Subgroup analysis in RE-MIND2, an observational, retrospective cohort study of tafasitamab plus lenalidomide versus systemic therapies in patients with relapsed/refractory diffuse large B-cell lymphoma

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Background

- Diffuse large B-cell lymphoma (DLBCL) is the most common subtype of non-Hodgkin's lymphoma, accounting for up to 45% of cases[1]
- Recommended first-line treatment is with R-CHOP (rituximab, cyclophosphamide, doxorubicin, vincristine, and prednisone)[2]
  - R-CHOP is curative in 60–70% of patients, while 30–40% experience relapsed/refractory (R/R) disease after an initial response[3–4]
  - Five-year overall survival (OS) for patients with high-risk disease is 27–36%[5]
- Salvage therapy for patients with R/R disease comprises chemotherapy followed by high-dose chemotherapy and autologous stem cell transplant (ASCT);[2] 40–65% of patients who proceed to ASCT subsequently relapse[6,7]
  - Patients with primary refractory disease or who relapse <12 months post-R-CHOP may receive chimeric antigen receptor T-cell (CAR-T) therapy[2]
- In the single-arm, Phase II L-MIND study (NCT02399085), the immunotherapy tafasitamab + lenalidomide (LEN) demonstrated efficacy in ASCT-ineligible patients with R/R DLBCL[8,9]
- Based on the results from L-MIND, tafasitamab + LEN was granted accelerated approval in the United States (2020), conditional marketing authorization in the European Union and Canada (2021), and temporary approval in Switzerland (2022) for ASCT-ineligible patients with R/R DLBCL. The combination is a preferred treatment option in the NCCN Clinical Practice Guidelines in Oncology (NCCN Guidelines®) in this setting[2,10–13]
- In the primary analysis of the observational, retrospective cohort study RE-MIND2 (NCT04697160), efficacy outcomes of patients treated with tafasitamab + LEN in L-MIND were closely matched with cohorts of real-world patients who received bendamustine + rituximab (BR), rituximab + gemcitabine and oxaliplatin (R-GemOx), or systemic therapies for DLBCL pooled in one cohort (STP)
  - A significant difference in OS was observed with tafasitamab + LEN (31.6–34.1 months) versus STP (11.6 months; HR: 0.553; p=0.0068), BR (9.9 months; HR: 0.418; p<0.0001), and R-GemOx (11.0 months; HR: 0.467; p=0.0003)[14]
- A secondary analysis of RE-MIND2 compared the efficacy of tafasitamab + LEN with polatuzumab vedotin + BR (pola-BR), rituximab + LEN (R2), and CD19 CAR-T therapies[15]
  - While CAR-T therapy was recently approved in second-line DLBCL,[16] the current analysis is limited to its use in the previous indication, i.e. after two or more lines of previous systemic therapy
- Here, we examine OS in patients from L-MIND matched with the STP, pola-BR, R2, and CAR-T cohorts from RE-MIND2 in clinically relevant subgroups

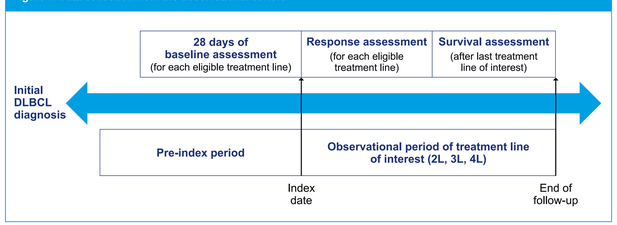
Objective

- To conduct hypothesis-generating analyses for clinically relevant patient subgroups to examine the relative effectiveness of tafasitamab + LEN versus selected systemic therapies for the treatment of ASCT-ineligible patients with high-risk R/R DLBCL

Patients and methods

- Data were collected from the electronic health records of patients diagnosed with DLBCL between 2010 and 2020 at academic hospitals, public hospitals and private practices in North America, Europe, and the Asia Pacific region
  - The analysis window for patients from L-MIND was defined as the interval between the index date and the data cut-off date (November 2019, approximately 2 years after the last patient was enrolled in RE-MIND2) (**Figure 1**)

