Poster
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Association of Pretreatment Tumor Characteristics and Clinical Outcomes Following Second-Line Axicabtagene Ciloleucel Versus Standard of Care in Patients With Relapsed/Refractory Large B-Cell Lymphoma

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BACKGROUND

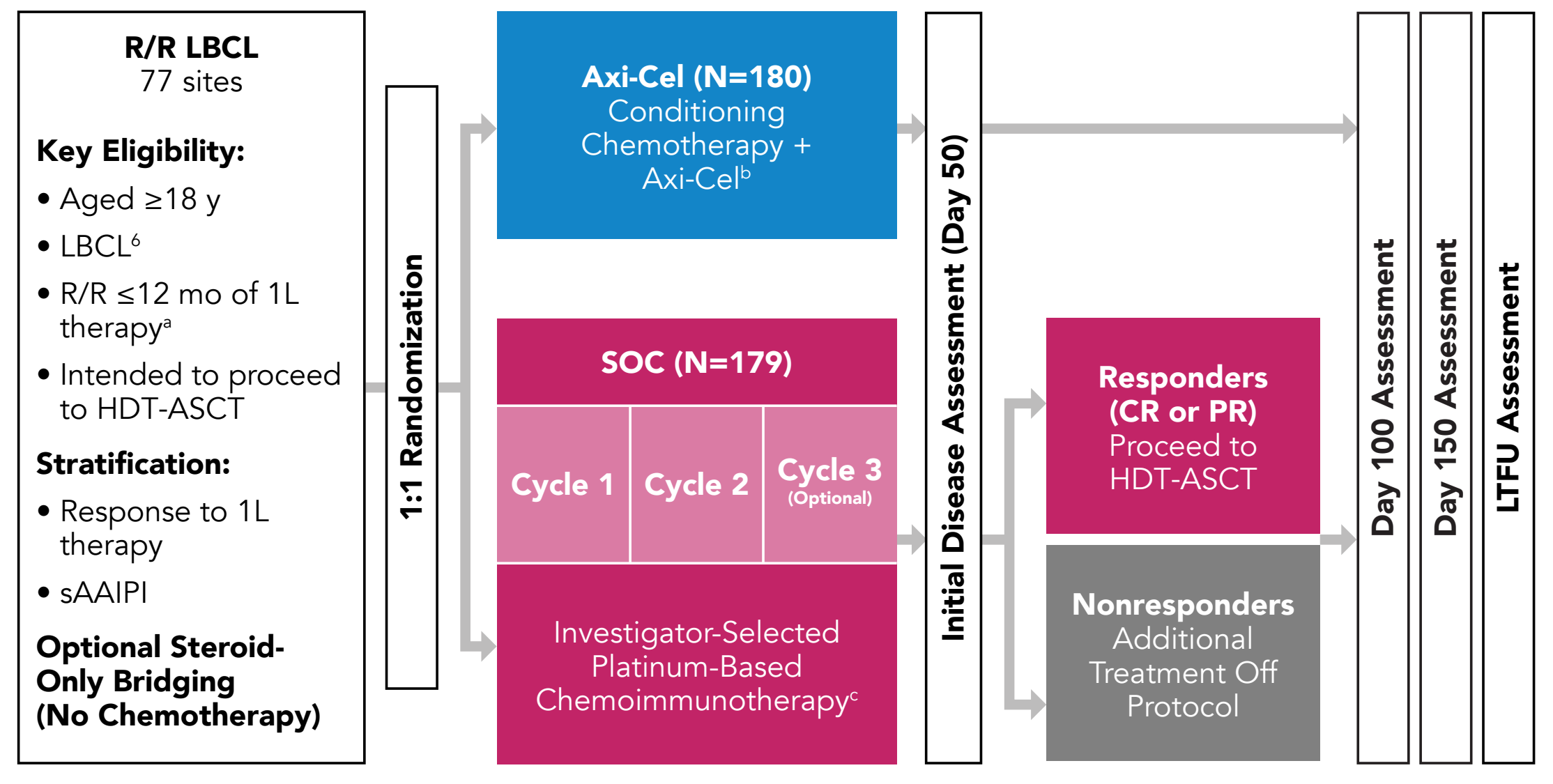
- Axicabtagene ciloleucel (axi-cel) is an autologous anti-CD19 chimeric antigen receptor (CAR) T-cell therapy approved for the treatment of adult patients with relapsed/refractory (R/R) large B-cell lymphoma (LBCL) after ≥2 lines of systemic therapy and most recently, in the United States, for R/R LBCL after first-line chemoimmunotherapy[1]
- In the Phase 3 randomized ZUMA-7 (NCT03391466) in second-line (2L) R/R LBCL[2]:
  - Axi-cel showed superiority to standard of care (SOC; salvage chemotherapy and high-dose chemotherapy with autologous stem cell transplantation [HDT-ASCT]) in event-free survival (EFS; hazard ratio, 0.398, *P*<.0001; median 8.3 vs 2 months, respectively; 24-month EFS rate: 41% vs 16%, respectively; 24.9-month median follow-up)
  - Axi-cel had a manageable safety profile that was consistent with that observed in the ZUMA-1 study of axi-cel in patients with refractory LBCL[3,4]
- In ZUMA-1, the strongest correlate of durable response was peak CAR T-cell levels normalized to pretreatment tumor burden[5]

OBJECTIVE

- To report results of exploratory analyses of tumor characteristics, including pretreatment tumor burden, tissue hypoxia-related lactate dehydrogenase (LDH) level, tumor gene expression signatures, and CD19 expression in ZUMA-7

