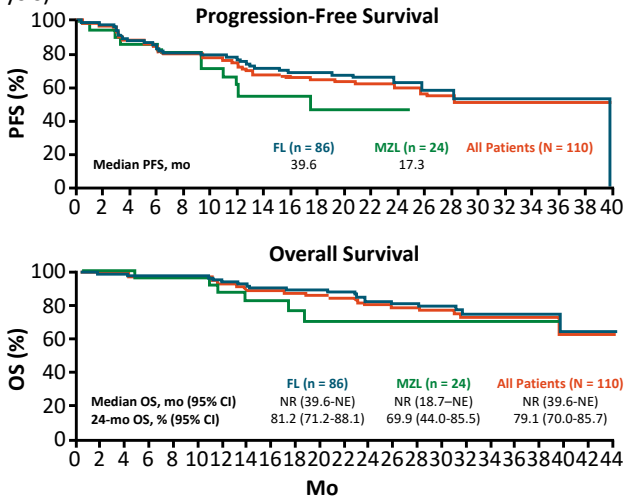
Sorigue et al, Cancers 2023

ZUMA-5: Axicabtagene Ciloleucel for R/R Indolent NHL (FL or MZL)

- Single-arm phase II study of axicabtagene ciloleucel for patients with R/R indolent B-cell NHL (FL or MZL) **with ≥2 prior therapies** (N = 110 eligible for efficacy analysis)

Outcome	FL (n = 86)	MZL (n = 24)	All (N = 110)
ORR, n (%)	81 (94)	20 (83)	--
▪ CR	68 (79)	15 (63)	--
▪ PR	13 (15)	5 (21)	--
▪ SD	3 (3)	0	--
▪ PD	0	1 (4)	--
▪ ND	2 (2)	3 (13)	
Median DoR, mo (95% CI)	38.6 (24.7-NE)	NR (8.2-NE)	38.6 (24.7-NE)
24-mo DoR, % (95% CI)	66.1 (53.9-75.8)	NR (NE-NE)	63.5 (52.4-72.7)

- CRS grade ≥3, 7% (6% FL); neurotoxicity grade ≥3, 19% (15% FL); tocilizumab, 49%; corticosteroids, 36%



Neelapu SS et al. ASH 2021. Abstract 93.

Weill Cornell Medicine

NewYork-Presbyterian

ZUMA-5 Outcomes by POD24 Status – ASH 2022

Parameter (95% CI)	Follicular Lymphoma (n=127)[a]	
	With POD24 (n=63)	Without POD24 (n=40)
Median DOR, months	NR (36.6–NE)	NR (24.7–NE)
36-month rate, %	64.6 (50.9–75.3)	52.7 (33.9–68.4)
Median PFS, months	40.2 (15.9–NE)	NR (25.4–NE)
36-month rate, %	59.2 (46.3–70.0)	52.2 (33.4–68.0)
Median OS, months	NR (NE–NE)	NR (NE–NE)
36-month rate, %	75.4 (63.4–83.9)	73.8 (56.5–85.0)

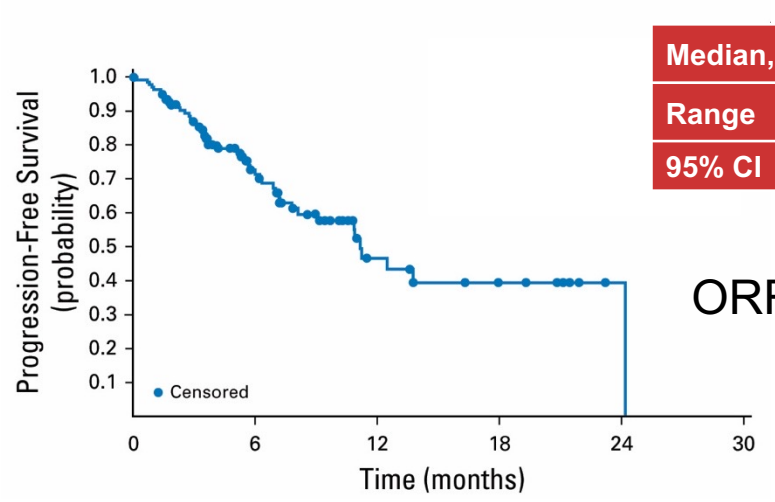
Neelapu SS et al. ASH 2022. Abstract 4660.

ZUMA-5 CRS and Neurologic Events

Parameter	CRS[a] FL (n=124)	CRS[a] MZL (n=22)	CRS[a] All Patients (N=146)	Neurologic Events[a] FL (n=124)	Neurologic Events[a] MZL (n=22)	Neurologic Events[a] All Patients (N=146)
Any grade	97 (78)	22 (100)	119 (82)	70 (56)	17 (77)	87 (60)
Grade ≥3	8 (6)	2 (9)	10 (7)	19 (15)	9 (41)	28 (19)
Most common CRS symptoms of any grade, n/n (%)						
Pyrexia	94/97 (97)	20/22 (91)	114/119 (96)	–	–	–
Hypotension	39/97 (40)	10/22 (45)	49/119 (41)	–	–	–
Most common neurologic events of any grade, n/n (%)						
Tremor	–	–	–	36/70 (51)	9/17 (53)	45/87 (52)
Confusional state	–	–	–	28/70 (40)	7/17 (41)	35/87 (40)
Tocilizumab use, n (%)	56 (45)	15 (68)	71 (49)	7 (6)	2 (9)	9 (6)
Corticosteroid use, n (%)	19 (15)	6 (27)	25 (17)	38 (31)	14 (64)	52 (36)
Median time to onset (range), days	4 (1–15)	4 (1–9)	4 (1–15)	7 (1–177)	7 (3–19)	7 (1–177)
Median duration of events (range), days	6 (1–27)	6 (2–14)	6 (1–27)	14 (1–452)	10 (2–81)	14 (1–452)
Patients with resolved events, n/n (%)	96/97 (99)[b]	22/22 (100)	118/119 (99)[b]	67/70 (96)	14/17 (82)	81/87 (93)