Figure 1. Data collection from the observational cohort



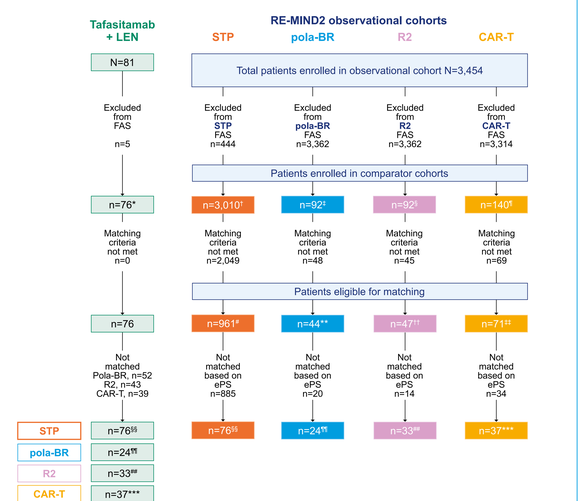
Patients who had received at least two prior therapy lines for DLBCL were assigned an index date (index date 2L, 3L, or 4L, i.e. second, third, or fourth line) for each eligible therapy line. Pre-index period: time between initial DLBCL diagnosis and index date of treatments (2L, 3L, or 4L). Index date: start of R/R DLBCL treatment (2L, 3L, or 4L). Observational period: time between index date and end of follow-up, including survival assessment. Baseline: 28 days of baseline assessment prior to index date.
DLBCL, diffuse large B-cell lymphoma; R/R, relapsed/refractory.

- Eligibility criteria were based on the L-MIND study: patients were aged ≥18 years with histologically confirmed DLBCL and had received at least two prior systemic therapies for R/R DLBCL (including ≥1 anti-CD20 therapy)[10]
- Matching criteria and an estimated propensity score (ePS)-based method were applied; efficacy outcomes from the L-MIND cohort were compared with patients treated with systemic regimens enrolled in RE-MIND2
- Separate matched analysis sets (MAS) were created for cohorts that received tafasitamab + LEN versus cohorts of STP, pola-BR, R2, and CAR-T
- The cohorts in each MAS were matched using an ePS-based 1:1 nearest neighbor (NN) method
  - The L-MIND and STP cohorts were balanced for nine baseline covariates: age (<70 vs ≥70 years), Ann Arbor stage (I/II vs III/IV), refractory to last therapy line (yes vs no), number of prior lines of therapy (1 vs 2/3), history of primary refractoriness (yes vs no), prior ASCT (yes vs no), elevated lactate dehydrogenase (LDH) (>upper limit of normal), neutropenia (cut-off <1.5 x 10^9/L), and anemia (cut-off <10 g/dL [6.21 mmol/L])
  - Six balancing covariates were used to compare the L-MIND and pola-BR, R2, and CAR-T cohorts (number/choice of covariates was driven by their clinical relevance and availability in patient records): number of prior lines of therapy (1 vs 2/3), refractory to last therapy (yes vs no), history of primary refractoriness (yes vs no), prior ASCT (yes vs no), age (<70 vs ≥70 years), and Eastern Cooperative Oncology Group performance status (ECOG PS) (0–1 vs ≥2)
  - To achieve a high quality of balance between cohorts, the absolute standardized difference of each covariate post-matching was pre-defined as ≤0.2
- The primary endpoint was OS
- To investigate the comparative effectiveness of the tafasitamab + LEN combination versus the comparator therapies for patients with high-risk disease, data in subgroups representative of risk factors from the International Prognostic Index for DLBCL[17] were examined
- Imbalances and high variability in the data for tafasitamab + LEN and the comparator therapies were detected in most subgroups; the number of extranodal sites (ENS) (0–1 vs ≥2) and elevated LDH (yes vs no) were determined to provide the most meaningful insights. OS was therefore assessed for these patient subgroups