METHODS

Figure 1. ZUMA-7 Study Schema and Endpoints



Primary Endpoint
- EFS[d] by blinded central review

Key Secondary Endpoints
- ORR
- OS

Secondary Endpoints
- PFS
- Safety
- PROs

No Protocol-Specified Crossover

[a] Refractory disease was defined as no CR to 1L therapy; relapsed disease was defined as CR followed by biopsy-proven disease relapse ≤12 months from completion of 1L therapy. [b] Axi-cel patients underwent leukapheresis followed by conditioning chemotherapy with cyclophosphamide (500 mg/m^2/day) and fludarabine (30 mg/m^2/day) 5, 4, and 3 days before receiving a single axi-cel infusion (target intravenous dose, 2×10^6 CAR T cells/kg). [c] Protocol-defined SOC regimens included R-GDP, R-DHAP, R-ICE, or R-ESHAP. [d] EFS was defined as time from randomization to the earliest date of disease progression per Lugano Classification,[7] commencement of new lymphoma therapy, or death from any cause.

1L, first-line; axi-cel, axicabtagene ciloleucel; CAR, chimeric antigen receptor; CR, complete response; EFS, event-free survival; HDT-ASCT, high-dose chemoimmunotherapy and autologous stem cell transplantation; LBCL, large B-cell lymphoma; LTFU, long-term follow-up; mo, month; ORR, objective response rate; OS, overall survival; PFS, progression-free survival; PR, partial response; PRO, patient-reported outcome; R-DHAP, rituximab, dexamethasone, cytarabine, and cisplatin; R-ESHAP, rituximab, etoposide, methylprednisolone, cytarabine, and cisplatin; R-GDP, rituximab, gemcitabine, cisplatin, and dexamethasone; R-ICE, rituximab, ifosfamide, carboplatin, and etoposide phosphate; R/R, relapsed/refractory; sAAIPI, second-line age-adjusted International Prognostic Index; SOC, standard of care; y, year.

- Tumor burden was calculated as the sum of product diameters (SPD) of ≤6 reference lesions[5]
- Serum LDH was assessed per local laboratory
- Pretreatment tumor samples were assessed for gene expression by the NanoString IO 360™ panel and for prespecified immune contexture signatures related to T-cell function and trafficking (Immunosign 15 [IS15] and 21 [IS21][8])
- ZUMA-1 Cohorts 1 and 2 data were used for comparison to third-line R/R LBCL
- CD19 protein expression was assessed by immunohistochemistry (H-score)
- Associations between biomarkers and clinical outcomes were assessed using descriptive statistics (*P*<.05 was considered significant)
- EFS was defined as time from randomization to the earliest date of disease progression per Lugano Classification,[7] commencement of new lymphoma therapy, or death from any cause
- Response definitions were defined according to response at time of data cutoff (primary analysis) and were as follows:
  - Ongoing responders: patients who achieved a complete or partial response and remained in response
  - Relapsed: patients who achieved a complete response (CR) or partial response and subsequently experienced disease progression
  - Nonresponders: patients who experienced stable or progressive disease as best response

RESULTS

Table 1. Baseline Tumor Characteristics

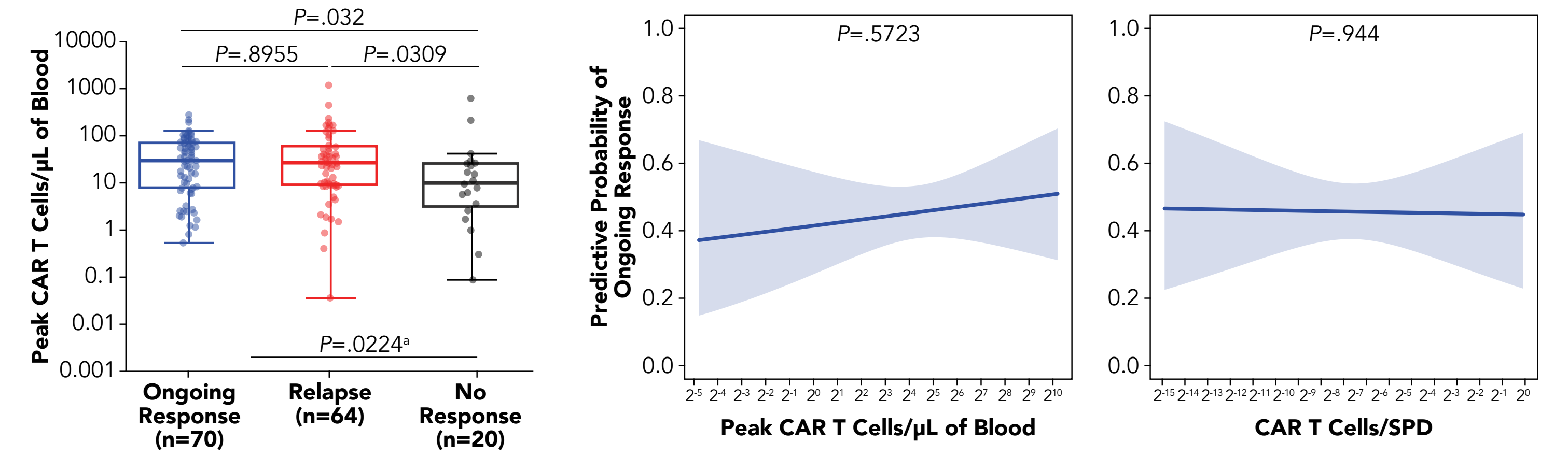
Characteristic	ZUMA-7 Axi-Cel N=170	ZUMA-7 SOC N=168	ZUMA-7 Overall N=338	ZUMA-1[a] Cohorts 1+2 N=101
Elevated LDH level, n (%)[b]	92 (54)	90 (54)	182 (54)	62 (61)
LDH ≥2× ULN, n (%)[b]	43 (25)	36 (21)	79 (23)	45 (45)
Median tumor burden[c] (Q1-Q3), [range], mm²	2118 (981-4368) [181-22,538]	2069 (926-4881) [252-20,117]	2115 (942-4755) [181-22,538]	3723 (2200-7138) [171-23,297]
Median CD19 H-score (range)[d]	140 (0-300)	160 (0-280)	150 (0-300)	210 (0-300)