Jacobson CA et al. ASH 2020. Abstract 700.

PFS of Copanlisib in R/R Indolent Lymphoma



Median, mo	11.2
Range	0.2-24.0
95% CI	8.1-24.0

ORR 59% (12% CR)

No. at Risk

142	54	14	8	1	0

Dreyling M, et al. *J Clin Oncol*. 2017;35:3898-3905.

Bispecific Ab Mosunetuzumab in R/R FL

Phase 2 Pivotal Study

N=90

- Patients aged ≥18 yr with R/R FL grades 1-3a
- CD20+
- ECOG PS ≤1
- ≥2 prior systemic therapies including ≥1 anti-CD20 antibody and ≥1 alkylating agent

Cycle 1 (21-Day Cycles)[a]: **Mosunetuzumab** D1: 1 mg; D8: 2 mg; D15: 60 mg

Cycle 2: **Mosunetuzumab** D1: 60 mg

Cycles 3-8: **Mosunetuzumab** D1: 30 mg

[a] Cycle 1 step-up dosing for CRS mitigation.

Discontinue if CR by cycle 8; if PR or SD, continue treatment for 17 cycles, unless PD or unacceptable toxicity occurs

Primary endpoints

CR (best response) rate by IRF, assessed vs 14% historical control CR rate

Secondary endpoints

ORR, DOR, PFS, safety and tolerability

Outcome, % (95% CI)	By IRF (N=90)	By INV (N=90)
ORR	80 (70-88)	78 (68-86)
▪ CR	60 (49-70)	60 (49-70)

Response by Double Refractory Disease Status, % (95% CI)[1]	Yes (n=48)	No (n=42)
ORR	71 (56-83)	90 (77-97)
▪ CR	50 (35-65)	71 (55-84)
Response by POD ≥24 mo of initial Tx, % (95% CI)[1]	Yes (n=47)	No (n=43)
ORR	85 (72-94)	74 (59-86)
▪ CR	57 (42-72)	63 (47-77)

Budde LE et al. *Lancet Oncol*. 2022;23(8):1055-1065.

Pivotal Phase 2 of Mosunetuzumab in R/R FL: CRS

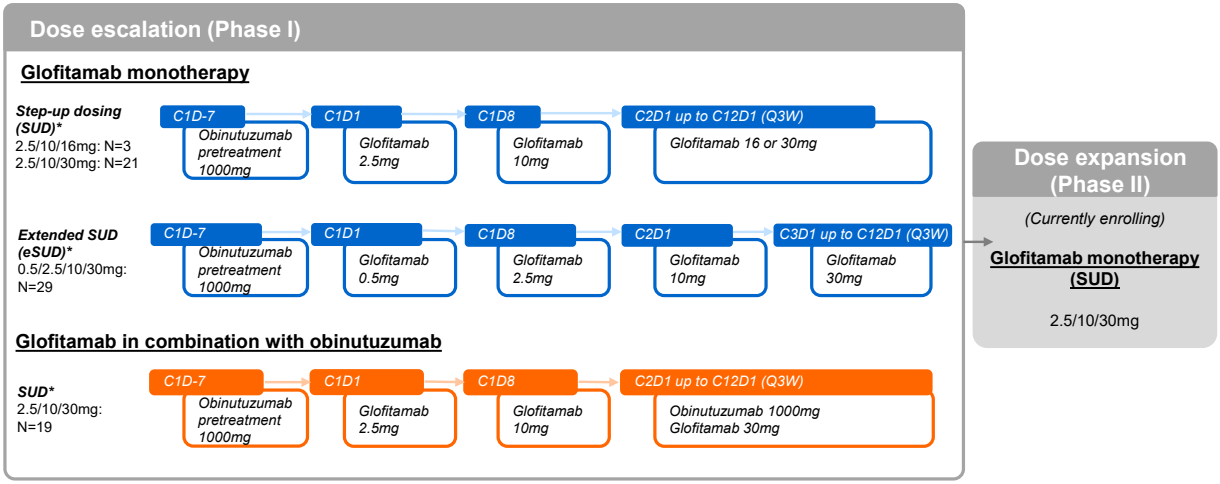
CRS Event	All Patients (N=90)
Any grade, n (%)	40 (44.4)
▪ Grade 1	23 (25.6)
▪ Grade 2	15 (16.7)
▪ Grade 3	1 (1.1)
▪ Grade 4	1 (1.1)
Median time to onset, hr (range)	
▪ C1D1	5.2 (1.2-23.7)
▪ C1D15	26.6 (0.1-390.9)
Median duration, days (range)	3 (1-29)
Patients who received Tx for CRS, n (%)	
▪ Corticosteroids	10 (11.1)
▪ Tocilizumab	7 (7.8)

