

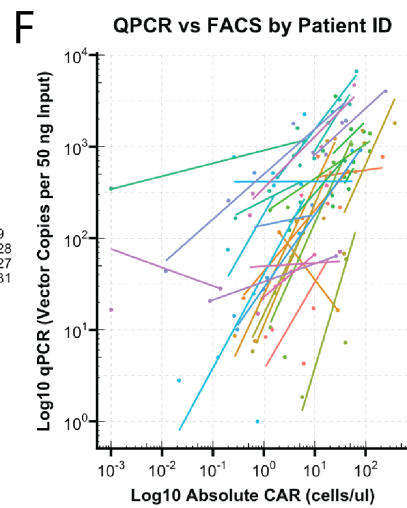
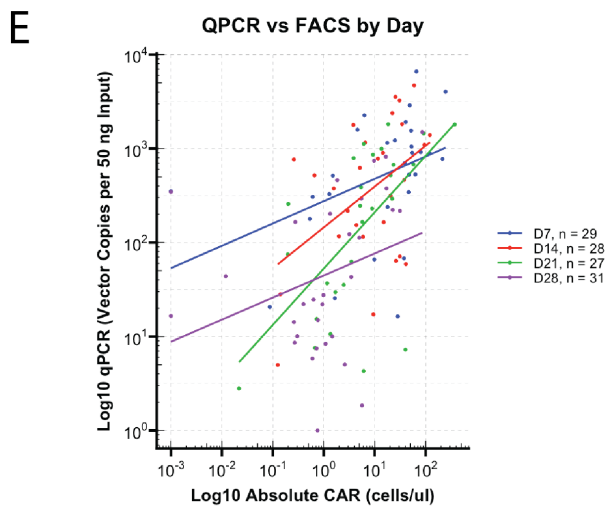
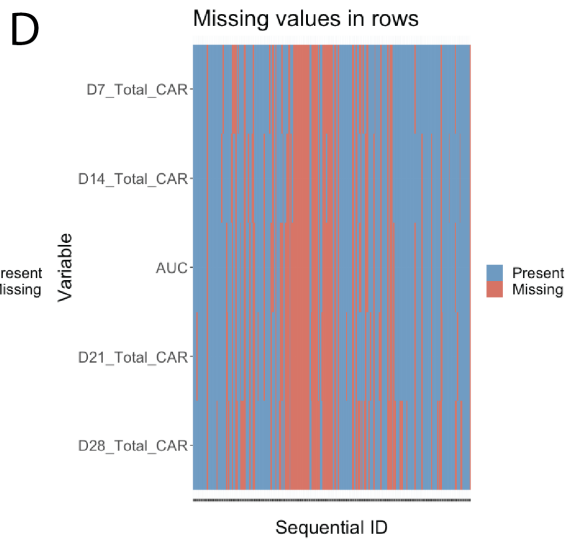
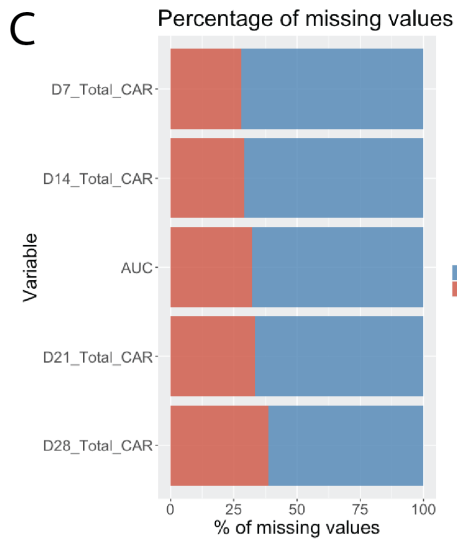
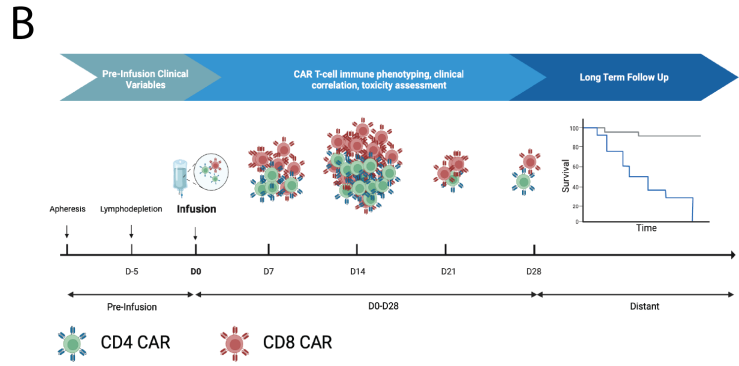
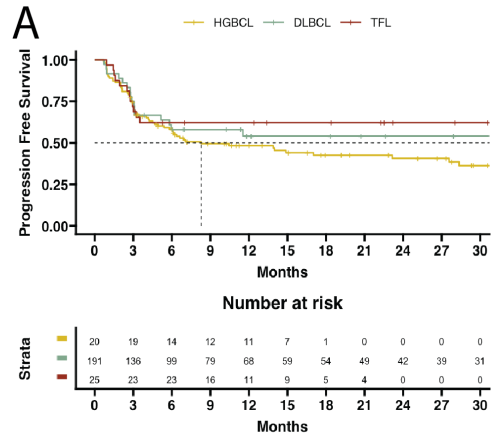
<b>Variable</b>	<b>No CAR-FACS</b>	<b>Any CAR-FACS</b>	<b>p</b>
n	48	188	
<b>Age at Apheresis (median [IQR])</b>	<b>66.50 [58.00, 70.25]</b>	<b>63.00 [54.00, 71.00]</b>	<b>0.23</b>
Sex = Female (%)	20 (41.7)	74 (39.4)	0.9
<b>ECOG (%)</b>			<b>0.132</b>
0	14 (29.2)	31 (16.5)	
1	<b>30 (62.5)</b>	<b>141 (75.0)</b>	
2+	4 (8.3)	16 (8.5)	
<b>Stage at Apheresis (%)</b>			<b>0.056</b>
1	6 (12.5)	18 (9.6)	
2	<b>10 (20.8)</b>	<b>16 (8.5)</b>	
3	3 (6.2)	25 (13.3)	
