Corporate presentation

Transformative genome-edited therapies for patients
Forward-looking statements

All statements in this presentation, other than statements of historical facts, are forward-looking statements, within the meaning of the Private Securities Litigation Reform Act of 1995. These forward-looking statements speak only as of the date of this presentation and are subject to a number of known and unknown risks, assumptions, uncertainties, and other factors that may cause the actual results, levels of activity, performance, or achievements of Caribou Biosciences, Inc. (the “Company,” “Caribou,” “we,” or “our”) to be materially different from those expressed or implied by any forward-looking statements. The words “may,” “will,” “should,” “expect,” “plan,” “anticipate,” “could,” “intend,” “target,” “project,” “contemplate,” “believe,” “estimate,” “predict,” “potential,” or “continue” or the negative of these terms or other similar expressions are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words. All statements, other than statements of historical facts contained in this presentation, are forward-looking statements, including but not limited to any statements regarding the initiation, timing, progress, strategy, plans, objectives, expectations (including as to the results) with respect to our product candidate preclinical studies, clinical trials, and research programs, including our expectations and timing regarding the release of dose expansion clinical data, and emerging translational data from our ongoing ANTLER phase 1 clinical trial for our CB-010 product candidate, disclosure of the recommended Phase 2 dose for CB-010, and an updated timeline for our planned phase 3 pivotal trial for CB-010 in second-line large B cell lymphoma patients (and the conditions to meet that timeline); the status, progress, and expectations relating to the timing of release of clinical data from our ongoing CaMMouflage phase 1 clinical trial for our CB-011 product candidate in patients with multiple myeloma; the status, progress, and expectations relating to the timing of release of clinical data from our ongoing AmpLify phase 1 clinical trial for our CB-012 product candidate in patients with acute myeloid leukemia; the timing for the initiation of our GALLOP phase 1 clinical trial for adults with lupus nephritis and extrarenal lupus; our ability to successfully develop our product candidates and to obtain and maintain regulatory approval for our product candidates; the number and type of diseases, indications, or applications we intend to pursue for our product candidates; the beneficial characteristics, safety, efficacy, therapeutic effects, and potential advantages of our product candidates; the expected timing or likelihood of regulatory filings and approval for our product candidates; our expected cash runway; and the sufficiency and anticipated use of our existing capital resources to fund our future capital expenditure requirements and needs for additional financing. You are cautioned not to place undue reliance on these forward-looking statements, which speak only as of the date this presentation is given. This presentation discusses product candidates that are or will be under clinical investigation and that have not yet been approved for marketing by the U.S. Food and Drug Administration. No representation is made as to the safety or effectiveness of these product candidates for the therapeutic uses for which our product candidates are being or will be studied.

As a result of many factors, including risks related to our limited operating history, history of net operating losses, financial position and our ability to raise additional capital as needed to fund our operations and product candidate development; uncertainties related to the initiation, cost, timing, and progress, and results of our current and future research and development programs, preclinical studies, and clinical trials; risks that initial or interim clinical trial data will not ultimately be predictive of the safety and efficacy of our product candidates or that clinical outcomes may differ as more clinical data becomes available; the risk that preclinical study results we observed will not be borne out in human patients; our ability to obtain and maintain regulatory approval for our product candidates; risks that our product candidates, if approved, may not gain market acceptance due to negative public opinion and increased regulatory scrutiny of cell therapies involving genome editing; our ability to meet future regulatory standards with respect to our products; our ability to obtain key regulatory input and approvals, our ability to establish and/or maintain intellectual property rights covering our product candidates and genome-editing technology; risks of third parties asserting that our product candidates infringe their patents; developments related to our competitors and our industry; our reliance on third parties to conduct our clinical trials and manufacture our product candidates; the impact of public health crises and geopolitical events on our business and operations; and other risks described in greater detail in our filings with the Securities and Exchange Commission (the “SEC”), including the section titled “Risk Factors” of our Annual Report on Form 10-K for the year ended December 31, 2023, and other filings we make with the SEC; the events and circumstances reflected in our forward-looking statements may not be achieved or may not occur, and actual results could differ materially from those described in or implied by the forward-looking statements contained in this presentation.

Caution should be exercised when interpreting results from separate trials involving other CAR-T cell therapies. The results of other CAR-T cell therapies presented or referenced in these slides have been derived from publicly available reports of clinical trials not conducted by us, and we have not performed any head-to-head trials comparing any of these other CAR-T cell therapies with CB-010. As such, the results of these other clinical trials may not be comparable to clinical results for CB-010. The design of these other trials vary in material ways from the design of the clinical trials for CB-010, including with respect to patient populations, follow-up times, the clinical trial stage, and subject characteristics. As a result, cross-trial comparisons may have no interpretive value on our existing or future results. For further information and to understand these material differences, you should read the reports for the other CAR-T cell therapies’ clinical trials and the sources included in this presentation.

In light of the foregoing, you are urged not to rely on any forward-looking statement in reaching any conclusion or making any investment decision about our securities. The forward-looking statements in this presentation are made only as of the date hereof. Except to the extent required by law, the Company assumes no obligation and does not intend to update any of these forward-looking statements after the date of this presentation or to confirm these statements to actual results or revised expectations. From time to time, we may release additional clinical data from our ongoing ANTLER phase 1 clinical trial, our CaMMouflage phase 1 clinical trial, our AmpLify phase 1 clinical trial, and our GALLOP phase 1 clinical trial. We make no representations regarding such additional clinical data or the timing of its release, or whether any such data will support or contradict the findings of the clinical data reported earlier.

This presentation shall not constitute an offer to sell or the solicitation of an offer to buy any securities.
Precision genome editing with industry-leading expertise

chRDNA precision genome-editing technology
- Novel, next-generation CRISPR technology engineered for superior specificity and precision
- Multiplex editing designed to maintain genomic integrity

Armored off-the-shelf cell therapies
- Allogeneic CAR-T enhanced activity
  - Checkpoint disruption
  - Immune cloaking

4 clinical-stage programs targeting hematologic malignancies and autoimmune diseases

Resourced for successful execution
- Experienced, mission-driven leadership
- Strong in-house process development capabilities
- Robust IP portfolio
- $312M\(^1\) in cash, runway into H2 2026

chRDNA: CRISPR hybrid RNA-DNA
\(^1\) $311.8M in cash, cash equivalents, and marketable securities as of June 30, 2024.
Advancing pipeline of clinical-stage allogeneic CAR-T cell therapies for hematologic malignancies and autoimmune diseases