[a] ZUMA-1 baseline tumor characteristics are shown for reference purposes. [b] LDH level greater than ULN per local laboratory reference range. [c] As determined by the sum of product diameters of ≤6 reference lesions.[5] [d] CD19 staining was not required for participation in the trial. Testing was retrospectively conducted per central laboratory. Numbers of patients included in median CD19 H-score were 170 in the axi-cel arm, 168 in the SOC arm, and 338 overall.

Axi-cel, axicabtagene ciloleucel; LDH, lactate dehydrogenase; Q, quartile; SOC, standard of care; ULN, upper limit of normal.

- Baseline tumor characteristics were generally balanced between axi-cel and SOC patients (**Table 1**)

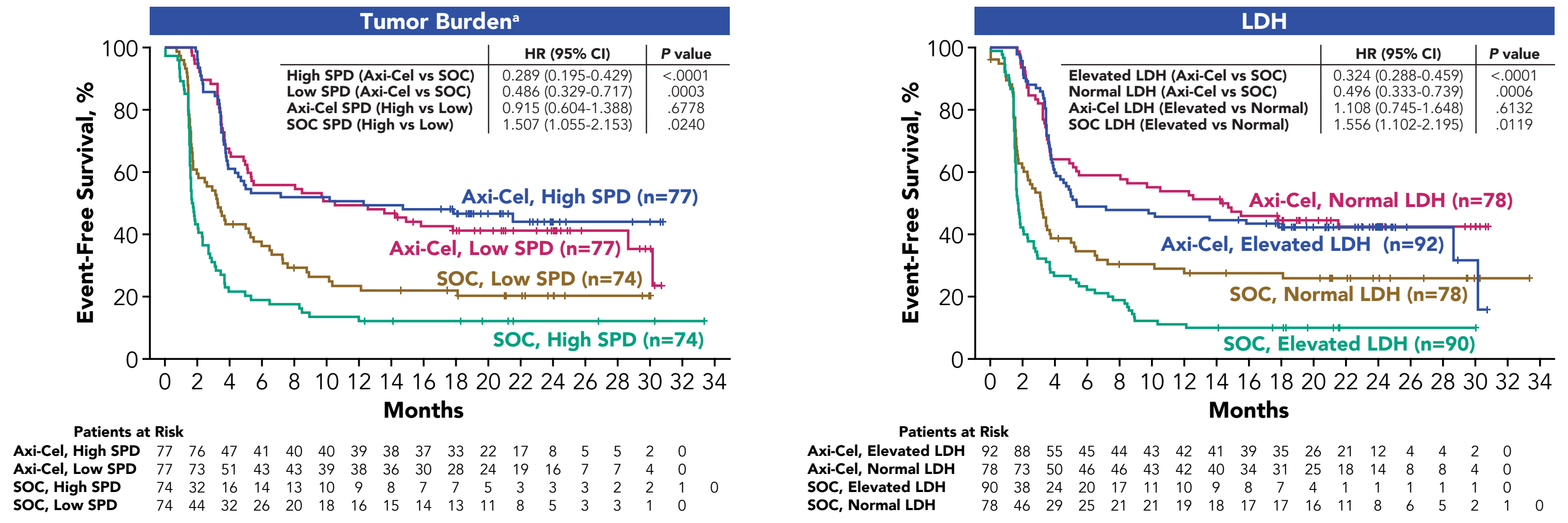
Figure 2. CAR T-Cell Expansion Was Associated With Response, But Not Ongoing Response, in ZUMA-7



P values calculated by Wilcoxon Rank-Sum test. [a] Shows *P* value comparing combined ongoing response and relapse versus no response.
CAR, chimeric antigen receptor; SPD, sum of product diameters.

- CAR T-cell expansion (peak) was significantly lower in patients who did not respond compared with patients in ongoing response or who relapsed (*P*<.05; **Figure 2, left**)
- There was no association between ongoing responses and CAR T-cell peak (**Figure 2**, **middle**) or CAR T-cell peak normalized to tumor burden (**Figure 2**, **right**)
- Additionally, peak CAR T-cell expansion was comparable between patients with high and low CD19 H-score (above vs below median CD19 H-score, *P*=.6704; data not shown)
- Consistent results were observed in the subgroup of patients who were followed up for at least 1 year (data not shown)
- CAR T-cell expansion (peak and area under the curve [AUC]) and tumor burden were lower in ZUMA-7 compared with ZUMA-1 (*P*<.05), whereas CAR T-cell peak expansion normalized to SPD was comparable between ZUMA-1 and ZUMA-7 (*P*=.5579; data not shown)
  - Sampling bias may have partly contributed to the differences in correlative analysis with ZUMA-1 Cohorts 1 and 2, as there were fewer collections on Day 14 for ZUMA-7 compared with ZUMA-1