Patients Who Experienced CRS by Cycle, %	All Patients (N=90)
Cycle (mosunetuzumab dose)	
▪ Cycle 1, D1-7 (1 mg)	23.3
▪ Cycle 1, D8-14 (2 mg)	5.6
▪ Cycle 1, D15-21 (60 mg)	36.4
Cycle (mosunetuzumab dose)	
▪ Cycle 2 (60 mg)	10.3
Cycle (mosunetuzumab dose)	
▪ Cycle 3+ (30 mg)	2.4

- CRS was primarily low grade and occurred mostly in cycle 1; all events of CRS resolved

Budde LE et al. ASH 2021. Abstract 127.

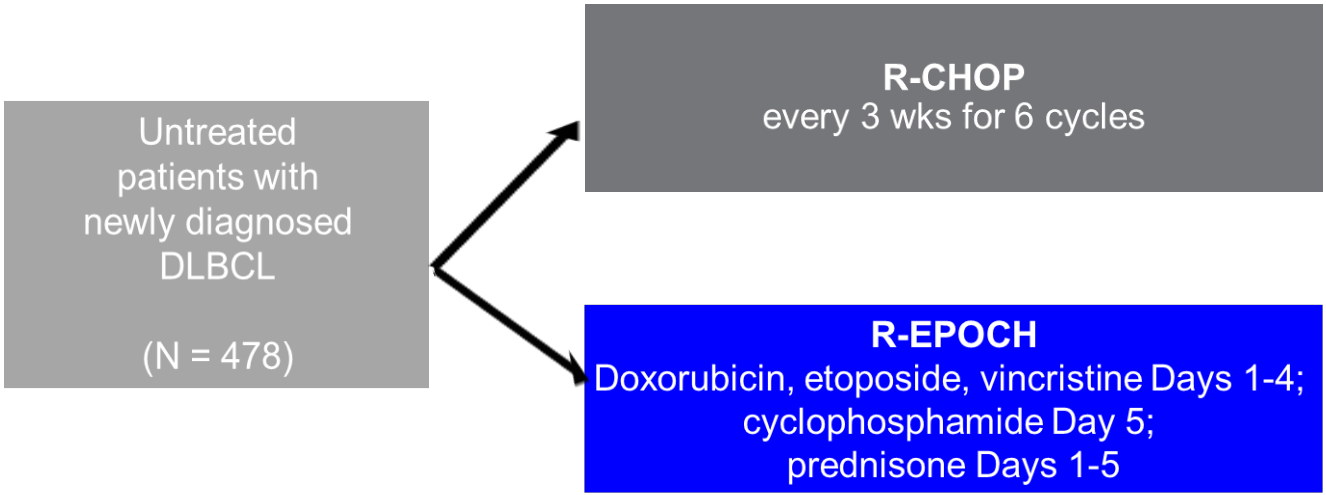
Glofitamab regimens investigated in R/R FL



Population characteristics: *R/R FL Gr 1–3A; ≥1 prior systemic therapy; age ≥18 years; ECOG PS ≤1*

Clinical cut-off date: May 18, 2021; *Glofitamab IV. Gr, Grade; ECOG PS, Eastern Cooperative Oncology Group Performance Status; IV, intravenous; Q3W, every three weeks