<table>
<thead>
<tr>
<th>Program</th>
<th>Clinical trial</th>
<th>Target</th>
<th>Indication</th>
<th>Preclinical</th>
<th>Phase 1</th>
<th>Phase 2</th>
<th>Phase 3</th>
<th>Designations</th>
</tr>
</thead>
<tbody>
<tr>
<td>CB-010</td>
<td>ANTLER Dose expansion</td>
<td>CD19</td>
<td>r/r B-NHL</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>RMAT, Fast Track, Orphan Drug</td>
</tr>
<tr>
<td>CB-011</td>
<td>CaMMouflage Dose escalation</td>
<td>BCMA</td>
<td>r/r MM</td>
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<td></td>
<td></td>
<td></td>
<td>Fast Track, Orphan Drug</td>
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<tr>
<td>CB-012</td>
<td>AMpLify Dose escalation</td>
<td>CLL-1*</td>
<td>r/r AML</td>
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<td></td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

**Hematologic malignancies**

**Autoimmune diseases**

ERL: extrarenal lupus; LN: lupus nephritis, RMAT: Regenerative Medicine Advanced Therapy

*Also known as CD371
chRDNA technology
chRDNA guides promote on-target and reduce off-target edits

First-generation all-RNA CRISPR-Cas

chRDNA CRISPR hybrid RNA-DNA

Promotes on-target edits

Reduces off-target edits

DNA bases

On-target DNA

Successful on-target cleavage

Off-target DNA

Mismatches

DNA incorporation prevents off-target cleavage
chRDNA guides significantly improve editing specificity

**Knockout**

<table>
<thead>
<tr>
<th>Cas9</th>
<th>Cas12a</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>all-RNA guide</strong></td>
<td><strong>chRDNA</strong></td>
</tr>
<tr>
<td>PDCD1</td>
<td></td>
</tr>
<tr>
<td>ON</td>
<td>OFF</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>

| Cas12a chRDNA genome editing + AAV6 transduction leads to >60% of manufacturing-scale engineered T cells with all 4 intended edits |

**Knock-in**

| Insert 1: 80% |
| Insert 2: 76% |
| 13.1 | 63.0 |

<table>
<thead>
<tr>
<th>PDCD1</th>
<th>TRAC</th>
<th>B2M</th>
</tr>
</thead>
<tbody>
<tr>
<td>ON OFF ON OFF</td>
<td>ON OFF</td>
<td>ON OFF</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

% editing

Corporation Presentation | July 2024
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Engineering for improved activity against disease is key to unlocking the full potential of allogeneic cell therapies

Caribou is implementing multiple armoring strategies

Checkpoint disruption (CB-010)
PD-1 knockout (KO) sustains initial activity due to exhaustion resistance

Immune cloaking (CB-011)
B2M KO plus B2M–HLA-E fusion knock-in may delay host immune rejection

Combination strategies (CB-012)
Checkpoint disruption AND immune cloaking
Caribou is a leader in the allogeneic CAR-T cell space with a platform of genome-edited cell therapies

1st allogeneic anti-CD19 CAR-T cell therapy in the clinic with **checkpoint disruption** via PD-1 knockout (KO)¹ to reduce CAR-T cell exhaustion

1st allogeneic anti-BCMA CAR-T cell therapy with **immune cloaking** via B2M KO and insertion of B2M-HLA-E fusion protein¹

1st allogeneic CAR-T cell therapy with both **checkpoint disruption** and **immune cloaking**¹

¹ To Caribou’s knowledge
Off-the-shelf CAR-T cell therapy programs

CB-010 for r/r B-NHL
CB-010 for lupus
CB-011 for r/r MM
CB-012 for r/r AML
Patients shouldn’t have to wait for treatment

<table>
<thead>
<tr>
<th>Allogeneic therapy</th>
<th>Autologous therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>N=many per batch</td>
<td>N=1 per batch</td>
</tr>
</tbody>
</table>

### Allogeneic Therapy

- **Screening + HLA typing**
- **Product shipment**
- **Days**
- **Lymphodepletion**

<table>
<thead>
<tr>
<th>Autologous Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Screening</strong></td>
</tr>
<tr>
<td><strong>Queuing, leukopheresis scheduling</strong></td>
</tr>
<tr>
<td><strong>Leukopheresis</strong></td>
</tr>
<tr>
<td><strong>Bridging therapy</strong></td>
</tr>
<tr>
<td><strong>Sample shipment</strong></td>
</tr>
<tr>
<td><strong>Manufacturing, product failure identification</strong></td>
</tr>
<tr>
<td><strong>Product shipment</strong></td>
</tr>
</tbody>
</table>

### The Future of Cell Therapy is Off-the-Shelf

- **Weeks to months**
- **Lymphodepletion**

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1. Mikhael, J. et al. JCO Oncology Practice 2022 18:12, 800-807

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CB-010

Allogeneic anti-CD19 CAR-T cell therapy with a PD-1 knockout for r/r B cell non-Hodgkin lymphoma (B-NHL)
CB-010 has a PD-1 KO designed to reduce CAR-T cell exhaustion

Armored with 3 genome edits

1. **TRAC gene knockout (KO)**
   - Eliminates TCR expression, reduces GvHD risk

2. **Anti-CD19 CAR site-specific insertion into TRAC locus**
   - Eliminates random integration, targets tumor antigen

3. **PD-1 KO for enhanced antitumor activity**
   - Reduces CAR-T cell exhaustion
   - Potentially contributes to initial tumor debulking

---

**To Caribou’s knowledge.**

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**1**st CAR-T in the clinic with **checkpoint disruption** via PD-1 KO

- Cas9 chRDNA editing for **reduced off-target editing** and enhanced genomic integrity
- Anti-CD19 scFv FMC63 with a 4-1BB costimulatory domain

---

**CAR:** chimeric antigen receptor; **KO:** knockout; **CD:** cluster of differentiation; **chRDNA:** CRISPR hybrid RNA-DNA; **CRISPR:** clustered regularly interspaced short palindromic repeats; **PD-1:** programmed cell death protein 1; **TCR:** T cell receptor; **TRAC:** T cell receptor alpha constant; **scFv:** single-chain variable fragment
**CB-010 ANTLER Phase 1 trial in 2L LBCL**

**Part A: 3+3 dose escalation - completed (N=16)**
- Eligibility: aggressive r/r B-NHL\(^1\) with ≥2 prior lines of chemoimmunotherapy or primary refractory
- Exclusion: prior CD19-targeted therapy

**Part B: dose expansion - enrolling**
- Eligibility: 2\(^{nd}\) line LBCL\(^2\)
- Exclusion: prior CD19-targeted therapy
- Objective: tumor response, RP2D

### r/r B-NHL

**Lymphodepletion**

<table>
<thead>
<tr>
<th>Time</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>-9 to -3 DAYS</td>
<td>DAY 0</td>
<td>28 DAYS</td>
<td>3 MONTHS</td>
<td>6 MONTHS</td>
<td>9 MONTHS</td>
<td>12 MONTHS</td>
<td></td>
</tr>
</tbody>
</table>