Results

- In total, 3,454 patients were enrolled from 200 sites
- The 1:1 NN matching method resulted in strictly matched pairs of patients for tafasitamab + LEN versus STP (76 pairs), tafasitamab + LEN versus pola-BR (24 pairs), tafasitamab + LEN versus R2 (33 pairs), and tafasitamab + LEN versus CAR-T (37 pairs) (**Figure 2**, **Table 1A/B**)
- A high degree of covariate balance was achieved between the tafasitamab + LEN and comparator therapy cohorts (an absolute standardized difference of ≤0.2 for the balancing covariates in each MAS was achieved) (**Figure 3A/B**)
- Median duration of follow-up (months) in the matched cohorts was 31.8 versus 33.3 for tafasitamab + LEN versus STP, 31.8 versus 16.6 for tafasitamab + LEN versus pola-BR, 31.8 versus 13.4 for tafasitamab + LEN versus R2, and 31.6 versus 10.2 for tafasitamab + LEN versus CAR-T

Figure 2. Number of patients analyzed per MAS for systemic therapies pooled, pola-BR, R2, and CAR-T



*Included patients who met the eligibility criteria of RE-MIND2 and who received at least one dose of tafasitamab and one dose of LEN and had a minimum of 6 months' follow-up. †Included patients who met the eligibility criteria of RE-MIND2, received any systemic therapy for R/R DLBCL, and had a minimum of 6 months' follow-up. ‡Included patients who met the eligibility criteria of RE-MIND2, received pola-BR, and had a minimum of 6 months' follow-up. §Included patients who met the eligibility criteria of RE-MIND2, received R2, and had a minimum of 6 months' follow-up. ¶Included patients who met the eligibility criteria of RE-MIND2, received CAR-T, and had a minimum of 6 months' follow-up. #Included a subset of enrolled patients who received any systemic therapy for R/R DLBCL and were eligible for matching. **Included a subset of enrolled patients who received pola-BR and were eligible for matching. ††Included a subset of enrolled patients who received R2 and were eligible for matching. ‡‡Included a subset of enrolled patients who received CAR-T and were eligible for matching. §§Included 1:1 matched patients from the L-MIND study and those who received any systemic therapy for R/R DLBCL. ¶¶Included 1:1 matched patients from the L-MIND study and those who received pola-BR. ##Included 1:1 matched patients from the L-MIND study and those who received R2. ***Included 1:1 matched patients from the L-MIND study and those who received CAR-T. CAR-T, CD19 chimeric antigen receptor T-cell; ePS, estimated propensity score; FAS, full analysis set; LEN, lenalidomide; pola-BR, polatuzumab vedotin + bendamustine + rituximab; R2, rituximab + LEN; STP, systemic therapies pooled.

Table 1A. Demographics and baseline characteristics for the tafasitamab + LEN versus systemic therapies pooled matched analysis set

		Patient disposition	
		Tafasitamab + LEN (n=76)	STP (n=76)
Sex, n (%)	Female	36 (47.4)	32 (42.1)
	Male	40 (52.6)	44 (57.9)
Age at index date, years	Mean (SD)	69.1 (9.71)	68.7 (11.88)
	Median (Q1–Q3)	71.5 (62.0–76.0)	72.0 (60.0–77.0)
	Range, min–max	41–86	37–87
ECOG PS, n (%)	0	29 (38.2)	17 (22.4)
	1	41 (53.9)	27 (35.5)
	2	6 (7.9)	18 (23.7)
	3	0	3 (3.9)
	4	0	0
	Missing	0	11 (14.5)
Primary progressive disease, n (%)	Yes	2 (2.6)	5 (6.6)
	No	74 (97.4)	71 (93.4)
Number of extranodal sites, n (%)	0–1	52 (68.4)	38 (50.0)
	≥2	24 (31.6)	31 (40.8)
	Missing	0	7 (9.2)

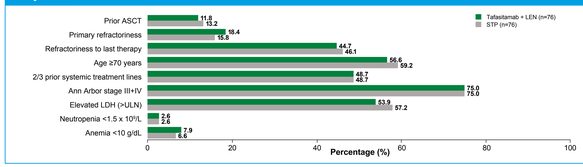
ECOG PS, Eastern Cooperative Oncology Group performance status; LEN, lenalidomide; Q1, lower quartile; Q3, upper quartile; SD, standard deviation; STP, systemic therapies pooled.