Alliance/CALGB 50303: R-CHOP vs R-EPOCH in Newly Diagnosed DLBCL

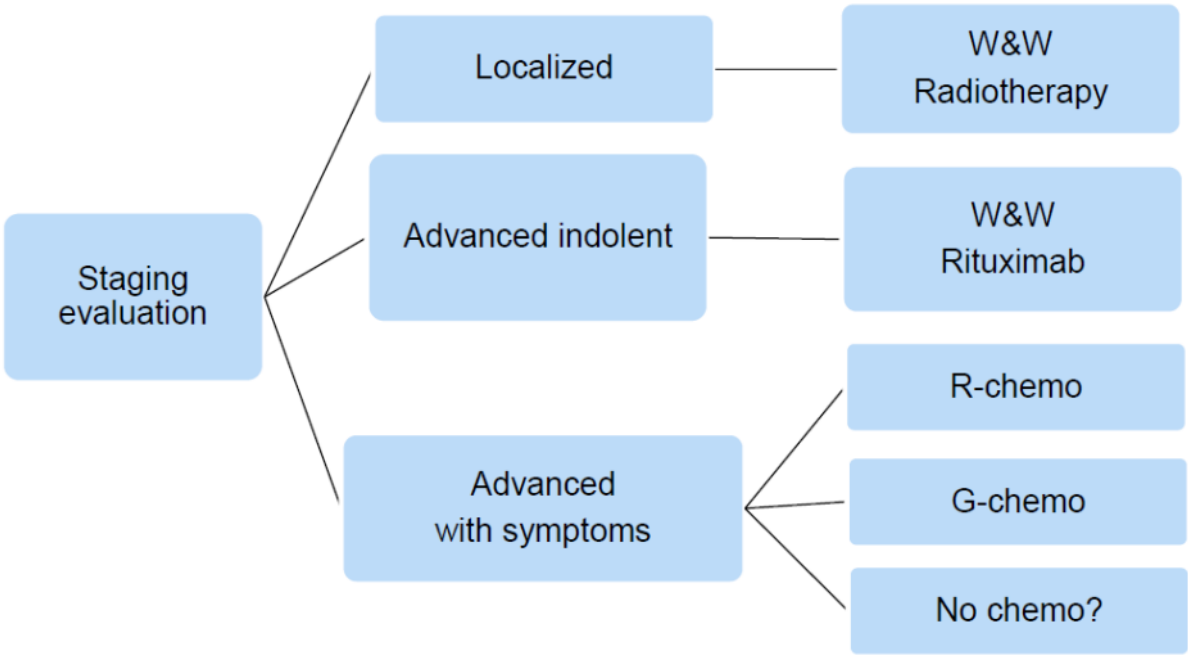


- Primary endpoints: EFS, molecular predictors of outcome for each regimen
- Secondary endpoints: RR,OS, toxicity, use of molecular profiling

Bartlett, et al. *J Clin Oncol*. 2019

Clinical Trials.gov. NCT00118209. http://www.clinicaltrials.gov

One general framework for initial therapy for FL

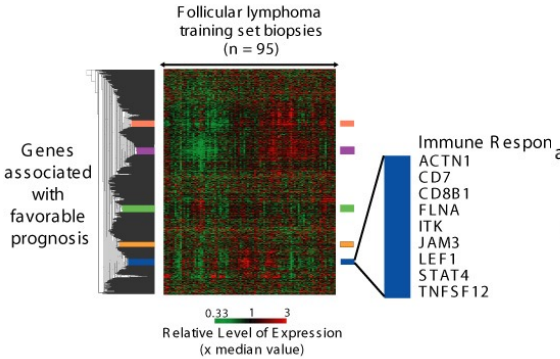


R, rituximab; G, obinutuzumab

Kahl BS, Yang DT. *Blood*. 2016;127:2055-2063.

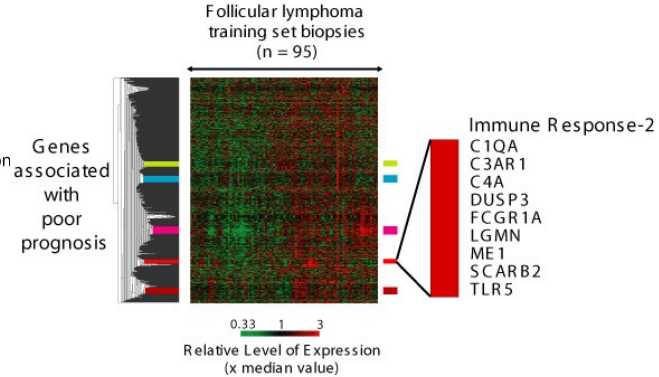
Funky fonts

Identification of gene expression signatures associated with favorable prognosis



Mostly T cell genes?

Identification of gene expression signatures associated with poor prognosis

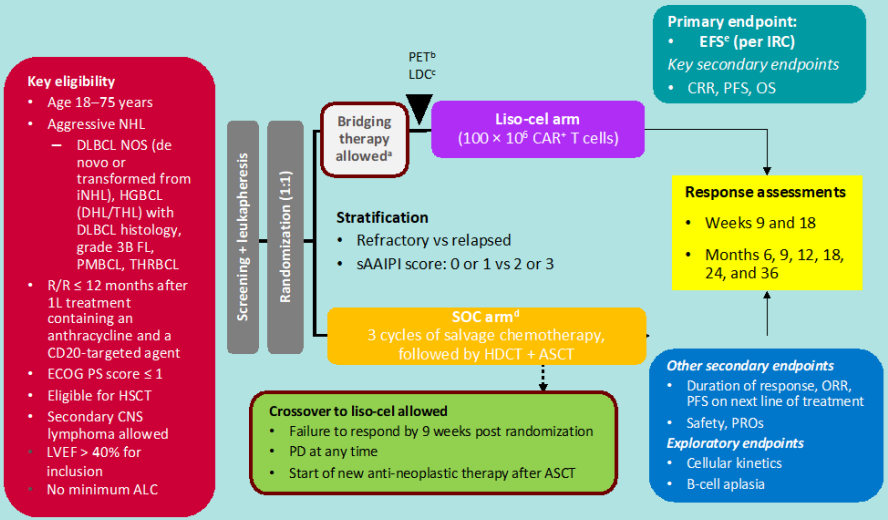


Mostly macrophage genes?

Dave et al NEJM 2004; 351 (21): 2159

Funky fonts and colors

TRANSFORM: liso-cel versus SOC in 2L LBCL



Characteristic	Liso-cel (n = 92)	SOC (n = 92)
Median age (range), years	60 (53.5–67.5)	58 (49–65)
LBCL subtypes, n (%)		
DLBCL NOS	53 (58)	49 (53)
HGBCL (incl. DHL/THL), n (%)	22 (24)	21 (23)
PMBCL	8 (9)	10 (11)
DLBCL transformed from iNHL	7 (8)	8 (9)
Primary refractory, n (%)	67 (73)	68 (74)
Relapsed, n (%)	25 (27)	24 (26)
sAAIPI score, n (%)		
0 or 1	55 (61)	55 (60)
2 or 3	36 (39)	37 (40)
ECOG PS score of 1, n (%)	44 (48)	35 (38)

[a]Patients may have received a protocol-defined SOC regimen to stabilize their disease during liso-cel manufacturing. [b]Only for patients who received bridging therapy. [c]Lymphodepletion with fludarabine 30 mg/m^2 and cyclophosphamide 300 mg/m^2 for 3 days. [d]SOC was defined as physician's choice of R-DHAP, R-ICE, or R-GDP. [e]EFS is defined as time from randomization to death due to any cause, PD, failure to achieve CR or PR by 9 weeks post randomization, or start of a new anti-neoplastic therapy, whichever occurs first. IRC, Independent Review Committee; LDC, lymphodepleting chemotherapy; LVEF, left ventricular ejection fraction; THRBCL, T-cell/histiocyte-rich large B-cell lymphoma.