Figure 3. Event-Free Survival in Major Prognostic Subgroups in ZUMA-7



	HR (95% CI)	*P* value
High SPD (Axi-Cel vs SOC)	0.289 (0.195-0.429)	<.0001
Low SPD (Axi-Cel vs SOC)	0.486 (0.329-0.717)	.0003
Axi-Cel SPD (High vs Low)	0.915 (0.604-1.388)	.6778
SOC SPD (High vs Low)	1.507 (1.055-2.153)	.0240

	HR (95% CI)	*P* value
Elevated LDH (Axi-Cel vs SOC)	0.324 (0.288-0.459)	<.0001
Normal LDH (Axi-Cel vs SOC)	0.496 (0.333-0.739)	.0006
Axi-Cel LDH (Elevated vs Normal)	1.108 (0.745-1.648)	.6132
SOC LDH (Elevated vs Normal)	1.556 (1.102-2.195)	.0119

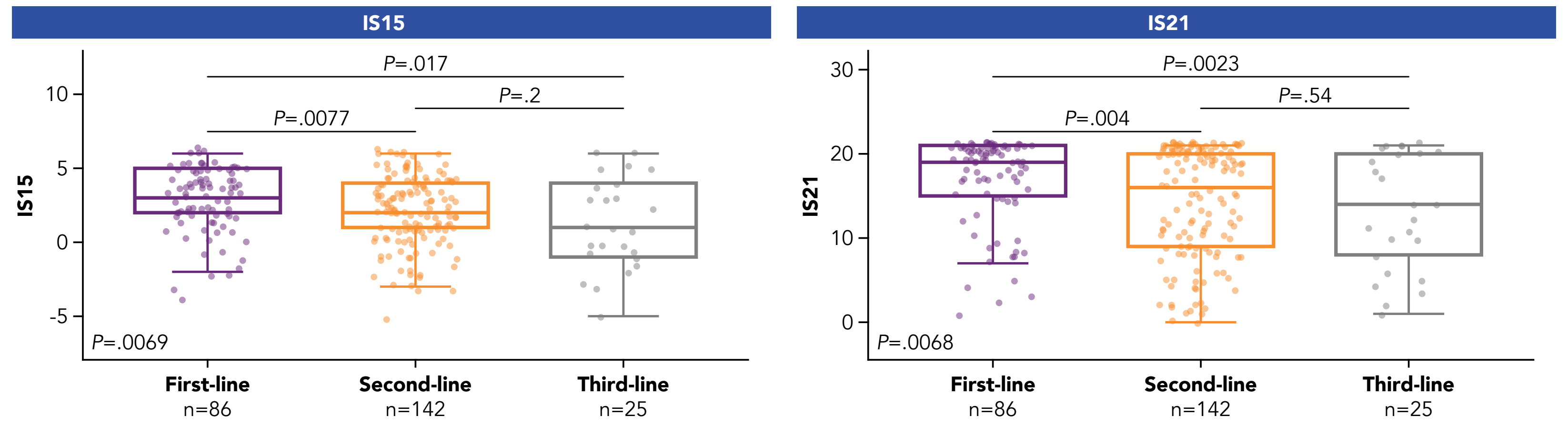
Patients at Risk	0	2	4	6	8	10	12	14	16	18	20	22	24	26	28	30	32	34
Axi-Cel, High SPD	77	76	47	41	40	40	39	38	37	33	22	17	8	5	5	2	0	
Axi-Cel, Low SPD	77	73	51	43	43	39	38	36	30	28	24	19	16	7	7	4	0	
SOC, High SPD	74	32	16	14	13	10	9	8	7	7	5	3	3	3	2	2	1	0
SOC, Low SPD	74	44	32	26	20	18	16	15	14	13	11	8	5	3	3	1	0	

Patients at Risk	0	2	4	6	8	10	12	14	16	18	20	22	24	26	28	30	32	34
Axi-Cel, Elevated LDH	92	88	55	45	44	43	42	41	39	35	26	21	12	4	4	2	0	
Axi-Cel, Normal LDH	78	73	50	46	46	43	42	40	34	31	25	18	14	8	8	4	0	
SOC, Elevated LDH	90	38	24	20	17	11	10	9	8	7	4	1	1	1	1	1	0	
SOC, Normal LDH	78	46	29	25	21	21	19	18	17	17	16	11	8	6	5	2	1	0

[a] Tumor burden was defined as high (>median) or low (≤median) SPD.
Axi-cel, axicabtagene ciloleucel; HR, hazard ratio; LDH, lactate dehydrogenase; SOC, standard of care; SPD, sum of product diameters.