4	<b>29 (60.4)</b>	<b>129 (68.6)</b>	
History CNS Disease = Yes (%)	3 (6.2)	14 (7.4)	1
<b>Prior Auto Transplant = Yes (%)</b>	<b>10 (20.8)</b>	<b>43 (22.9)</b>	<b>0.914</b>
Prior Systemic Lines (median [IQR])	3 [2, 4]	3 [2, 4]	0.092
<b>Normalized PreLD LDH (median [IQR])</b>	<b>1.01 [0.84, 1.24]</b>	<b>1.09 [0.82, 1.51]</b>	<b>0.286</b>
ALC at Leukapheresis (median [IQR])	0.79 [0.53, 1.15]	0.78 [0.50, 1.19]	0.814
<b>COO = non-GCB (%)</b>	<b>16 (39.0)</b>	<b>61 (43.0)</b>	<b>0.787</b>
<b>Histology (%)</b>			<b>0.062</b>
<b>DLBCL/Other LBCL</b>	<b>32 (66.7)</b>	<b>91 (48.4)</b>	
FL	0 (0.0)	20 (10.6)	
<b>HGBCL</b>	<b>8 (16.7)</b>	<b>28 (14.9)</b>	
MCL	4 (8.3)	21 (11.2)	
<b>TFL</b>	<b>4 (8.3)</b>	<b>28 (14.9)</b>	