Kamdar M, et al. Lancet 2022.

What is the message?

Odronextamab

- In R/R FL ORR 78% all doses. With doses of 5 mg or greater: 91%
- CR: 63% CR: 72%

- Median progression free survival: 17.1 mos (range 7.5-not reached)

Odronextamab induces durable FL responses across a variety of dose levels

- In R/R FL ORR 78% all doses. With doses of 5 mg or greater: 91%
- CR: 63% CR: 72%

- Median progression free survival: 17.1 mos (range 7.5-not reached)

Case Presentation

- A 65-year-old male with a history of hypertension and hypercholesterolemia presents with a 2-week history of cervical mass. He has a 30 pack-year smoking history. Feels well.
- Exam shows bilateral cervical LN, firm, 2 cm range.
- CBC normal, LDH and chemistries normal
- Excisional biopsy shows B cell lymphoma, follicular grade II, mixed small and large cell
- What staging tests do you want to perform?

Glofitamab in R/R Follicular lymphoma

- Glofitamab is a T-cell-engaging, CD20xCD3 bispecific, full-length, 2:1 format antibody with bivalent binding to CD20 (B cells) and monovalent binding to CD3 (T cells).
- Glofitamab monotherapy with obinutuzumab pretreatment or combined with obinutuzumab has shown efficacy and manageable safety in heavily pretreated R/R NHL.
- Here, updated results of glofitamab with three different step-up dosing (SUD) regimens as monotherapy (mono) or combined with obinutuzumab (combo) in R/R FL.
- Obinutuzumab (1000mg) was given 7 days prior to the first dose of glofitamab.
- For the 3 mono cohorts, intravenous glofitamab SUD was given on Days (D) 1 and 8 of Cycle (C) 1; then at target dose on C2, or as SUD on C1D1, C1D8, C2D1 and target dose on C3D1.
- For the combo cohort, glofitamab SUD was given on D1 and D8 of C1, then at target dose combined with obinutuzumab 1000mg from C2D1 and onwards (every 21 days for up to 12 cycles). Response rates were based on the Lugano criteria (Cheson *et al*. J Clin Oncol 2014).

Morschhauser et al, ASH 2021, Abstract 128

Introduction

- Patients with FL and MZL typically respond well to first-line immunochemotherapy[1-3]
- Despite being distinct entities, recurrent FL and MZL are treated similarly, commonly with single-agent rituximab[2-4]
- The combination of the immunomodulatory agent lenalidomide with rituximab (R^2) has previously demonstrated promising efficacy in patients with R/R FL[5]
- In the AUGMENT study (NCT01938001), R^2 demonstrated superior efficacy versus R-placebo in patients with R/R iNHL[6]
  - R^2 demonstrated a higher ORR (78% vs 53%) and CRR (34% vs 18%) compared with R-placebo
- Based on these results, R^2 was approved for the treatment of adult patients with previously treated FL or MZL in the US, Japan, and Brazil, and for FL in Europe[7-10]
- We now report updated long-term follow-up results from AUGMENT

AE, adverse event; CRR, complete response rate; FL, follicular lymphoma; iNHL, NHL, non-Hodgkin lymphoma; MZL, marginal zone lymphoma; NHL, non-Hodgkin lymphoma; ORR, overall response rate; R^2, lenalidomide and rituximab; R-placebo, rituximab and placebo; R/R, relapsed/refractory.

1. Teras LR, et al. *CA Cancer J Clin* 2016;66:443-459; 2. Dreyling M, et al. *Ann Oncol* 2013;24:857-877; 3. Ghielmini M, et al. *Ann Oncol* 2013;24:561-576; 4. Izutsu K. *J Clin Exp Hematop* 2014;54:31-37; 5. Leonard JP, et al. *J Clin Oncol* 2015;33:3635-3640; 6. Leonard JP, et al. *J Clin Oncol* 2019;37:1188-1199; 7. Revlimid® (lenalidomide) Medication guide. Princeton, NJ: Bristol Myers Squibb; 2022. 8. Japanese approval. 9. Brazil approval. 10. Revlimid® (lenalidomide) [summary of product characteristics]. Dublin, Ireland: Bristol Myers Squibb; 2021.