- Cyclophosphamide (60 mg/kg/d for 2 days) followed by Fludarabine (25 mg/m\(^2\)/d for 5 days)\(^3\)

**CB-010**

**SINGLE DOSE**

**Dose level 1:** 40x10\(^6\) CAR-T cells

**Dose level 2:** 80x10\(^6\) CAR-T cells

**Dose level 3:** 120x10\(^6\) CAR-T cells

**Dose expansion:** 30\(^{th}\) patient dosed; 80x10\(^6\) CAR-T cells selected as RP2D

Will enroll ~20 patients at RP2D to prospectively evaluate partial (≥4) HLA matching, DSA screening

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**Subtypes include:** DLBCL (diffuse large B cell lymphoma), HGBL (high-grade B cell lymphoma), tFL (transformed DLBCL from follicular lymphoma, PMBCL (primary mediastinal large B cell lymphoma), FL (follicular lymphoma, aggressively behaving with POD24 (high risk)), MZL (marginal zone lymphoma).

**NCT04637763**

DSA: donor-specific antibodies; HLA: human leukocyte antigen

\(^1\) Subtypes include: DLBCL (diffuse large B cell lymphoma), HGBL (high-grade B cell lymphoma), tFL (transformed DLBCL from follicular lymphoma, PMBCL (primary mediastinal large B cell lymphoma), FL (follicular lymphoma, aggressively behaving with POD24 (high risk)), MZL (marginal zone lymphoma).

\(^2\) LBCL subtypes include: DLBCL NOS (DLBCL not otherwise specified), HGBL, transformed DLBCL from FL or MZL, and PMBCL.

CB-010’s foundational data: durable responses in dose escalation

4 of 4 DLBCL patients remain in CR since last data cutoff June 20, 2023

<table>
<thead>
<tr>
<th>Subtype</th>
<th>Dose</th>
<th>PLoT</th>
<th>Pt #</th>
<th>≥4 HLA</th>
</tr>
</thead>
<tbody>
<tr>
<td>FL¹</td>
<td>40M</td>
<td>8</td>
<td>1</td>
<td>✓</td>
</tr>
<tr>
<td>DLBCL</td>
<td>40M</td>
<td>4</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>DLBCL</td>
<td>80M</td>
<td>1</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>DLBCL</td>
<td>80M</td>
<td>1</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>PMBCL²</td>
<td>40M</td>
<td>2</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>MCL</td>
<td>40M</td>
<td>2</td>
<td>13</td>
<td>✓</td>
</tr>
<tr>
<td>DLBCL</td>
<td>80M</td>
<td>2</td>
<td>9</td>
<td>✓</td>
</tr>
<tr>
<td>MZL</td>
<td>80M</td>
<td>4</td>
<td>15</td>
<td></td>
</tr>
<tr>
<td>MCL</td>
<td>40M</td>
<td>4</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>FL¹</td>
<td>40M</td>
<td>2</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>DLBCL</td>
<td>40M</td>
<td>2</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>DLBCL</td>
<td>120M</td>
<td>2</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>HGBL</td>
<td>40M</td>
<td>1</td>
<td>14</td>
<td></td>
</tr>
<tr>
<td>MCL</td>
<td>80M</td>
<td>2</td>
<td>16</td>
<td></td>
</tr>
<tr>
<td>HGBL</td>
<td>120M</td>
<td>1</td>
<td>12</td>
<td></td>
</tr>
<tr>
<td>DLBCL</td>
<td>120M</td>
<td>2</td>
<td>11</td>
<td></td>
</tr>
</tbody>
</table>

CR: complete response  
PR: partial response  
SD: stable disease  
PD: progressive disease

1 Aggressively behaving, with POD24 (high risk).
2 Patient 5’s 3-month scan conducted on day 63 post CB-010 as per investigator’s discretion.

Denotes patients with continued CRs since June 20, 2023 on June 20, 2023 data

Overall r/r B-NHL dose escalation

5 of 6 patients with CR as of June 20, 2023 data cutoff remain in CR as of April 1, 2024

2 patients completed 24-month follow-up in CR

Data collection ongoing, efficacy based on Lugano criteria

DLBCL: diffuse large B cell lymphoma; FL: follicular lymphoma; HGBL: high-grade B cell lymphoma; MCL: mantle cell lymphoma; MZL: marginal zone lymphoma; PLoT: prior lines of therapy (#); PMBCL: primary mediastinal large B cell lymphoma

* = patients with ≥4 HLA (human leukocyte antigen) matches (all other patients have ≤3 HLA matches).

ANTLER Phase 1 clinical trial as of April 1, 2024 cutoff date, data collection ongoing.
CB-010 with partial HLA matching shows safety, efficacy, and durability can potentially rival autologous CAR-T cell therapies

<table>
<thead>
<tr>
<th>1 dose per patient, 3 dose levels evaluated, all generally well tolerated</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>RP2D selected</strong> 80x10^6 CAR-T cells</td>
</tr>
</tbody>
</table>
| **2L LBCL at RP2D**  
CR rate: 50%  
Median duration of CR: NR |

Retrospective analysis in 13 patients with ≥4 HLA allele matching; subset includes: 2L LBCL (N=10), 3L LBCL (N=1), and 3L+ B-NHL (N=2).

- **Median PFS 14.4 months**  
  (95% CI: 1.7-NE)  
  observed in 13 patients with partial (≥4) HLA matching¹

- **Advancing CB-010 with partial HLA matching** in 2L LBCL and lupus  
  Phase 1 clinical trials

2L: second-line; 3L: third-line; B-NH: B cell non-Hodgkin's lymphoma; CI: confidence interval; CR: complete response; HLA: human leukocyte antigen; LBCL: large B cell lymphoma; NE: not estimable; NR: not reached; PFS: progression free survival; partial HLA matching: patient has ≥4 HLA alleles that match donor T cells used for CB-010 manufacturing; RP2D: recommended Phase 2 dose; CR: complete response; NR: not reached

¹Retrospective analysis in 13 patients with ≥4 HLA allele matching; subset includes: 2L LBCL (N=10), 3L LBCL (N=1), and 3L+ B-NHL (N=2). ANTLER Phase 1 clinical trial as of April 1, 2024 cutoff date, data collection ongoing.
Improved PFS for all patients treated with CB-010 from a donor with partial HLA matching

PFS by level of HLA matching

All patients
N=46*, median PFS (95% CI)

<table>
<thead>
<tr>
<th>HLA matching</th>
<th>Survival probability (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>≥4 matches</td>
<td>100, 90, NE</td>
</tr>
<tr>
<td>2-3 matches</td>
<td>80, 60, 40, 20</td>
</tr>
<tr>
<td>&lt;2 matches</td>
<td>60, 40, 20</td>
</tr>
</tbody>
</table>

PFS by level of HLA matching

LBCL patients
N=40*, median PFS (95% CI)

<table>
<thead>
<tr>
<th>HLA matching</th>
<th>Survival probability (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>≥4 matches</td>
<td>100, 90, NE</td>
</tr>
<tr>
<td>2-3 matches</td>
<td>80, 60, 40, 20</td>
</tr>
<tr>
<td>&lt;2 matches</td>
<td>60, 40, 20</td>
</tr>
</tbody>
</table>

CI: confidence interval; HLA: human leukocyte antigen; NE: not estimable; partial HLA matching: patient has ≥4 HLA alleles that match donor T cells used for CB-010 manufacturing

* Retrospective analysis of HLA allele matching for class I and class II antigens.

