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

Grzegorz S. Nowakowski,[1] Dok Hyun Yoon,[2] Erel Joffe,[3] Pier Luigi Zinzani,[4,5] Lorenzo Sabatelli,[6] Eva E. Waltl,[7] Carmelita G. Alvero,[8] Georg Hess,[9] Peter A. Riedell,[10] Kibum Kim,[11,12] Diana Brixner,[11] Gilles Salles[3]

[1]Division of Hematology, Mayo Clinic, Rochester, MN, USA; [2]Department of Oncology, Asan Medical Center, University of Ulsan College of Medicine, Seoul, South Korea; [3]Department of Medicine, Memorial Sloan Kettering Cancer Center, New York, NY, USA; [4]IRCCS Azienda Ospedaliero-Universitaria di Bologna Istituto di Ematologia "Seràgnoli"; [5]Dipartimento di Medicina Specialistica, Diagnostica e Sperimentale Università di Bologna, Bologna, Italy; [6]Incyte Biosciences International Sàrl, Morges, Switzerland; [7]MorphoSys AG, Planegg, Germany; [8]MorphoSys US Inc, Boston, MA, USA; [9]Department of Hematology, Oncology and Pneumology, University Medical Center, Johannes Gutenberg-University Mainz, Germany; [10]Division of Hematology and Oncology, University of Chicago Medicine, Chicago, IL, USA; [11]University of Utah, Salt Lake City, UT, USA; [12]University of Illinois at Chicago, Chicago, IL, USA

Abstract: 7560. Session: Hematologic Malignancies—Lymphoma and Chronic Lymphocytic Leukemia

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 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 regimen in National Comprehensive Cancer Network 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)
  - Significantly prolonged OS was reported with tafasitamab + LEN (31.6–34.1 months) versus STP (11.6 months), BR (9.9 months), and R-GemOx (11.0 months)[14]
- A secondary analysis of RE-MIND2 compared the efficacy of tafasitamab + LEN with polatuzumab vedotin + BR (pola-BR), rituximab + LEN (R2), and CD19 chimeric antigen receptor T-cell (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

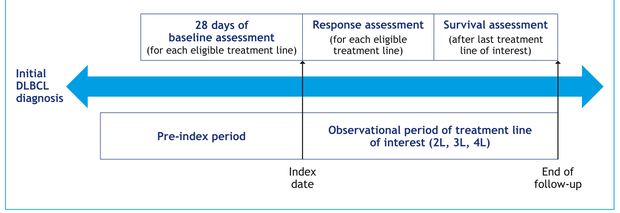
Objective

- 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



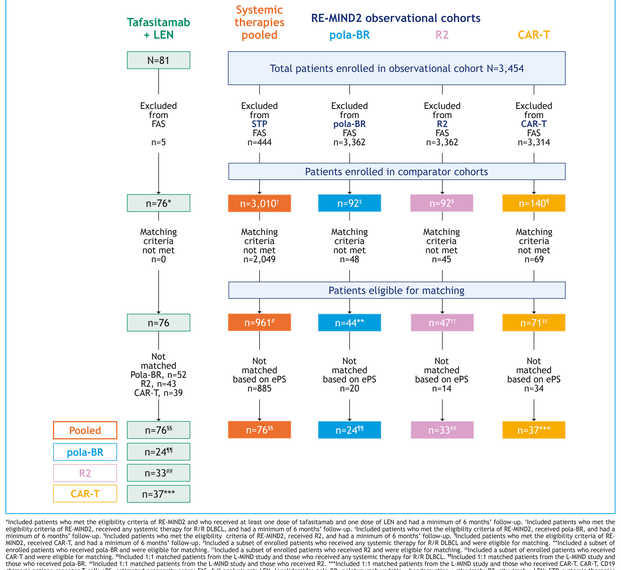
Patients who had received at least two prior therapy lines for DLBCL were assigned an index date (index date 2L, 3L, or 4L, i.e., second, third or fourth line) for each eligible therapy line. Pre-index period: time between initial DLBCL diagnosis and index date of treatments (2L, 3L, or 4L). Index date: 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; L, line; 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 (>upper limit of normal), neutropenia (cut-off <1.5 × 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 lactate dehydrogenase (LDH) (yes vs no) were determined to provide most meaningful insight. 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 therapy; 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)