Leonard JP, et al. ASH 2022 [Abstract 230]

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Leonard JP, et al. ASH 2022 [Abstract 230]

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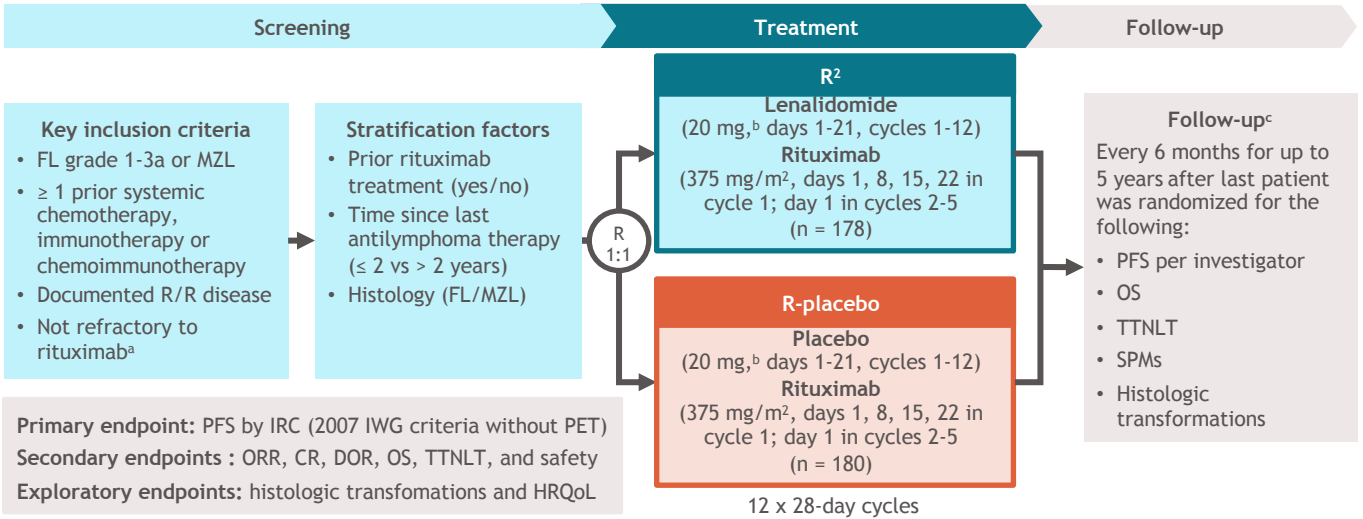
CRR, complete response rate; iNHL, indolent non-Hodgkin lymphoma; ORR, overall response rate; R-placebo, rituximab and placebo.

1. Leonard JP, et al. *J Clin Oncol* 2019;37:1188-1199; 2. Revlimid® (lenalidomide) Medication guide. Princeton, NJ: Bristol Myers Squibb; 2022; 3. Revlimid® (lenalidomide) [summary of product characteristics]. Dublin, Ireland: Bristol Myers Squibb; 2021.

Leonard JP, et al. ASH 2022 [Abstract 230]

AUGMENT study design

Multicenter, double-blind, randomized phase 3 study of R^2 versus R-placebo (NCT01938001)



Primary endpoint: PFS by IRC (2007 IWG criteria without PET)
Secondary endpoints : ORR, CR, DOR, OS, TTNLT, and safety
Exploratory endpoints: histologic transfomations and HRQoL

[a]Refractory was defined as < partial response to rituximab or rituximab-chemotherapy, or disease progression < 6 months after last rituximab dose; [b]20 mg if CrCl ≥ 60 mL/min, 10 mg if CrCl ≥ 30 to < 60 mL/min; [c]Included patients who discontinued treatment or withdrew from the study early for any reason without evidence of disease progression or relapse. CR, complete response; CrCl, creatinine clearance; DOR, duration of response; HRQoL, health-related quality of life; IRC, Independent Review Committee; IWG, international Working Group; OS, overall survival; PET, positron emission tomography; PFS, progression-free survival; R, randomized; SPM, second primary malignancy; TTNLT, time to next lymphoma treatment.

Leonard JP, et al. ASH 2022 [Abstract 230]

Conclusions

- After long-term follow-up (65.9 months), R^2 continues to demonstrate a superior efficacy over R-placebo as measured by the primary endpoint of PFS (per investigator)
- Fewer patients who received R^2 needed subsequent therapy to date, well beyond the 1-year treatment period
- The safety profile of R^2 and R-placebo remained consistent with the primary analysis,[1] with continued lower rates of SPM and histologic transformations compared with historical experience
- The OS Kaplan–Meier curve separation after 5 years continues to favor R^2, providing evidence for a survival benefit
  - The updated results for OS are consistent with the improvement observed in PFS
- These updated results, including OS data, further support the use of the R^2 regimen as a standard of care for patients with R/R iNHL

Leonard JP, et al. ASH 2022 [Abstract 230]

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Leonard JP, et al. ASH 2022 [Abstract 230]