ANTLER Phase 1 clinical trial as of April 1, 2024 cutoff date, data collection ongoing.
Preliminary PFS with partial HLA matching has potential to be on par with an approved autologous CAR-T cell therapy

ANTLER LBCL patients with partial HLA matching and Yescarta ZUMA-7 trial

ANTLER 1st assessment at 28 days after CB-010 infusion
ZUMA-7 1st assessment at 50 days after randomization

FOR ILLUSTRATIVE PURPOSES ONLY: The results of other CAR-T cell therapies presented on this slide have been derived from publicly available reports of clinical trials run independently of Caribou and the data has been digitally recreated from publicly available original sources to compare approximations of the findings. The Company has not performed any head-to-head trials comparing any of these other CAR-T cell therapies with CB-010. As such, the results of these other clinical trials may not be comparable to clinical results for CB-010. The design of these other trials vary in material ways from the design of the clinical trials for CB-010, including with respect to patient populations, follow-up times, the clinical trial phase, and subject characteristics. As a result, cross-trial comparisons may have no interpretive value on the Company’s existing or future results. For further information and to understand these material differences, you should read the reports for the other trials at the sources included below.

Source: ZUMA-7, Locke et al, NEJM, 2022

PFS: progression free survival; 2L: second-line; 3L: third-line; LBCL: large B cell lymphoma; HLA: human leukocyte antigen; NE: not estimable; partial HLA matching: patient has ≥4 HLA alleles that match donor T cells used for CB-010 manufacturing

1 N=11 ≥4 HLA matching subset includes: 2L LBCL patients (N=10) and 3L LBCL patient (N=1).

ANTLER Phase 1 clinical trial as of April 1, 2024 cutoff date, data collection ongoing.
Patients in ANTLER all had aggressive r/r B-NHL

<table>
<thead>
<tr>
<th>Patient and disease characteristics</th>
<th>All treated (N=46)</th>
<th>Dose escalation (N=16)</th>
<th>Dose expansion (N=30)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, years, median (range)</td>
<td>65.0 (21-82)</td>
<td>66.0 (55-82)</td>
<td>63.0 (21-78)</td>
</tr>
<tr>
<td>Men, n (%)</td>
<td>36 (78.3)</td>
<td>14 (87.5)</td>
<td>22 (73.3)</td>
</tr>
<tr>
<td>ECOG performance status, n (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>21 (45.7)</td>
<td>6 (37.5)</td>
<td>15 (50.0)</td>
</tr>
<tr>
<td>1</td>
<td>25 (54.3)</td>
<td>10 (62.5)</td>
<td>15 (50.0)</td>
</tr>
<tr>
<td>Time since diagnosis, months, median (range)</td>
<td>10.6 (2.9-196.4)</td>
<td>29.0 (2.9-196.4)</td>
<td>9.5 (4.9-79.6)</td>
</tr>
<tr>
<td>NHL subtype, n (%)</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>LBCL</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>DLBCL</td>
<td>26 (56.5)</td>
<td>7 (43.8)</td>
<td>19 (63.3)</td>
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<tr>
<td>HGBL</td>
<td>8 (17.4)</td>
<td>2 (12.5)</td>
<td>6 (20.0)</td>
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<tr>
<td>tFL</td>
<td>4 (8.7)</td>
<td>0</td>
<td>4 (13.3)</td>
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<tr>
<td>PMBCL</td>
<td>2 (4.3)</td>
<td>1 (6.3)</td>
<td>1 (3.3)</td>
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<tr>
<td>Other B-NHL</td>
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<tr>
<td>MCL</td>
<td>3 (6.5)</td>
<td>3 (18.8)</td>
<td>0</td>
</tr>
<tr>
<td>FL(^1)</td>
<td>2 (4.3)</td>
<td>2 (12.5)</td>
<td>0</td>
</tr>
<tr>
<td>MZL</td>
<td>1 (2.2)</td>
<td>1 (6.3)</td>
<td>0</td>
</tr>
<tr>
<td>Prior systemic therapies, median (range)(^2)</td>
<td>1 (1-8)</td>
<td>2 (1-8)</td>
<td>1 (1-1)</td>
</tr>
<tr>
<td>IPI score at screening, n (%)(^3)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0 or 1</td>
<td>11 (23.9)</td>
<td>4 (25.0)</td>
<td>7 (23.3)</td>
</tr>
<tr>
<td>2</td>
<td>8 (17.4)</td>
<td>2 (12.5)</td>
<td>6 (20.0)</td>
</tr>
<tr>
<td>≥3</td>
<td>18 (39.1)</td>
<td>3 (18.8)</td>
<td>15 (50.0)</td>
</tr>
<tr>
<td>Maximum lesion diameter ≥7.5 cm, n (%)</td>
<td>10 (21.7)</td>
<td>3 (18.8)</td>
<td>7 (23.3)</td>
</tr>
<tr>
<td>LDH at screening, U/L, median (range)</td>
<td>216 (126-1799)</td>
<td>202 (126-710)</td>
<td>233.5 (140-1799)</td>
</tr>
<tr>
<td>Baseline LDH &gt; ULN, n (%)</td>
<td>23 (50.0)</td>
<td>5 (31.3)</td>
<td>18 (60.0)</td>
</tr>
<tr>
<td>LDH &gt;2 x ULN, n (%)</td>
<td>7 (15.2)</td>
<td>1 (6.3)</td>
<td>6 (20.0)</td>
</tr>
</tbody>
</table>

DLBCL: diffuse large B cell lymphoma; FL: follicular lymphoma; HGBL: high-grade B cell lymphoma; MCL: mantle cell lymphoma; MZL: marginal zone lymphoma; PMBCL: primary mediastinal large B cell lymphoma; IPI: International Prognostic Index; LDH: lactate dehydrogenase; ULN: upper limit of normal

\(^1\) Aggressively behaving, with POD24 (high risk).