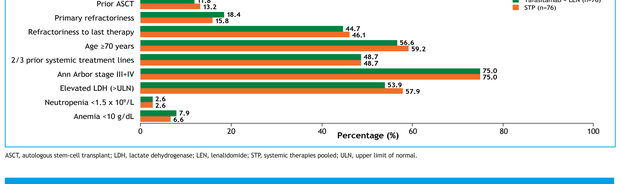
ECOG PS, Eastern Cooperative Oncology Group performance status; LEN, lenalidomide; Q1, lower quartile; Q3, upper quartile; SD, standard deviation; STP, systemic therapies for DLBCL pooled in one cohort.

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 (0.0)	4 (16.7)	0 (0.0)	1 (3.0)	0 (0.0)	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

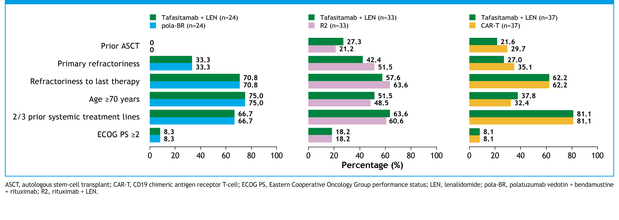
CAR-T, CD19 chimeric antigen receptor T-cell therapy; LEN, lenalidomide; LDH, lactate dehydrogenase; MAS, matched analysis set; pola-BR, polatuzumab vedotin + bendamustine + rituximab; R2, rituximab + lenalidomide; Q1, lower quartile; Q3, upper quartile; SD, standard deviation; ULN, upper limit of normal.

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 MAS (**Table 2**)
- 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

Table 2. Analyses of OS for subgroups for tafasitamab + LEN versus systemic therapies pooled, pola-BR, R2, and CAR-T

		Tafa + LEN vs STP	Tafa + LEN vs pola-BR	Tafa + LEN vs R2	Tafa + LEN vs CAR-T
		N/N* Median (95% CI) HR (95% CI)	N/N* Median (95% CI) HR (95% CI)	N/N* Median (95% CI) HR (95% CI)	N/N* Median (95% CI) HR (95% CI)
Overall		76/76	24/24	33/33	37/37
		34.1 (18.3–NR) **11.6** (8.8–16.1)	**20.1** (8.6–NR) **7.2** (4.9–11.6)	**24.6** (12.1–NR) **7.4** (4.2–11.1)	**22.5** (8.6–NR) **15.0** (10.1–NR)
		0.553 (0.358–0.855) p=0.0068†	**0.441** (0.203–0.956) p=0.0340†	**0.435** (0.224–0.847) p=0.0122†	**0.953** (0.475–1.913) p=0.8929†
Addressed					
Number of extranoal sites	0–1	52/38	18/11	20/17	23/23
		NR (19.3–NR) **14.5** (10.0–30.8)	**24.8** (8.6–NR) **8.5** (4.9–32.0)	**31.6** (8.6–NR) **9.5** (3.4–NR)	**31.6** (12.4–NR) **27.3** (4.6–NR)
		0.476 (0.27–0.85)	**0.573** (0.20–1.65)	**0.491** (0.19–1.28)	**0.717** (0.28–1.85)
	≥2	24/31	6/12	13/13	14/14
		14.8 (6.1–NR) **9.4** (5.2–42.9)	**7.6** (0.8–NR) **6.1** (2.5–7.4)	**24.6** (1.3–NR) **6.2** (4.2–11.1)	**7.6** (1.3–26.4) **14.6** (9.9–NR)
		0.803 (0.40–1.61)	**0.524** (0.14–2.02)	**0.478** (0.17–1.34)	**1.459** (0.52–4.11)
Elevated LDH	No	35/32	10/4	13/8	18/15
		NR (31.6–NR) **21.7** (11.0–NR)	**NR** (1.7–NR) **8.5** (4.6–32.0)	**NR** (6.6–NR) **NR** (6.9–NR)	**NR** (13.1–NR) **14.6** (9.9–27.3)
		0.448 (0.21–0.96)	**0.388** (0.08–1.79)	**0.664** (0.15–3.01)	**0.371** (0.12–1.15)
	Yes	41/44	14/18	20/22	19/21
		18.3 (9.4–34.1) **8.3** (5.3–11.8)	**11.6** (1.9–NR) **6.7** (4.9–11.6)	**13.8** (2.7–NR) **5.2** (3.3–7.9)	**8.6** (2.7–26.4) **15.9** (4.1–NR)
		0.627 (0.37–1.07)	**0.585** (0.24–1.41)	**0.420** (0.19–0.94)	**1.663** (0.66–4.19)