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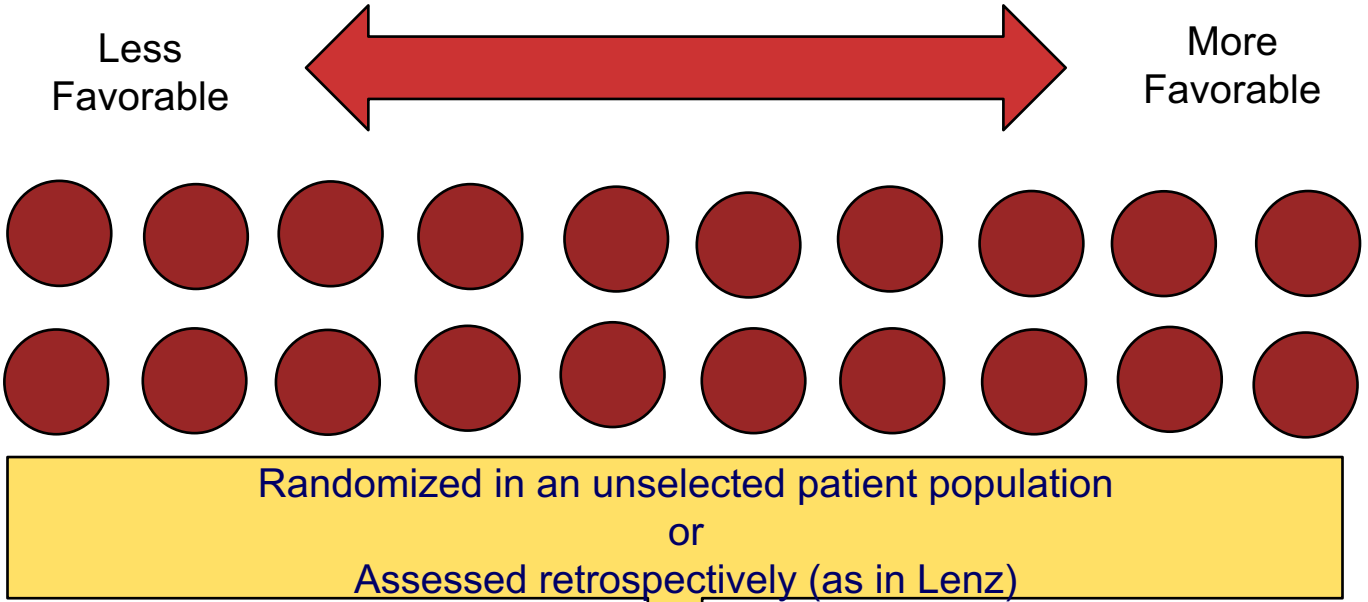
Leonard JP, et al. ASH 2022 [Abstract 230]

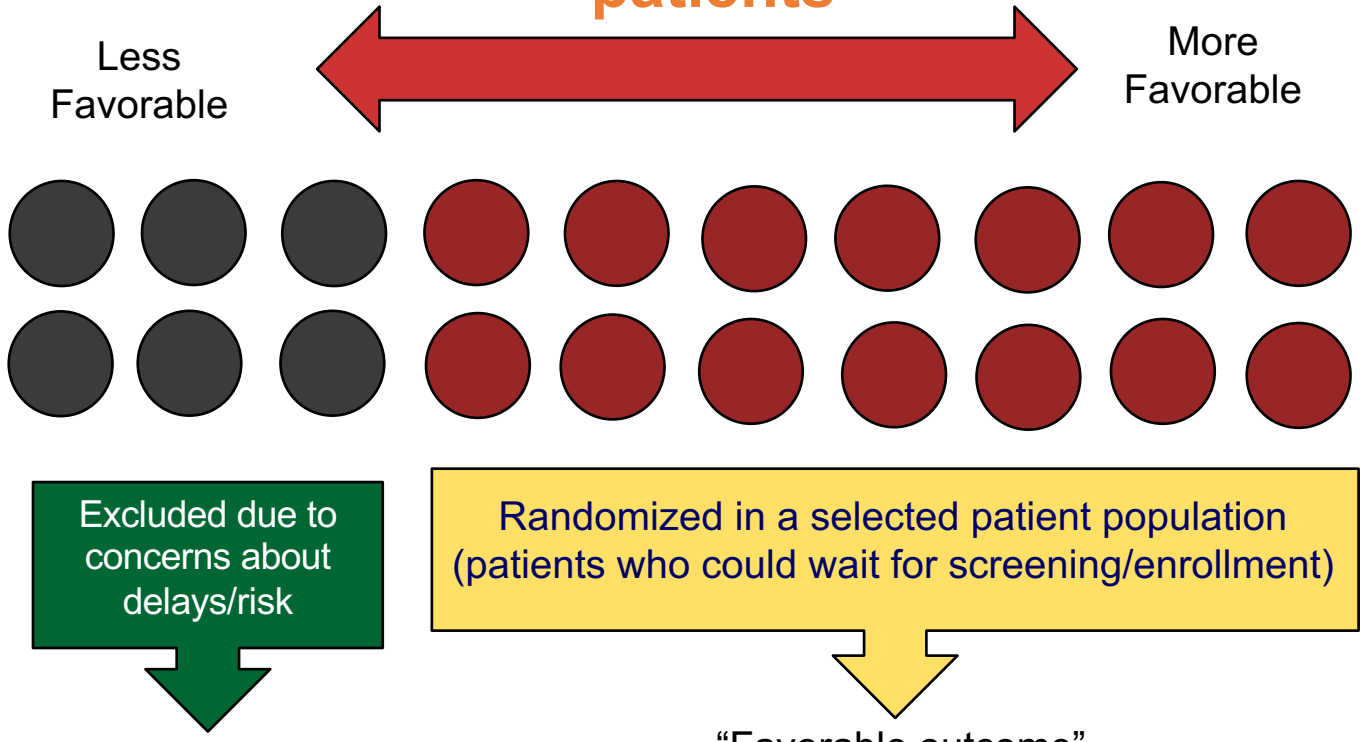
Acknowledgments

- The patients and families who are making the study possible
- The clinical study teams who participated
- The study was supported by Bristol Myers Squibb
- All authors contributed to and approved the presentation
- Writing and editorial assistance were provided by Joels Wilson-Nieuwenhuis, PhD, of Caudex, funded by Bristol Myers Squibb