Table 1B. Demographics and baseline characteristics for the tafasitamab + LEN versus pola-BR, R2, and CAR-T matched analysis sets

		MAS for pola-BR		MAS for R2		MAS for CAR-T	
		Tafasitamab + LEN (n=24)	pola-BR (n=24)	Tafasitamab + LEN (n=33)	R2 (n=33)	Tafasitamab + LEN (n=37)	CAR-T (n=37)
Sex, n (%)	Female	10 (41.7)	10 (41.7)	17 (51.5)	11 (33.3)	18 (48.6)	17 (45.9)
	Male	14 (58.3)	14 (58.3)	16 (48.5)	22 (66.7)	19 (51.4)	20 (54.1)
Age at index date, years	Mean (SD)	72.3 (8.19)	73.7 (13.66)	67.9 (10.54)	69.9 (11.80)	65.8 (10.79)	63.7 (11.45)
	Median (Q1–Q3)	73.0 (69.5–78.5)	78.5 (69.5–81.0)	72.0 (58.0–75.0)	69.0 (63.0–78.0)	68.0 (58.0–75.0)	64.0 (57.0–70.0)
	Range, min–max	55–86	30–91	47–82	31–91	41–82	30–92
Neutropenia (cut-off <1.5 x 10^9/L), n (%)	Yes	0	3 (12.5)	0	4 (12.1)	0	1 (2.7)
	No	24 (100)	17 (70.8)	33 (100)	28 (84.8)	37 (100)	36 (97.3)
	Missing	0	4 (16.7)	0	1 (3.0)	0	0
Anemia (cut-off hemoglobin <10 g/dL), n (%)	Yes	1 (4.2)	6 (25.0)	2 (6.1)	6 (18.2)	5 (13.5)	8 (21.6)
	No	23 (95.8)	17 (70.8)	31 (93.9)	26 (78.8)	32 (86.5)	29 (78.4)
	Missing	0	1 (4.2)	0	1 (3.0)	0	0
Elevated LDH (>ULN), n (%)	Yes	14 (58.3)	18 (75.0)	20 (60.6)	22 (66.7)	19 (51.4)	21 (56.8)
	No	10 (41.7)	4 (16.7)	13 (39.4)	8 (24.2)	18 (48.6)	15 (40.5)
	Missing	0	2 (8.3)	0	3 (9.1)	0	1 (2.7)
Primary progressive disease, n (%)	Yes	1 (4.2)	3 (12.5)	2 (6.1)	8 (24.2)	2 (5.4)	6 (16.2)
	No	23 (95.8)	21 (87.5)	31 (93.9)	25 (75.8)	35 (94.6)	31 (83.8)
Ann Arbor stage, n (%)	I+II	3 (12.5)	4 (16.7)	8 (24.2)	2 (6.1)	6 (16.2)	8 (21.6)
	III+IV	21 (87.5)	14 (58.3)	25 (75.8)	17 (51.5)	31 (83.8)	18 (48.6)
	Missing	0	6 (25.0)	0	14 (42.4)	0	11 (29.7)
Number of extranodal sites, n (%)	0–1	18 (75.0)	11 (45.8)	20 (60.6)	17 (51.5)	23 (62.2)	23 (62.2)
	≥2	6 (25.0)	12 (50.0)	13 (39.4)	13 (39.4)	14 (37.8)	14 (37.8)
	Missing	0	1 (4.2)	0	3 (9.1)	0	0