- Axi-cel EFS was superior to SOC arm regardless of tumor burden, LDH (**Figure 3**), or molecular subclass (germinal center B-cell–like [GCB] vs non-GCB–like; data not shown)
- Tumor burden and LDH strongly associated with each other (data not shown)
- Tumor burden and LDH negatively associated with EFS in SOC patients
- Consistent results were observed with a 3721 mm² threshold of tumor burden for high versus low groups (median from ZUMA-1 Cohorts 1 and 2) and with a 2× upper limit of normal LDH threshold (data not shown)

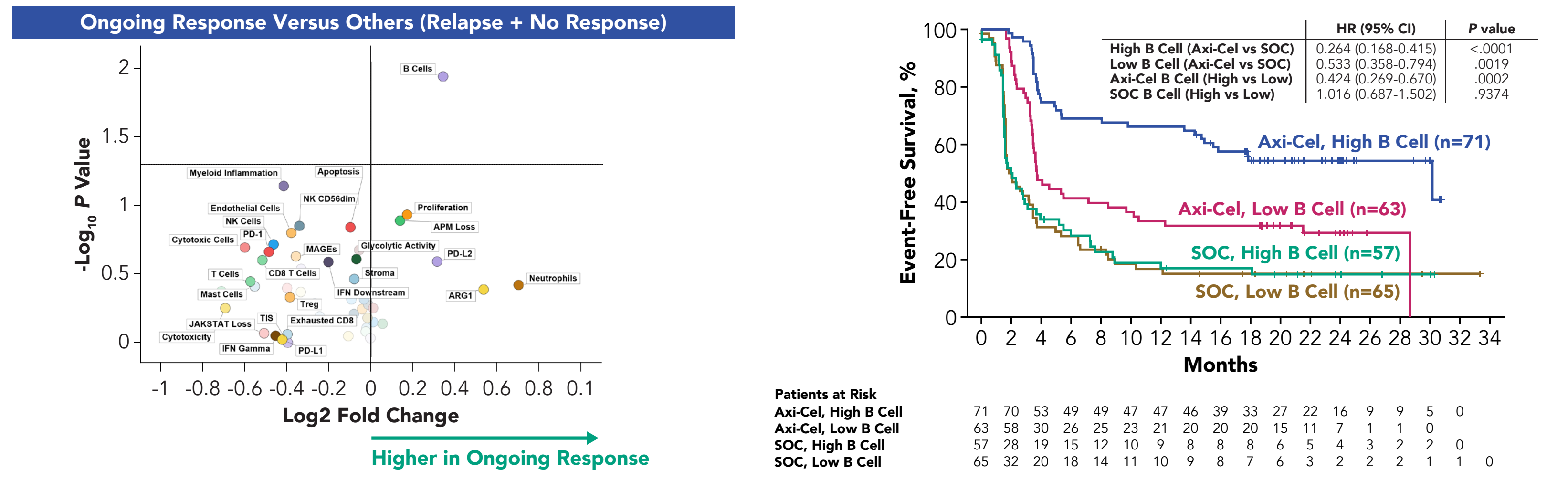
Figure 4. Immunosign 15 (IS15) and 21 (IS21) Indices Suggested a Favorable Immune Contexture in Earlier Lines of Therapy



First-line samples were obtained from ZUMA-7 biopsies collected before first-line therapy (archival); second-line samples were obtained from ZUMA-7 biopsies collected after first-line therapy; third-line samples were obtained from ZUMA-1 biopsies collected after last line of therapy.
IS, Immunosign.

- IS15 and IS21 decreased through lines of therapy, possibly underlying a more favorable tumor microenvironment (TME) immune contexture in earlier lines (**Figure 4**)
- IS21 previously associated with CR rate and PFS following treatment with axi-cel in third-line of therapy[9]

Figure 5. Associations of B-Cell Lineage Nanostring Signature With Improved EFS in ZUMA-7



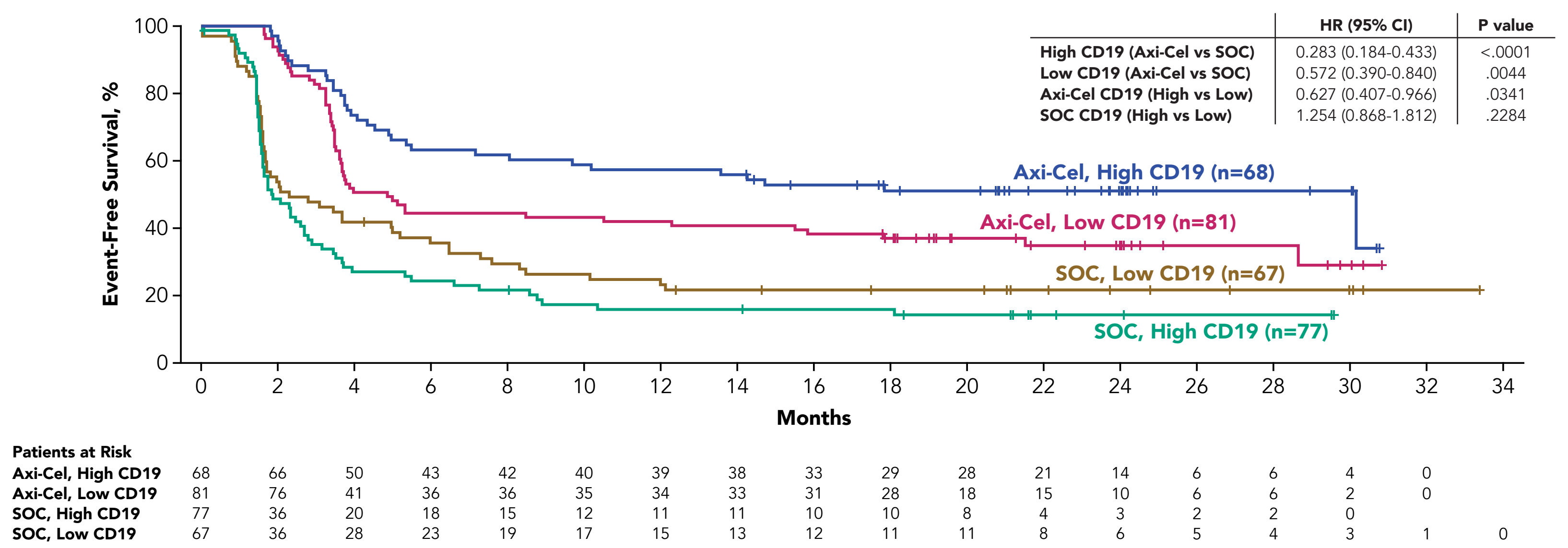
	HR (95% CI)	*P* value
High B Cell (Axi-Cel vs SOC)	0.264 (0.168-0.415)	<.0001
Low B Cell (Axi-Cel vs SOC)	0.533 (0.358-0.794)	.0019
Axi-Cel B Cell (High vs Low)	0.424 (0.269-0.670)	.0002
SOC B Cell (High vs Low)	1.016 (0.687-1.502)	.9374