Spectrum of ABC/Non-GCB DLBCL patients



Spectrum of ABC/Non-GCB DLBCL patients



Early descriptions of MCL (“mantle zone”)

Dennis Weisenburger
(“Mantle zone lymphoma”)
1982

Steven Swerdlow
(“Centrocytic lymphoma”)
1983

Stefano Pileri
(Mantle cell vs Marginal zone)
1985

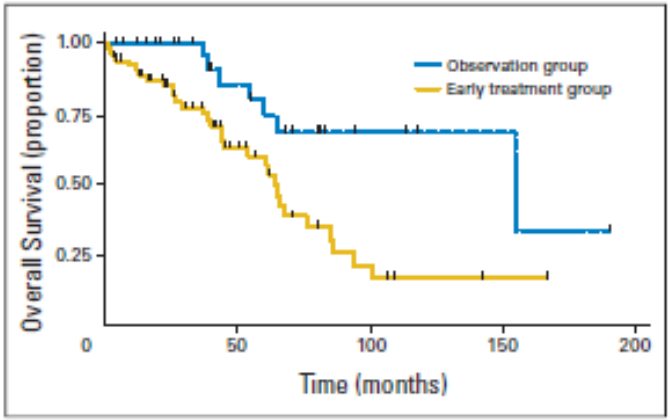
What are some of the key advances that have led to improvements in MCL options and outcomes?

Watch and wait is a reasonable approach in MCL

JOURNAL OF CLINICAL ONCOLOGY ORIGINAL REPORT

Outcome of Deferred Initial Therapy in Mantle-Cell Lymphoma

Peter Martin, Amy Chadburn, Paul Christos, Karen Weil, Richard R. Furman, Jia Ruan, Rebecca Elstrom, Ruben Niesvizky, Scott Ely, Maurizio DiLiberto, Ari Melnick, Daniel M. Knowles, Selina Chen-Kiang, Morton Coleman, and John P. Leonard



Martin, et al. *J Clin Oncol*. 2009

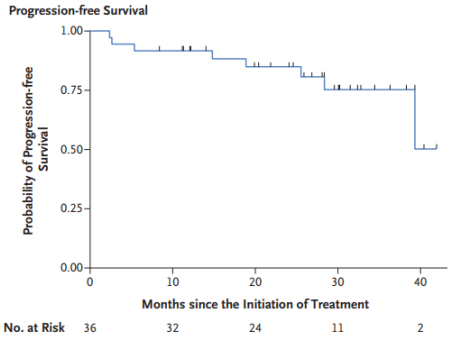
Chemotherapy is not necessary in MCL

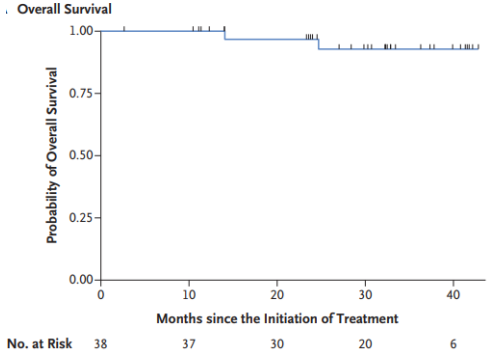
The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Lenalidomide plus Rituximab as Initial Treatment for Mantle-Cell Lymphoma

Jia Ruan, M.D., Ph.D., Peter Martin, M.D., Bijal Shah, M.D., Stephen J. Schuster, M.D., Sonali M. Smith, M.D., Richard R. Furman, M.D., Paul Christos, Dr.P.H., Amelyn Rodriguez, R.N., Jakub Svoboda, M.D., Jessica Lewis, P.A., Orel Katz, P.A., Morton Coleman, M.D., and John P. Leonard, M.D.





Ruan, et al. *N Engl J Med*. 2015

Many bright and dedicated researchers will continue to move MCL research forward

WCM/NYP Lymphoma Program Clinical/Translational Team

Is it better to pause or speak slowly, or use “um” and “uh”?

← Tweet

Adam Grant ✓
@AdamMGrant

It's a mistake to stop saying "um" and "uh" altogether.

Evidence: filler words signal that new information is coming, making it easier for listeners to understand and remember what comes next.

Hesitations don't make you sound weak. They help you... uh... communicate clearly.

Maybe better to say “This is a key point” or “If you remember one thing” or have a list of “Take-home messages”

Handling questions

- Train yourself to predict 5-10 questions and practice
- Add a “pitfalls” and “limitations of the study” slide to “vaccinate” yourself from tough questions
- “Thank you for your thoughtful question”
- “That is a great question”
- “We have thought of that and are working on it, that analysis is underway…planned…”
- I don’t know
- Answer the question you want to answer

Things you should do

- Take your time but be on time
- Make sure your main messages are clear
- Make sure the main message of each slide is clear
- Tell a story
- Acknowledge those who contributed to the work
- Acknowledge those who did work in the area before you
- Leave with some ideas about future questions