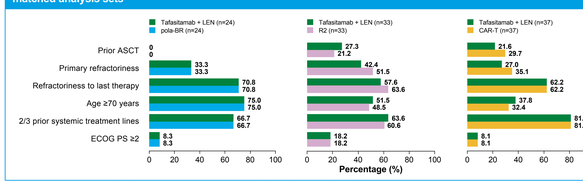
CAR-T, CD19 chimeric antigen receptor T-cell; LEN, lenalidomide; LDH, lactate dehydrogenase; MAS, matched analysis set; pola-BR, polatuzumab vedotin + bendamustine + rituximab; R2, rituximab + lenalidomide; Q1, lower quartile; Q3, upper quartile; SD, standard deviation; ULN, upper limit of normal.

Figure 3A. Cohort balancing covariates from the tafasitamab + LEN versus systemic therapies pooled matched analysis set



ASCT, autologous stem cell transplant; LDH, lactate dehydrogenase; LEN, lenalidomide; STP, systemic therapies pooled; ULN, upper limit of normal.

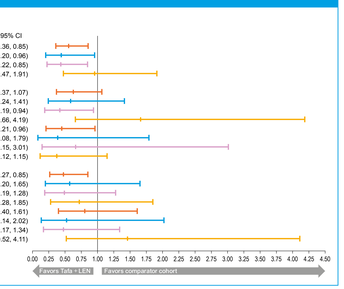
Figure 3B. Cohort balancing covariates from the tafasitamab + LEN versus pola-BR, versus R2, and versus CAR-T matched analysis sets



ASCT, autologous stem cell transplant; CAR-T, CD19 chimeric antigen receptor T-cell; ECOG PS, Eastern Cooperative Oncology Group performance status; LEN, lenalidomide; pola-BR, polatuzumab vedotin + bendamustine + rituximab; R2, rituximab + LEN.

- Median OS and hazard ratios for OS indicated a trend toward favoring tafasitamab + LEN in each MAS and in patient subgroups across most MASs (**Figure 4**)
- The analyses did not show or suggest a clear difference in the relative treatment effect of tafasitamab + LEN versus comparator therapies according to number of ENS or elevated LDH

Figure 4. Analyses of OS for subgroups for tafasitamab + LEN versus systemic therapies pooled, pola-BR, R2, and CAR-T



*Number of patients in the tafasitamab + lenalidomide and observational cohorts, respectively. †HR estimated using Cox proportional hazard model with observational cohort as reference.
CAR-T, CD19 chimeric antigen receptor T-cell; CI, confidence interval; E/E, number of events in the tafasitamab + LEN cohort/the comparator cohort; HR, hazard ratio; LDH, lactate dehydrogenase; LEN, lenalidomide; NN, number of patients in the tafasitamab + LEN cohort/the comparator cohort; NR, not reached; OS, overall survival; pola-BR, polatuzumab vedotin + bendamustine + rituximab; R2, rituximab + lenalidomide; STP, systemic therapies pooled; tafa, tafasitamab.

Conclusions

- In each subgroup, there was a trend favoring enhanced OS with tafasitamab + LEN when compared with STP, R2, and pola-BR, indicating the combination may improve OS in patients with high- and lower-risk R/R DLBCL versus other therapies in the setting
- The differences in OS duration observed with CAR-T versus tafasitamab + LEN warrant further investigation
- The analyses between tafasitamab + LEN and each comparator therapy were not powered for statistical comparison. Small sample sizes result in wide confidence intervals; therefore, results must be interpreted with caution but warrant further evidence generation within high-risk patient populations
- However, despite the limitations of these analyses and the small sample sizes, these results may help contextualize therapeutic options for treating high-risk patients with R/R DLBCL