Patients at Risk	0	2	4	6	8	10	12	14	16	18	20	22	24	26	28	30	32	34
Axi-Cel, High B Cell	71	70	53	49	49	47	47	46	39	33	27	22	16	9	9	5	0	
Axi-Cel, Low B Cell	63	58	30	26	25	23	21	20	20	20	15	11	7	1	1	0		
SOC, High B Cell	57	28	19	15	12	10	9	8	8	8	6	5	4	3	2	2	0	
SOC, Low B Cell	65	32	20	18	14	11	10	9	8	7	6	3	2	2	2	1	1	0

The fold change was calculated as Log2([group one]/[group two]). Statistical analysis was conducted using Kruskal–Wallis test (numerical vs categorical).
High B-cell lineage refers to a Nanostring IO-360 score >median score; low B-cell lineage refers to a Nanostring IO-360 score ≤median score.
APM, antigen presentation machinery; ARG1, arginase 1; axi-cel, axicabtagene ciloleucel; EFS, event-free survival; HR, hazard ratio; IFN, interferon; MAGEs, melanoma antigen genes; NK, natural killer; PD-1, programmed cell death protein 1; PD-L, programmed cell death ligand; SOC, standard of care; TIS, tumor inflammation signature; T_{reg}, regulatory T cell.

- Axi-cel patients with a stronger B-cell lineage signature exhibited improved EFS (**Figure 5**)
  - B-cell lineage signature refers to a predefined signature from Nanostring IO-360, derived from a proprietary algorithm incorporating gene expression values of BLK, CD19, MS4A1, TNFRSF17, FCRL2, FAM30A, PNOC, SPIB, and TCL1A

Figure 6. Axi-Cel Showed Improved EFS Versus SOC Regardless of CD19 Protein Expression[a]



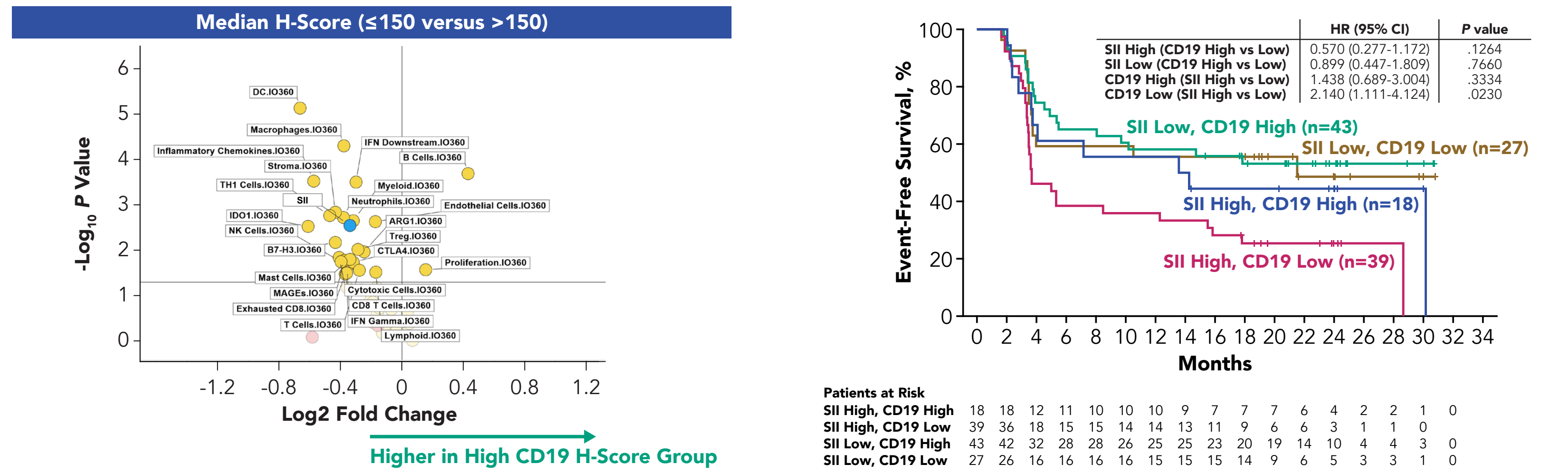
	HR (95% CI)	P value
High CD19 (Axi-Cel vs SOC)	0.283 (0.184-0.433)	<.0001
Low CD19 (Axi-Cel vs SOC)	0.572 (0.390-0.840)	.0044
Axi-Cel CD19 (High vs Low)	0.627 (0.407-0.966)	.0341
SOC CD19 (High vs Low)	1.254 (0.868-1.812)	.2284