\(^2\) Patients are CD19 CAR-T naïve.

\(^3\) IPI scores were not recorded for all patients.

As of April 1, 2024 cutoff date.
CB-010 has generally well-tolerated safety profile
No Grade ≥3 CRS, no GvHD observed (N=46)

<table>
<thead>
<tr>
<th></th>
<th>All CB-010 treated (N=46)</th>
<th>Yescarta (N=170)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Any grade (n, %)</td>
<td>Grade ≥3 (n, %)</td>
</tr>
<tr>
<td>Prolonged cytopenias</td>
<td>9 (20)</td>
<td>9 (20)</td>
</tr>
<tr>
<td>CRS</td>
<td>26 (57)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Infections</td>
<td>22 (47)</td>
<td>10 (22)</td>
</tr>
<tr>
<td>ICANS</td>
<td>10 (22)</td>
<td>3 (7)</td>
</tr>
<tr>
<td>Hemophagocytic lymphohistiocytosis (HLH)</td>
<td>1 (2)</td>
<td>0</td>
</tr>
<tr>
<td>GvHD</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

FOR ILLUSTRATIVE PURPOSES ONLY: The results of other CAR-T cell therapies presented on this slide have been derived from publicly available reports of clinical trials run independently of Caribou. The Company has not performed any head-to-head trials comparing any of these other CAR-T cell therapies with CB-010. As such, the results of these other clinical trials may not be comparable to clinical results for CB-010. The design of these other trials vary in material ways from the design of the clinical trials for CB-010, including with respect to patient populations, follow-up times, the clinical trial phase, and subject characteristics. As a result, cross-trial comparisons may have no interpretive value on the Company's existing or future results. For further information and to understand these material differences, you should read the reports for the other trials at the sources included below.

CB-010 has generally well-tolerated safety profile
No Grade ≥3 CRS, no GvHD observed (N=46)

For illustrative purposes only: The results of other CAR-T cell therapies presented on this slide have been derived from publicly available reports of clinical trials run independently of Caribou. The Company has not performed any head-to-head trials comparing any of these other CAR-T cell therapies with CB-010. As such, the results of these other clinical trials may not be comparable to clinical results for CB-010. The design of these other trials vary in material ways from the design of the clinical trials for CB-010, including with respect to patient populations, follow-up times, the clinical trial phase, and subject characteristics. As a result, cross-trial comparisons may have no interpretive value on the Company’s existing or future results. For further information and to understand these material differences, you should read the reports for the other trials at the sources included below.

CRS: cytokine release syndrome; GvHD: graft-versus-host disease; ICANS: immune effector cell-associated neurotoxicity syndrome; NR: not reported
1 Prolonged cytopenias are defined as grade 3 or higher events lasting beyond 30 days following CB-010 infusion; 37/46 (80%) recovered from cytopenias to grade ≤2 by day 35 post CB-010 treatment.
2 Prolonged cytopenias of grade 3 or higher that were present at or after 30 days from Yescarta infusion.
3 Median time of onset was 3 days (range 0-22) and median duration was 3 days (range 1-19).
4 Infection events reported were on or after CB-010 infusion, with highest grade reported per patient; median onset 8 days (range 0-279) and median duration is 14 days (range 1-239).
5 Median time of onset was 7.5 days (range 6-34) and median duration was 2 days (range 1-27).
6 Hemophagocytic lymphohistiocytosis (HLH) 1 (2) 0 NR NR
8 Grade 3 and 1 Grade 4; all resolved with supportive care. Median time of onset was 8 days and median duration 2 days.
ANTLER Phase 1 clinical trial as of April 1, 2024 cutoff date, data collection ongoing.
Source: ZUMA-7, Locke et al, NEJM, 2022 (prolonged cytopenia at 30 days); Westin et al, NEJM, 2023 (CRS, infections, ICANS/neurological events)
CB-010 ANTLER efficacy assessment for patients with ≥4 HLA matching
(N=13)

As of April 1, 2024, data collection ongoing, efficacy based on Lugano criteria.

CR: complete response
PR: partial response
SD: no response/stable disease
PD: progressive disease
Death

SINGLE CB-010 DOSE

Median PFS
14.4 months

CR₁ rate
46%

Median duration of CR
Not reached

DLBCL: diffuse large B cell lymphoma; CR: complete response; HGBL: high-grade B cell lymphoma; PFS: progression free survival; PMBCL: primary mediastinal large B cell lymphoma; tFL: transformed DLBCL from follicular lymphoma; PLoT: prior lines of therapy

¹46% CR rate measures the number of patients (6 of 13) achieving a CR at any time point after treatment with CB-010.

KOL discussion CB-010 ANTLER Phase data | June 2024
©2024 Caribou Biosciences, Inc.
CB-010 ANTLER efficacy assessment by all patients and LBCL subgroups

<table>
<thead>
<tr>
<th>Endpoints</th>
<th>All patients (N=46)</th>
<th>LBCL (N=40)</th>
<th>2L LBCL 80M (N=20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overall response rate (ORR)¹</td>
<td>35 (76%)</td>
<td>29 (73%)</td>
<td>15 (75%)</td>
</tr>
<tr>
<td>DoR, median months (range)</td>
<td>5 (1-23+)</td>
<td>2 (1-23+)</td>
<td>5 (1-20+)</td>
</tr>
<tr>
<td>Complete response (CR) rate¹</td>
<td>21 (46%)</td>
<td>17 (43%)</td>
<td>10 (50%)</td>
</tr>
<tr>
<td>Duration of CR, Median months (range)</td>
<td>7 (1-23+)</td>
<td>7 (1-23+)</td>
<td>NR (1-12+)</td>
</tr>
<tr>
<td>6-month PFS</td>
<td>35%</td>
<td>28%</td>
<td>38%</td>
</tr>
<tr>
<td>PFS, median months (range)</td>
<td>3 (1-24+)</td>
<td>3 (1-24+)</td>
<td>3.5 (1-21+)</td>
</tr>
</tbody>
</table>

¹ + censored observation

As of April 1, 2024 cutoff date.
## CB-010 ANTLER efficacy assessment with and without partial HLA matching