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Disclosures

About Tafasitamab
Tafasitamab is a humanized, Fc-modified, cytolytic CD19-targeting monoclonal antibody. In 2010, MorphoSys licensed exclusive worldwide rights to develop and commercialize tafasitamab from Xencor, Inc. Tafasitamab incorporates an XmAb® engineered Fc domain, which mediates B-cell lysis through apoptosis and immune effector mechanisms, including antibody-dependent cell-mediated cytotoxicity (ADCC) and antibody-dependent cellular phagocytosis (ADCP). In January 2020, MorphoSys and Incyte entered into a collaboration and licensing agreement to further develop and commercialize tafasitamab globally. Following accelerated approval by the U.S. Food and Drug Administration in July 2020, tafasitamab is being co-commercialized by MorphoSys and Incyte in the United States. Conditional/accelerated approvals were granted by the European Medicines Agency and other regulatory authorities. Incyte has exclusive commercialization rights outside the United States. XmAb® is a registered trademark of Xencor, Inc.

Conflicts of interest
GSN: Consultancy or advisory role: Celgene, MorphoSys, Genentech, Selvita, Debiopharm Group, Kite/Gilead, TG Therapeutics, Kymera, Karyopharm Therapeutics, Ryvu Therapeutics, Bantham; research funding: Celgene, NanoString Technologies, MorphoSys. **DHY:** Consulting or advisory: Roche, Janssen, Amgen, Celgene, GC Pharma, Novartis, Abclone; honoraria: Celtrion, Roche, Janssen, Amgen, Celgene, Samyang, Kirin Pharmaceuticals, Takeda; research funding: Samyang, Abclone, Roche/Genentech, Janssen Oncology, Genmab, Boryung, Eutilex. **EJ:** Consultancy: AstraZeneca, Epizyme. **PLZ:** Consultancy: Celltrion, Gilead Sciences, Janssen-Cilag, Bristol-Myers Squibb, SERVIER, Sandoz, MSD, Roche, EUSA Pharma, Kyowa Kirin, Takeda, Secura BIO, TG Therapeutics, Novartis, ADC Therapeutics, Incyte, BeiGene; speakers' bureau: MSD, EUSA Pharma, Novartis. **LS:** Employee and stockholder of Incyte Biosciences International Sarl. **EEW:** Employment: MorphoSys AG; research funding: MorphoSys AG. **CGA:** Employment: MorphoSys US Inc; stock or other ownership: MorphoSys US Inc; research funding: MorphoSys US Inc; travel accommodations, expenses: MorphoSys US Inc. **GH:** Honoraria: AbbVie, AstraZeneca, BeiGene, EUSA, Genmab, Gilead, Incyte, Janssen, Novartis, Roche; consulting or advisory role: AbbVie, AstraZeneca, BeiGene, EUSA, Genmab, Gilead, Incyte, Janssen, Novartis, Roche; research funding: AbbVie, Celgene, Gilead/Kite, Incyte, Janssen, Roche; travel, accommodations, expenses: Janssen. **PR:** Consultancy: Kite, a Gilead Company, Novartis, Bristol-Myers Squibb, Takeda, BeiGene, Karyopharm Therapeutics, Verastem, Bayer; speakers' bureau: Kite, a Gilead Company, Bayer; honoraria: Novartis; research funding: Novartis, Bristol-Myers Squibb/Celgene, MorphoSys AG, Kite, a Gilead Company, Calibr. **KK:** Research funding: MorphoSys AG, AstraZeneca, Grail, Pacira Pharmaceuticals, Myriad Genetics. **DB:** Consulting or advisory role: Millicrek Outcomes Group, Sage, Haymarket, Otsuka, Value Demonstration, LLC. **GS:** Consultancy: Roche/Genentech, Gilead Sciences, Janssen, Celgene, Novartis, MorphoSys AG, Epizyme, Alimera Sciences, Genmab, Debiopharm Group, Velosbio, Bristol-Myers Squibb, BeiGene, Incyte, Miltenyi Biotec, Ipsen; honoraria: Roche/Genentech, Janssen, Celgene, Gilead Sciences, Novartis, AbbVie, MorphoSys AG.

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