Patients at Risk	0	2	4	6	8	10	12	14	16	18	20	22	24	26	28	30	32	34
Axi-Cel, High CD19	68	66	50	43	42	40	39	38	33	29	28	21	14	6	6	4	0	
Axi-Cel, Low CD19	81	76	41	36	36	35	34	33	31	28	18	15	10	6	6	2	0	
SOC, High CD19	77	36	20	18	15	12	11	11	10	10	8	4	3	2	2	0		
SOC, Low CD19	67	36	28	23	19	17	15	13	12	11	11	8	6	5	4	3	1	0

[a] Results were consistent by mRNA expression.
Axi-cel, axicabtagene ciloleucel; EFS, event-free survival; HR, hazard ratio; mRNA, messenger ribonucleic acid; SOC, standard of care.

- Axi-cel remained superior to SOC irrespective of high (>median) or low (≤median) CD19 expression protein/H-score; **Figure 6**)
- Patients deemed CD19 negative by immunohistochemistry (H-score <5) still presented substantial responses to axi-cel with 85% objective response rate (ORR) versus 67% ORR in the SOC arm (data not shown)

Figure 7. Association Between Efficacy and CD19 Expression (H-Score) in the Context of Immunosuppression in the ZUMA-7 Axi-Cel Arm



	HR (95% CI)	*P* value
SII High (CD19 High vs Low)	0.570 (0.277-1.172)	.1264
SII Low (CD19 High vs Low)	0.899 (0.447-1.809)	.7660
CD19 High (SII High vs Low)	1.438 (0.689-3.004)	.3334
CD19 Low (SII High vs Low)	2.140 (1.111-4.124)	.0230

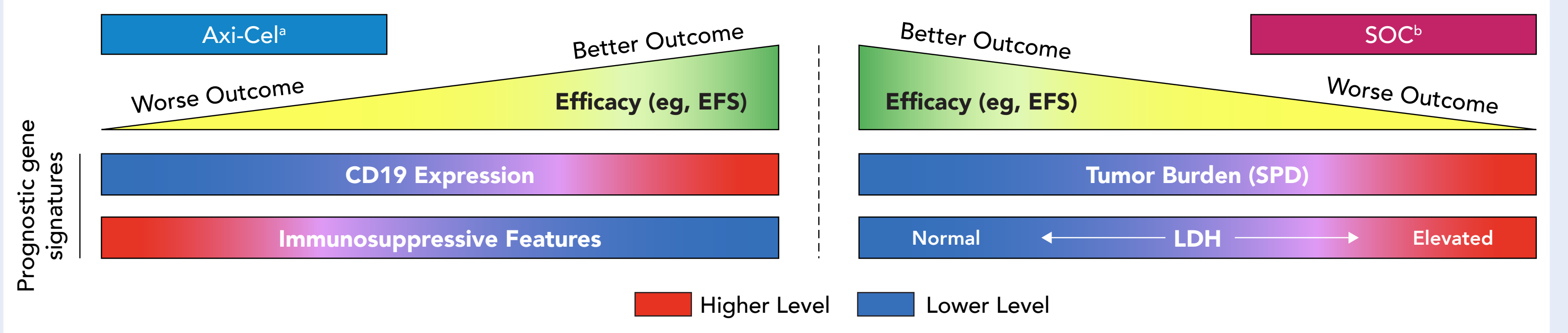
Patients at Risk	0	2	4	6	8	10	12	14	16	18	20	22	24	26	28	30	32	34
SII High, CD19 High	18	18	12	11	10	10	10	9	7	7	7	6	4	2	2	1	0	
SII High, CD19 Low	39	36	18	15	15	14	14	13	11	9	6	6	3	1	1	0		
SII Low, CD19 High	43	42	32	28	28	26	25	25	23	20	19	14	10	4	4	3	0	
SII Low, CD19 Low	27	26	16	16	16	16	15	15	15	14	9	6	5	3	3	1	0	

Negative associations between CD19 H-score and immune-infiltrated TME in ZUMA-7, including with a SII, which was generated through the calculation of root mean square of IO360 signatures: hypermutation, MMR loss, hypoxia, apoptosis, NOS2, MAGEs, mast cells, TGF-beta, ARG1, endothelial cells, stroma, B7-H3, myeloid inflammation. The fold change was calculated as Log([group one]/[group two]). Statistical analysis was conducted using Kruskal–Wallis test (numerical vs categorical). Only the predefined IO360 signatures and the immunosuppressive cluster are depicted.
ARG1, arginase 1; IFN, interferon; IS, Immunosign; MAGEs, melanoma antigen genes; MMR, mismatch repair; NK, natural killer; NOS, nitric oxide synthase; TH1, T helper type 1; TGF, transforming growth factor; SII, Stromal and Immunosuppressive Index; T_{reg}, regulatory T cell; TME, tumor microenvironment.

- Lower CD19 protein expression (H-score) overlapped with a more complex/immune-infiltrated TME, possibly enriched with a number of immunosuppressive features, including regulatory T cells, markers of T-cell exhaustions, ARG1, IDO1, B7-H3, CTLA4, and macrophage and myeloid gene expression signatures (**Figure 7**)
  - This underscores that the reduced efficacy of axi-cel in the CD19 H-score low (≤median) subgroup might be dependent on low/suboptimal target expression and/or concurrent immunosuppressive environment

CONCLUSIONS

Figure 8. Key Prognostic Markers in 2L LBCL in ZUMA-7



[a] Tumor burden, LDH and GCB subgroup did not impact outcomes in the axi-cel arm. [b] CD19 expression did not impact outcomes in the SOC arm.
Axi-cel, axicabtagene ciloleucel; EFS, event-free survival; GCB, germinal center B-cell–like; LDH, lactate dehydrogenase; SOC, standard of care; SPD, sum of product diameters.