<table>
<thead>
<tr>
<th>Endpoints</th>
<th>All patients ≤3 HLA matches (N=33)</th>
<th>All patients ≥4 HLA matches (N=13)</th>
<th>LBCL ≥4 HLA matches (N=11)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Overall response rate (ORR)</strong></td>
<td>23 (69%)</td>
<td>12 (92%)</td>
<td>10 (91%)</td>
</tr>
<tr>
<td>Duration of response (DoR), median months (range)</td>
<td>2.0 (1-23+)</td>
<td>13.5 (1-23+)</td>
<td>NR (1-15+)</td>
</tr>
<tr>
<td><strong>Complete response (CR) rate</strong></td>
<td>15 (45%)</td>
<td>6 (46%)</td>
<td>4 (36%)</td>
</tr>
<tr>
<td>Duration of CR, median months (range)</td>
<td>5.0 (1-23+)</td>
<td>NR (5-23+)</td>
<td>NR (5-15+)</td>
</tr>
<tr>
<td><strong>6-month PFS</strong></td>
<td>25%</td>
<td>62%</td>
<td>53%</td>
</tr>
<tr>
<td><strong>PFS, median months (range)</strong></td>
<td>2.8 (1-24+)</td>
<td>14.4 (2-24+)</td>
<td>NR (2-16+)</td>
</tr>
</tbody>
</table>

+ censored observation

HLA: human leukocyte antigen; partial HLA matching: patient has ≥4 HLA alleles that match donor T cells used for CB-010 manufacturing; NR: not reached ANTLER Phase 1 clinical trial as of April 1, 2024 cutoff date, data collection ongoing.
Partial HLA matching improves exposure of CB-010

Pharmacokinetic (PK) exposure

- Peak expansion ($C_{max}$) occurred 7 to 10 days post infusion
- Persistence was observed up to ~30 days
- PK consistent for three dose levels evaluated

Partial HLA matching impact on PK

- Higher numbers of HLA matched alleles demonstrate more expansion and persistence vs. lower numbers

LLOQ: lower limit of quantification
Mean values represented by dots with standard error shown; values below LLOQ converted to 0; Includes all available data from the V2 ddPCR assay; visits up to D28 shown; D0 values represent pre-infusion level set to 0.
N=35 total number of patients included in PK analysis based on samples analyzed as of data cutoff of April 1, 2024.
Partial HLA matching will not impact time to treatment

How does HLA matching work?

- Human leukocyte antigens (HLAs) help the immune system identify “self” from “non-self”
- Patient’s immune cells recognize allogeneic CAR-T cells as “non-self” and initiate rejection

Partial HLA matching and DSA screening for ANTLER and GALLOP Phase 1 trials

- HLA typing and DSA analysis occur within screening timeline and will not impact time to receive treatment
- Partial HLA matching could result in enhanced outcomes for patients¹

¹ Based on data from the ongoing ANTLER Phase 1 trial in r/r B-NHL and to be confirmed in the ANTLER and GALLOP Phase 1 trials.
CB-010 is an off-the-shelf CAR-T cell therapy that is easily matched to patients

~13 different batches

~90% of 2L LBCL patients for planned Phase 3 clinical trial\(^1\) are expected to receive \(\geq 4\) HLA matched product

Only a small number of manufacturing batches are needed to provide partially HLA matched CB-010 to \(~90\)% of patients

HLA: human leukocyte antigen; partial HLA match: patient has \(\geq 4\) HLA alleles that match donor T cells used for CB-010 manufacturing

\(^1\) Planned pivotal Phase 3 intends to enroll CD19 naïve 2L LBCL patients who will be dosed with best matched CB-010
Advancing CB-010 to establish new standard of care for 2L LBCL and broaden patient access

- With partial HLA matching, safety, efficacy, durability has the potential to rival approved autologous CAR-T cell therapies\(^1\)
- Generally well-tolerated safety profile
- Off-the-shelf, readily-available single dose cell therapy
- RMAT and Fast Track designations enable FDA interactions
- Safety and efficacy profile supports clinical development for 2L LBCL and lupus patients and in outpatient setting

**Progression free survival**

14.4 months
median (95% CI: 1.7-NE)
all patients with $\geq 4$ HLA matches

NR
median (95% CI: 1.6-NE)
all LBCL patients with $\geq 4$ HLA matches

---

2L: second-line; LBCL: large B cell lymphoma; PFS: progression free survival; HLA: human leukocyte antigen
\(^1\)To be confirmed with additional clinical data.
ANTLER Phase 1 clinical trial as of April 1, 2024 cutoff date, data collection ongoing.
CB-010

Allogeneic anti-CD19 CAR-T cell therapy with a PD-1 knockout for lupus
Single dose of CB-010 results in extended B cell aplasia and rapid recovery of immune cells

B cell, T cell, and NK cell depletion and recovery

- CB-010 specifically targets B cells, resulting in extended B cell aplasia for ~114 days
- B cells recover to normal levels by ~268 days
- T cells and NK cells recovered ~3 weeks after LD regimen

LD: lymphodepletion; LLOQ: lower limit of quantification
Baseline B cells absolute levels calculated with samples of 10 cells/µl or above.
### CB-010 duration of B cell aplasia is similar to lupus case studies

<table>
<thead>
<tr>
<th>Duration of B cell aplasia</th>
<th>Days</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CB-010</strong></td>
<td></td>
</tr>
<tr>
<td>N=44</td>
<td>114</td>
</tr>
<tr>
<td>Mean (IQR 42-150)</td>
<td></td>
</tr>
</tbody>
</table>

| **Müller et al**          |      |
| N=14\(^1\)               | 112  |
| Mean (IQR 72-153)         |      |

---

\(^1\) Patient population included severe SLE (8 patients), idiopathic inflammatory myositis (3 patients), or systemic sclerosis (3 patients) who received a single infusion of CD19 chimeric antigen receptor (CAR) T cells after preconditioning with fludarabine and cyclophosphamide. Source: Müller et al., NEJM, 2024; B cell aplasia defined as being below the limit of quantification.
CB-010 GALLOP Phase 1 trial design

Eligibility and matching
- Non-responsive to glucocorticoids and have tried and failed at least 2 defined immunosuppressive therapies
- Excludes cardiac and CNS involvement
- Partial HLA matching and absence of baseline DSAs

Patient cohorts
- Cohort 1: Lupus nephritis (LN)
  Renal SLEDAI ≥ 8 or Class III/IV glomerular nephritis
- Cohort 2: Extrarenal lupus (ERL)
  SLEDAI ≥ 8

Treatment and objective
- Single dose level of CB-010 following LD
- Primary endpoint: safety

Cohort 1: Lupus nephritis (LN)
Renal SLEDAI ≥ 8 or Class III/IV glomerular nephritis

Cohort 2: Extrarenal lupus (ERL)
SLEDAI ≥ 8

S SINGLE DOSE CB-010
- 5 to -3 DAYS
DAY 0 28 DAYS 3 MONTHS 6 MONTHS 9 MONTHS 12 MONTHS

Safety and tolerability
Response assessment

Fludarabine
25 mg/m²/d x 3 days
Days -5, -4, -3
Cyclophosphamide
20 mg/kg/d x 2 days
Days -4 and -3