**Supplemental Table 1.** The populations with and without CAR T-cell expansion data were comparable. There were more patients in the population with CAR T-cell data than without CAR T-cell data due to the temporal nature of missing data. Most missing data was due to the pandemic, which mainly occurred prior to regular treatment of MCL and FL patients with CAR T-cell therapy. There was a trend towards more patients with stage 2 disease in the population without CAR T-cell expansion data. P-values were derived from unadjusted Kruskal Wallis test.

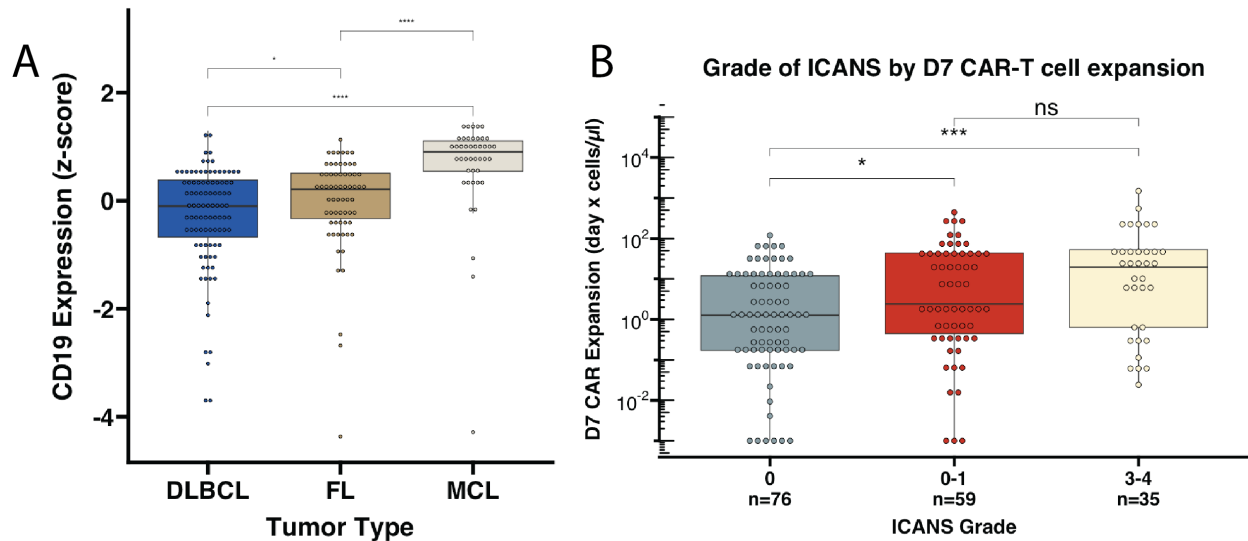
Variable	CAR <sup>low</sup> /LDH <sup>low</sup>	CAR <sup>high</sup> /LDH <sup>low</sup>	p
n	27	32	
Age at apheresis (median [IQR])	61.00 [54.50, 65.50]	60.00 [49.00, 68.00]	0.933
Sex = Female (%)	11 (40.7)	13 (40.6)	1
ECOG = 2+ (%)	0 (0.0)	1 (3.1)	1
ALC Leukapheresis (median [IQR])	0.90 [0.56, 1.10]	0.66 [0.50, 1.11]	0.394
History CNS Disease = Yes (%)	2 (7.4)	2 (6.2)	1
Prior Auto = Yes (%)	7 (25.9)	11 (34.4)	0.676
Systemic Lines (median [IQR])	3.00 [2.00, 3.00]	3.00 [2.00, 4.00]	0.464
Stage at Apheresis (%)			0.052
1	2 (7.4)	4 (12.5)	
2	0 (0.0)	7 (21.9)	
3	4 (14.8)	4 (12.5)	
4	21 (77.8)	17 (53.1)	
Normalized PreLD LDH (median [IQR])	0.77 [0.52, 0.93]	0.81 [0.66, 0.99]	0.263
Pre-infusion ctDNA (median [IQR])	6.23 [0.29, 35.53]	19.72 [6.16, 43.52]	0.318
Histology (%)			0.135
DLBCL/Other LBCL	18 (66.7)	13 (40.6)	
HGBCL	4 (14.8)	9 (28.1)	
TFL	5 (18.5)	10 (31.2)	
COO = non-GCB (%)	12 (48.0)	13 (41.9)	0.854

**Supplemental Table 2.** Patient characteristics of CAR<sup>low</sup>/LDH<sup>low</sup> population vs CAR<sup>high</sup>/LDH<sup>low</sup> population. The CAR high population trended towards having higher stage disease at apheresis, though there was no difference in pre-LD LDH or pre-infusion ctDNA where available.