- In 2L LBCL, axi-cel was superior to SOC across common prognostic subgroups, including higher tumor burden and LDH, and non-GCB status (**Figure 8**)
  - High tumor burden, elevated LDH, and non-GCB status were associated with poorer responses to SOC, but did not impact responses to axi-cel in ZUMA-7
- Markers of T-cell function and trafficking (gene expression signatures, IS15 and IS21) might decrease through lines of therapy as disease progresses, supporting earlier axi-cel intervention due to a more favorable immune contexture (higher T-cell signature in TME in 2L compared with third-line)
  - Responses to axi-cel were substantial and superior to SOC for both high and low CD19 expression
  - Lower CD19 protein expression (H-score) overlapped with a more complex/immune-infiltrated TME, possibly enriched with a number of immunosuppressive features
- Axi-cel showed improved EFS versus SOC irrespective of B-cell lineage signature strength or level of CD19 protein or mRNA expression
- Axi-cel intervention in 2L is supported by a favorable immune contexture and efficacy superior to SOC, including for patients with high tumor burden and elevated LDH

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- † Current affiliation: US Department of Veterans Affairs; Dr. Cheng was an employee of Kite when the studies reported here were conducted
- ‡ Current affiliation: Capstan Therapeutics; Dr. Bot was an employee of Kite when the studies reported here were conducted
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DISCLOSURES

FLL: consulting or advisory role with EcoR1, Emerging Therapy Solutions Gerson Lehman Group, Allogene, Amgen, bluebird bio, Bristol Myers Squibb/Celgene, Calibr, Iovance, Kite, Janssen, Legend Biotech, Novartis, Umoja, Cowen, Cellular Biomedicine Group, GammaDelta Therapeutics, Wugen; research funding from Kite, Allogene and Novartis; and patents, royalties, other intellectual property from several patents held by the institution in my name (unlicensed) in the field of cellular immunotherapy. **JC:** employment with Kite; stock or other ownership in Gilead Sciences and Five Prime Therapeutics; and travel support from Kite. **SV:** employment with Kite; stock or other ownership in Gilead; research funding from Kite. **RP:** employment with HalioDx. **PD:** consulting or advisory role for Gilead and Novartis; honoraria from Gilead and Novartis; speakers' bureau for Gilead and Novartis. **BTH:** honoraria from Kite; consultancy or advisory role for Kite; research funding from Kite; and travel support from Kite. **CL:** honoraria: Bristol Myers Squibb, Kadmon, Kite, Jazz, CareDX; consulting/advisory: Jazz, Incyte, Fresenius Kabi; researching funding: Incyte. **PLZ:** honoraria: Merck, Novartis, Servier, Incyte, Takeda, EUSA Pharma, Kyowa Kirin, AstraZeneca, AbbVie, Roche; consulting/advisory: Merck, Novartis, Servier, Incyte, Takeda, EUSA Pharma, Kyowa Kirin, AstraZeneca, AbbVie, Roche; speakers' bureau: Merck, Novartis, Servier, Incyte, Takeda, EUSA Pharma, Kyowa Kirin, AstraZeneca, AbbVie, Roche. **NK:** consulting or advisory role: Kite, Novartis, Amgen, Bristol Myers Squibb; speakers' bureau: Sanofi, Miltenyi; research funding: Riemser, Neovii, Novartis, Celgene; travel support: Jazz, Sanofi, Riemser, Bristol Myers Squibb. **AL-G:** consulting/advisory: Kite/Gilead, Celgene/Bristol Myers Squibb, Incyte, Roche, Takeda; researching funding: Roche, Celgene, Gilead, Incyte. **HG:** consulting or advisory role for Gilead/Kite, Novartis, Amgen, Therakos, Takeda, Miltenyi, Sanofi. Speakers' bureau of Novartis, Celgene, Jazz, Takeda, Therakos. **WZ:** employment with Kite; stock or other ownership in Gilead. **GT:** employment with Kite. **CT:** employment with Kite; stock or other ownership in Gilead Sciences. **PC:** employment with Kite; stock or other ownership in Gilead Science; and travel support from Kite. **AB:** leadership and stock ownership with Capstan Therapeutics, former employment with Kite; stock or other ownership in Kite, CeroTx, Elicio Tx; advisory role for Elicio Tx and CeroTx. **RS:** employment with Kite, and Atara; leadership role with Kite and Atara; stock or other ownership in Kite and Atara; and patents, royalties and other intellectual property from Kite and Atara. **SF:** employment with Kite; stock or ownership with Kite; and patents, royalties, other intellectual property from Tusk Therapeutics. **JG:** employment from HalioDx Veracyte; leadership: Northwest Biotherapeutics, Lunaphore; stock or other ownership in HalioDx Veracyte; consulting or advisory role: Northwest Biotherapeutics, Lunaphore; research funding: Imcheck Therapeutics, HalioDx Veracyte; travel support and accommodations: HalioDx Veracyte; and patents, royalties, or other intellectual property from Inserm.

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