Cy: cyclophosphamide; DSAs: donor-specific antibodies; Flu: fludarabine; HLA: human leukocyte antigen; LD: lymphodepletion; SLEDAI: Systemic Lupus Erythematosus Disease Activity Index
CB-011

Allogeneic anti-BCMA CAR-T cell therapy with immune cloaking for r/r multiple myeloma (MM)
CB-011: anti-BCMA allogeneic CAR-T cell therapy with immune cloaking to blunt rejection

Armored with 4 genome edits

1. *TRAC* gene knockout (KO)
   - Eliminates TCR expression, reduces GvHD risk

2. Humanized anti-BCMA CAR site-specifically inserted into *TRAC* gene
   - Eliminates random integration, targets tumor antigen

3. B2M gene KO
   - Reduces HLA class I presentation and T cell-mediated rejection

   - Blunts NK cell-mediated rejection

1st CAR-T in the clinic with immune cloaking using a B2M KO and B2M–HLA-E-peptide fusion insertion

Cas12a chRDNA editing for reduced off-target editing and enhanced insertion rates

Patented, potent, humanized anti-BCMA scFv with a 4-1BB costimulatory domain

---

1. To Caribou’s knowledge
2. Four U.S. patents granted to date covering CB-011 scFv
CB-011 editing strategy designed to reduce both T cell- and NK cell-mediated rejection

- B2M KO removes all endogenous HLA class I presentation to reduce T cell-mediated rejection
- B2M–HLA-E-peptide fusion insertion blunts NK cell-mediated rejection
- The Cas12a chRDNA editing platform achieves high insertion efficiencies facilitating the insertion of the B2M–HLA-E-peptide fusion and CAR into different genomic locations
**B2M KO and B2M-HLA-E fusion strategy protects CB-011 CAR-T cells from NK and T cell-mediated lysis**

**B2M-HLA-E fusion enables CB-011 cells to resist killing by NK cells**

CAR-T cell co-incubation with NK-92 cells*

*In vitro cytotoxicity measured 24 hours after co-incubation


**B2M KO enables CB-011 cells to resist killing by T cells**

CAR-T cell co-incubation with PBMC-derived CD8⁺ T cells*

*In vitro cytotoxicity measured 24 hours after co-incubation
CB-011 enhanced long-term survival in preclinical studies

CB-011 led to statistically significant and longer survival of tumor-bearing mice relative to an alternative anti-BCMA CAR-T cell therapy after a single dose

- Established subcutaneous MM tumor xenograft
- Single dose CAR-T cell treatment

CB-011 CaMMouflage Phase 1 trial design

**Patients with r/r MM**
- ≥3 prior lines of therapy, including a PI, an IMiD, and an anti-CD38 antibody
- Exclusions: prior CAR-T cell therapy and/or BCMA-targeted therapy within last 3 months

**Part A: 3+3 dose escalation**
- Objective: safety, determine MTD, RDE

**Part B: dose expansion**
- Objective: antitumor response, RP2D

---

**r/r MM**

**Lymphodepletion**

<table>
<thead>
<tr>
<th>-5 to -3 DAYS</th>
<th>DAY 0</th>
<th>28 DAYS</th>
<th>3 MONTHS</th>
<th>6 MONTHS</th>
<th>9 MONTHS</th>
<th>12 MONTHS</th>
</tr>
</thead>
<tbody>
<tr>
<td>CB-011</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**SINGLE DOSE**

- Cyclophosphamide
- Fludarabine

**Safety and tolerability**

**Response assessment**

**Dose level 1**: 50x10^6 CAR-T cells (N=3, completed)

**Dose level 2**: 150x10^6 CAR-T cells (N=3, completed)

**Dose level 3**: 450x10^6 CAR-T cells (enrolling patients)
CB-012

Allogeneic anti-CLL-1 CAR-T cell therapy with a PD-1 knockout and immune cloaking for r/r acute myeloid leukemia (AML)
CB-012: anti-CLL-1 allogeneic CAR-T cell therapy with a PD-1 knockout and immune cloaking

Armored with 5 genome edits

1. TRAC gene knockout (KO)
   - Eliminates TCR expression, reduces GvHD risk

2. Human anti-CLL-1 CAR site-specifically inserted into TRAC gene
   - Eliminates random integration, targets tumor antigen

3. PD-1 KO for enhanced antitumor activity
   - Potentially better therapeutic index via initial tumor debulking

4. B2M gene KO
   - Reduces HLA class I presentation and T cell-mediated rejection

5. B2M-HLA-E-peptide fusion site-specifically inserted into B2M gene
   - Blunts NK cell-mediated rejection

1st CAR-T with checkpoint inhibition and immune cloaking (PD-1 KO, B2M KO + B2M-HLA-E-peptide fusion) to enter the clinic

Cas12a chRDNA editing for reduced off-target editing and enhanced insertion rates

Potent, fully human anti-CLL-1 scFv with a CD28 costimulatory domain

1 To Caribou’s knowledge
2 Anti-CLL-1-specific scFv exclusively licensed from Memorial Sloan Kettering Cancer Center for allogeneic cell therapies
CB-012 significantly reduced tumor burden and increased overall survival in preclinical studies

Single dose of CB-012 **significantly reduced tumor burden** over a longer duration compared to vehicle treatment in an AML xenograft model.

Addition of PD-1 KO in genome-editing strategy **increased overall survival** compared to control CAR-T cell without PD-1 KO.

---

1 Orthotopic engraftment of HL-60 CLL-1-expressing AML model in NSG mice
2 Orthotopic engraftment of U937 CLL-1- and PD-L1-expressing cell line in NSG mice
CB-012 AMpLify Phase 1 trial design

**Patients with r/r AML**
- Relapsed or refractory AML patients should have received at least 1 but not more than 3 prior lines of therapy
- Patients with prior allo or auto SCT are allowed
- Exclusions: prior CAR-T cell therapy and/or CLL-1-targeted therapy

**Part A: 3+3 dose escalation - enrolling**
- Objective: safety, determine MTD/RDE

**Part B: dose expansion**
- Objective: antitumor response, determine RP2D, safety

---

**r/r AML**

**Lymphodepletion**

-5 to -3 DAYS  
DAY 0  
28 DAYS  
3 MONTHS  
6 MONTHS  
9 MONTHS  
12 MONTHS

- Cyclophosphamide (750 mg/m²/d)  
- Fludarabine (30 mg/m²/d)

**CB-012**

**Dose level 1:** 25 x10⁶ CAR-T cells (enrolling patients)

**SINGLE DOSE**

AML: acute myeloid leukemia; SCT: stem cell transplant; MTD: maximum tolerated dose; RDE: recommended dose or doses for expansion; RP2D: recommended phase 2 dose
# Upcoming clinical catalysts