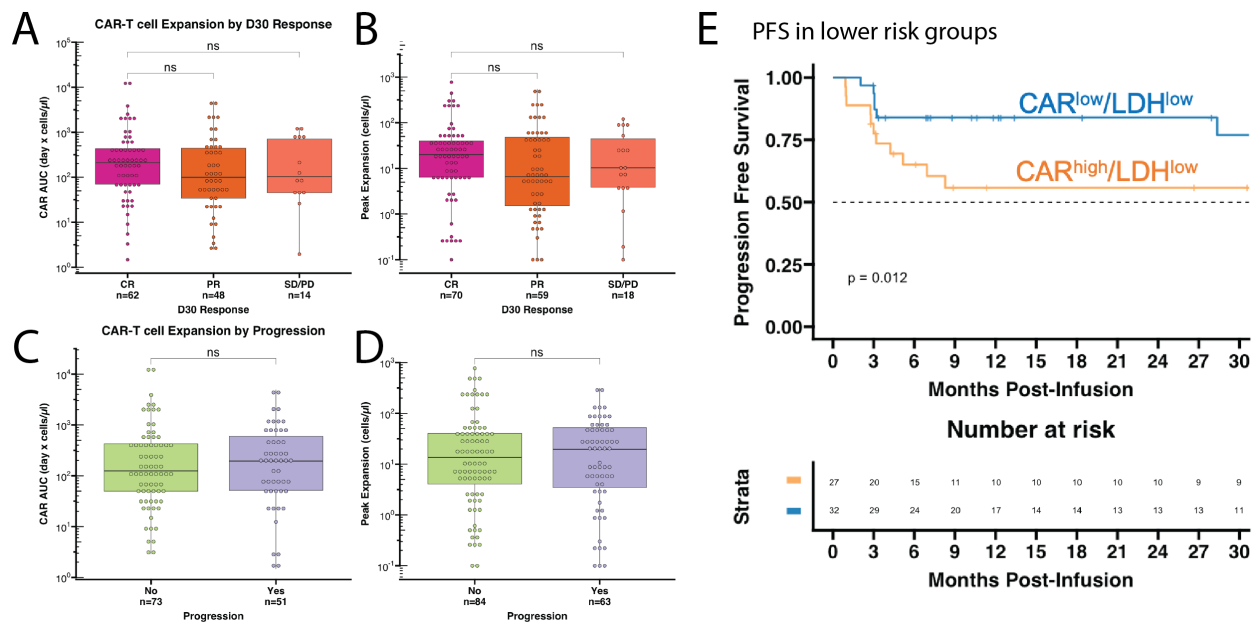
## Supplemental Figures:



**Supplemental Figure 1. A large cohort of CAR19 expansion data.** **A)** There was no difference in LBCL PFS outcomes based on measured histologic outcomes of HGBCL, TFL, and DLBCL. **B)** Schematic of data collection and monitoring in this study. **C-D)** Missing CAR-FACS data, expressed by percentage missing plot and row-wise missing plot, respectively. CAR-FACS data was partially lost during D28 samples due to outpatient follow up. Most missing datapoints occurred when collection was paused during the COVID19 pandemic. Overall, 79.6% of prospectively followed patients had at least one available timepoint. **E-F)** CAR19 quantification by flow cytometry is highly correlated with CAR19 quantification by qPCR. The left panel (**E**) demonstrates the linear trend line for correlation between day and the right panel demonstrates the linear trend line by patient (**F**).



**Supplemental Figure 2. Variation in CAR19 expansion informs toxicity differences.** **A)** There is increased CD19 expression in MCL tumors relative to FL or LBCL tumors as determined by mRNA microarray. **B)** The figure demonstrates the association between D7 CAR T-cell expansion and the development of high grade ICANS (Wilcoxon test). Similar analysis using multivariate ordinal regression with backwards step elimination of variables demonstrates that CAR T-cell expansion, but not histology, was most strongly associated with development of ICANS, with or without excluding D7 values that occur after initial ICANS development.



**Supplemental Figure 3.** Association of CAR19 expansion with disease response. **A-B)** CAR T-cell expansion defined by peak expansion or AUC was not increased in patients whose best response was a CR versus patients whose best response was PR, SD, or PD. **C-D)** CAR T-cell expansion defined by AUC or peak expansion was not higher in patients who did not progress versus those who did. **E)** Patients in the CAR<sup>+</sup>/LDH<sup>-</sup> group had worse PFS outcomes than patients in the CAR<sup>-</sup>/LDH<sup>-</sup> group in head-to-head comparison (Cox proportionate hazard ratio). AUC = area under the curve, CR = complete response, PR = partial response, SD = stable disease, PD = progressive disease. All binary significance values represent Wilcoxon tests, survival analysis is by log-rank test.