*Number of patients in the tafasitamab + lenalidomide and observational cohorts, respectively. †Log-rank test. HR estimated using Cox proportional hazard model with observational cohort as reference.
CAR-T, CD19 chimeric antigen receptor T-cell therapy; CI, confidence interval; HR, hazard ratio; LDH, lactate dehydrogenase; LEN, lenalidomide; 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 small sample size, these results may help contextualize therapeutic options for treating high-risk patients with R/R DLBCL

Acknowledgments

This study was funded by MorphoSys AG. Medical writing assistance was provided by Eoin Duffy, PhD, CMPP of Syneos Health, UK, and funded by MorphoSys AG.

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. Incyte has exclusive commercialization rights outside the United States. XmAb® is a trademark of Xencor, Inc.

Conflicts of interest
GSN: Consultancy or advisory role: Celgene, MorphoSys, Genentech, Selvita, Bebiopharm 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: Celltrion, 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; 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: Millcreek Outcomes Group, Sage, Haymarket, Otsuka, Value Demonstration, LLC. **GS:** Consultancy: Roche/Genentech, Gilead Sciences, Janssen, Celgene, Novartis, MorphoSys AG, Epizyme, Alimera Sciences, Genmab, Bebiopharm Group, Velosbio, Bristol-Myers Squibb, BeiGene, Incyte, Miltenyi Biotec, Ipsen; honoraria: Roche/Genentech, Janssen, Celgene, Gilead Sciences, Novartis, AbbVie, MorphoSys AG.

References

1. WHO. World Cancer Report: Cancer Research for Cancer Prevention. IARC Press; 2020.
2. NCCN Clinical Practice Guidelines: B-Cell Lymphomas V3.2022.
3. Coiffier B, Sarkozy C. Hematol Am Soc Hematol Educ Progr 2016;2016(1):366–78.
4. Sarkozy C, Sehn LH. Ann Lymphoma 2019;3:10.
5. Johnson NA, et al. J Clin Oncol 2012;30(28):3452–9.
6. González-Barca E, et al. Bone Marrow Transplant 2020;55(2):393–9.
7. Chihara D, et al. Biol Blood Marrow Transplant 2014;20(5):684–9.
8. Salles G, et al. Lancet Oncol 2020;21(7):978–88.
9. Duell J, et al. Haematologica 2021;106(9):2417–26.
10. MONJUVI Prescribing information. Boston, MA: MorphoSys.https://www.monjuvi.com/pi/monjuvi-pi.pdf [Accessed April 2022].
11. Health Canada. Minjuvi. https://healthproducts.canada.ca/dpdbdpp/info do?lang=en&code=100793 [Accessed April 2022].
12. European Medicines Agency. Minjuvi. https://www.ema.europa.eu/en/medicines/human/EPAR/minjuvi [Accessed April 2022].
13. Swiss Agency for Therapeutic Products. https://www.swissmedic.ch/swissmedic/en/home/humanarzneimittel/authorisations/new-medicines/minjuvitm-pulver-tafasitamabum.html [Accessed April 2022].
14. Nowakowski GS, et al. Presented at SOHO. September 2021. Poster ABCL-346.
15. Nowakowski GS, et al. Blood 2021;138(Suppl 1):183.
16. FDA. https://www.fda.gov/drugs/resources-information-approved-drugs/fdaapproves-axicabtagene-ciloleucel-second-line-treatment-large-b-cell-lymphoma [Accessed April 2022].
17. The International Non-Hodgkin's Lymphoma Prognostic Factors Project. N Engl J Med 1993;329(14):987–94.

Correspondence: Prof. MD Grzegorz S. Nowakowski (Nowakowski.Grzegorz@mayo.edu)