<table>
<thead>
<tr>
<th>Program</th>
<th>Clinical milestone</th>
<th>Expected timing</th>
</tr>
</thead>
<tbody>
<tr>
<td>CB-010 2L LBCL</td>
<td>Present initial data on partial HLA matching (~20 patients, some outpatient), CD19 relapsed (~10 patients) from the ANTLER Phase 1 clinical trial</td>
<td>H1 2025</td>
</tr>
<tr>
<td></td>
<td>Initiate pivotal Phase 3 trial</td>
<td>H2 2025</td>
</tr>
<tr>
<td>CB-011 r/r MM</td>
<td>Present initial dose escalation data from CaMMouflage Phase 1 trial</td>
<td>YE 2024</td>
</tr>
<tr>
<td>CB-010 LN/ERL</td>
<td>Initiate GALLOP Phase 1 trial</td>
<td>YE 2024</td>
</tr>
</tbody>
</table>
Thank you

https://cariboubio.com
info@cariboubio.com
CB-010 is generally well tolerated
Treatment-emergent adverse events (TEAE\(^1\)) in ≥20% of all patients

<table>
<thead>
<tr>
<th>System organ class, n (%)</th>
<th>Preferred term, n (%)</th>
<th>All treated (N = 46)</th>
<th>LBCL subgroup (N=40)</th>
<th>2L LBCL RP2D subgroup (N=20)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Any grade</td>
<td>Grade ≥3</td>
<td>Related grade ≥3</td>
<td>Any grade</td>
</tr>
<tr>
<td>Any TEAE</td>
<td>46 (100)</td>
<td>41 (89)</td>
<td>23 (50)</td>
<td>40 (100)</td>
</tr>
<tr>
<td>Thrombocytopenia</td>
<td>30 (65)</td>
<td>29 (63)</td>
<td>15 (33)</td>
<td>26 (65)</td>
</tr>
<tr>
<td>Anemia</td>
<td>27 (59)</td>
<td>24 (52)</td>
<td>10 (22)</td>
<td>24 (60)</td>
</tr>
<tr>
<td>Neutropenia</td>
<td>22 (48)</td>
<td>19 (41)</td>
<td>7 (15)</td>
<td>18 (45)</td>
</tr>
<tr>
<td>White blood cell count decreased</td>
<td>15 (33)</td>
<td>14 (30)</td>
<td>6 (13)</td>
<td>14 (35)</td>
</tr>
<tr>
<td>CRS</td>
<td>26 (57)</td>
<td>0</td>
<td>0</td>
<td>23 (58)</td>
</tr>
<tr>
<td>Infections</td>
<td>22 (48)</td>
<td>10 (22)</td>
<td>4 (9)</td>
<td>19 (48)</td>
</tr>
<tr>
<td>Hypokalemia</td>
<td>11 (24)</td>
<td>0</td>
<td>0</td>
<td>9 (23)</td>
</tr>
<tr>
<td>Pyrexia</td>
<td>11 (24)</td>
<td>0</td>
<td>0</td>
<td>10 (25)</td>
</tr>
<tr>
<td>ICANS</td>
<td>10 (22)</td>
<td>3 (7)</td>
<td>3 (7)</td>
<td>8 (20)</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>10 (22)</td>
<td>0</td>
<td>0</td>
<td>7 (18)</td>
</tr>
</tbody>
</table>

Five patients died due to adverse events following CB-010 infusion (4 unrelated, 1 possibly related\(^2\) to CB-010)

---

\(^1\) TEAEs are defined as adverse events (AEs) with a start date on or after the CB-010 infusion date.

\(^2\) One death possibly related to CB-010 per investigator due to complications of a bladder perforation in the context of BK virus hemorrhagic cystitis. As of April 1, 2024 cutoff date.
CB-010 ANTLER efficacy assessment all patients

As of April 1, 2024, data collection ongoing, efficacy based on Lugano criteria. *Denotes patient that did not have Day 28 efficacy scan.
Potential to address high unmet medical need in 2L LBCL

**LBCL patient treatment journey**
(U.S. incidence 2022)

- **Diagnosed** ~32.4K patients
- **1L** ~30.8K patients
- **2L** ~10.0K patients
- **3L** ~4.4K patients

ANTLER dose expansion
Allogeneic CAR-T cell manufacturing process overview for CB-010

Caribou’s process development team created the manufacturing process and transferred it to a CMO to generate phase 1 cGMP clinical material.

cGMP starting material
Genetic modification via chRDNA and AAV
Gene-modified CAR-T cells
In vitro expansion of edited donor T cells
Removal of residual TCR+ cells
Final formulation & cryopreservation

Healthy donor leukapheresis

AAV
Cas9
chRDNA

TCR
PD1
Anti-CD19 CAR

Anti-TCR microbeads

Treatment

TRAC
PDCD1

Anti-CD19 CAR

Chr 14
Chr 2

Healthy donor leukapheresis

AAV
Cas9
chRDNA

TCR
PD1
Anti-CD19 CAR

Anti-TCR microbeads

Treatment

TRAC
PDCD1

Anti-CD19 CAR

Chr 14
Chr 2
CB-010 is an allogeneic CAR-T cell therapy that targets autoantibody-producing B cells

- **Engineered for improved activity**
  - chRDNA genome editing enables **precision engineering** and **reduced off-target edits**
  - CB-010 is **engineered with a PD-1 KO**\(^1\) to potentially enhance anti-B cell activity and may drive **sustained remission**

- **Encouraging clinical data**
  - Encouraging initial **safety and efficacy** demonstrated for CB-010 in ANTLER Phase 1 trial
  - ANTLER **B cell depletion is on par** with depletion data published on autologous CAR-T cells in lupus\(^2\)

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1. To Caribou’s knowledge, CB-010 is the first allogeneic CAR-T in the clinic with checkpoint disruption via PD-1 KO
Lupus is a chronic, inflammatory autoimmune disease driven by autoantibody-producing B cells

Lupus is caused by B cell production of autoantibodies that drive damage of healthy tissue

Lupus can cause widespread organ damage, increase cardiovascular risk, and significantly impair patient quality of life

Urgent unmet need for new treatment options that can offer sustained, drug-free remission


Lupus is a chronic disease affecting ~320,000 individuals in the US1
CB-010 demonstrated differentiated, long-term antitumor activity in preclinical studies

A single dose of CB-010 resulted in profound tumor regression of metastatic CD19+ tumor xenografts and led to a significantly longer antitumor response and survival vs. conventional CD19-specific allogeneic CAR-T cells (expressing PD-1)

- NALM-6/PD-L1+ B-ALL tumors were established by IV engraftment for 23 days (Day -1)
- A single dose treatment was administered by IV on Day 24 (PBS or $10^7$ cells where indicated)