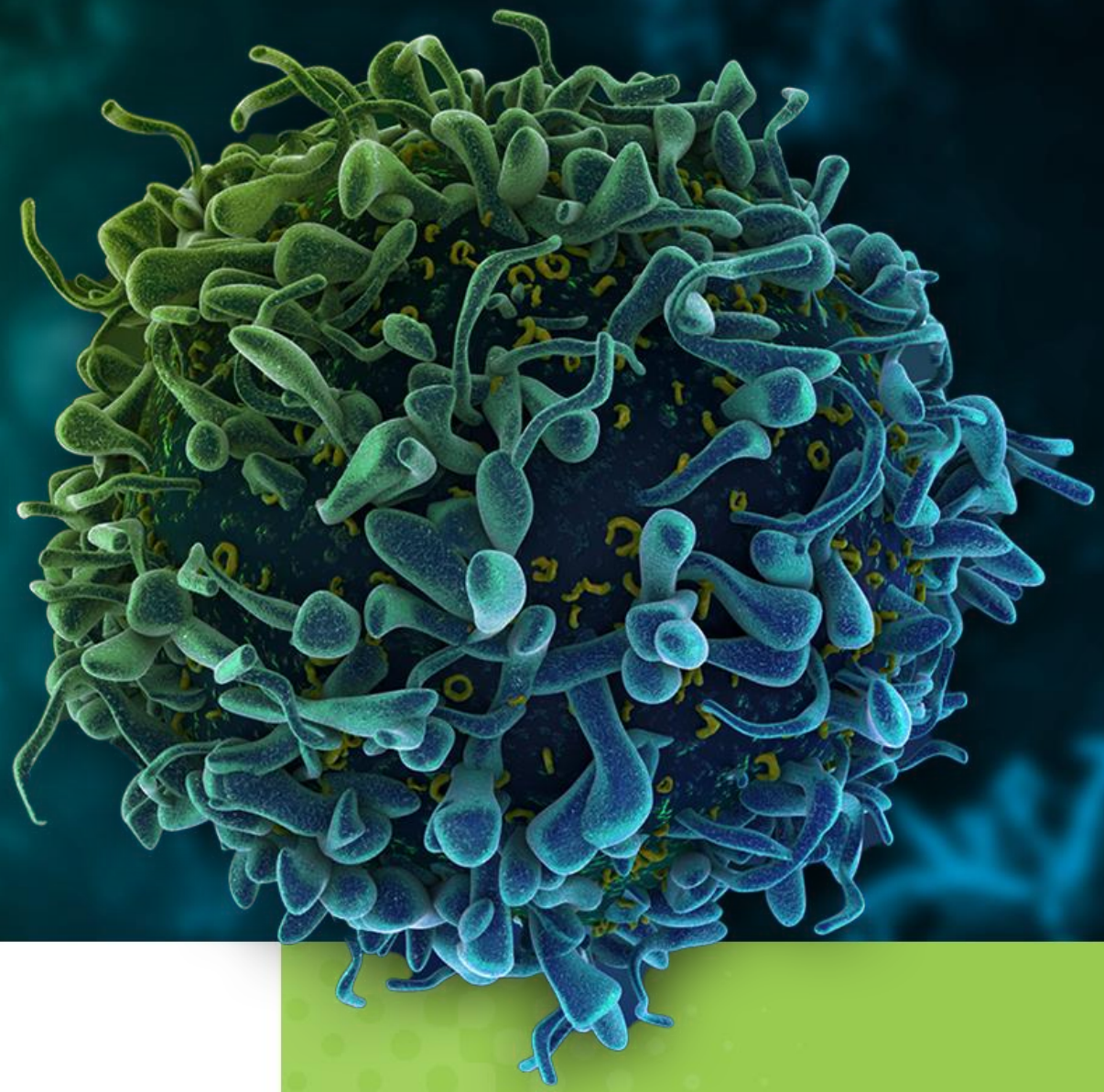
Cullinan Therapeutics

Autoimmune Disease Strategy

April 16, 2024



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On Today's Call

Nadim Ahmed
Chief Executive Officer

Jeff Jones, MD, MPH, MBA
Chief Medical Officer

Patrick Baeuerle, PhD
Chief Scientific Advisor

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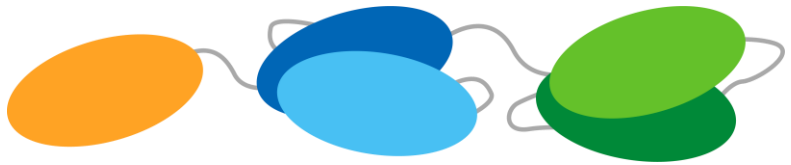
Announcing Cullinan Therapeutics

- New Company name, Cullinan Therapeutics, reflects expansion of portfolio into autoimmune diseases.
- CLN-978 to be developed exclusively in autoimmune diseases, with system lupus erythematosus (SLE) as a first indication.
- New observations from the B-NHL study show rapid, deep and sustained B cell depletion and clinical activity.
- CLN-978 has the potential to be a first-in-class, off-the-shelf, disease-modifying treatment with a favorable safety profile in autoimmune disease.
- Recently announced equity finance transaction adds an additional $280M to our balance sheet and extends our cash runway into 2028.

CLN-978 Opportunity in Autoimmune Diseases

CLN-978
CD19 x CD3 T Cell Engager



α-HSA (VHH)

α-CD19 (scFv)

α-CD3 (scFv)

High affinity, enabling lysis of target cells with low CD19 target expression

Albumin binding for extended serum half-life

CD3 binder for T cell redirected B cell lysis

CLN-978 program update

Development of CD19xCD3 T cell engager in autoimmune diseases

- IND submission planned for SLE in 3Q 2024
- Planning for other autoimmune disease indications
- Enrollment in B-NHL study has been discontinued

Initial proof of concept from B-NHL clinical trial

- Clinical response achieved at first dose level
- Observed rapid, deep and sustained B cell depletion
- Favorable safety with only Grade 1 CRS; No ICANS

Strong potential for differentiation

- Potent modality due to broad T cell engagement
- Potential advantages over CAR T cell therapy
- Subcutaneous administration
- CD19 target has potential advantages over other targets (CD20, BCMA)

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HSA = human serum albumin binding domain, SLE = systemic lupus erythematosus,
CRS = cytokine release syndrome, ICANS = immune effector cell-associated neurotoxicity syndrome

Cullinan Oncology Pipeline Remains on Track

Program Modality/MOA	IND-Enabling	Phase 1	Phase 2	Phase 3	Status	Geographic Rights
CLN-619 Anti-MICA/B antibody	Pan-cancer				Initial combo data and monotherapy update in 2Q24; Disease specific expansion data in 1H25	or its subsidiary owns worldwide rights
CLN-978 CD19xCD3 T-cell engager	Systemic lupus erythematosus				IND submission expected in 3Q24	owns worldwide rights
Zipalertinib (CLN-081/TAS6417) EGFRex20ins inhibitor	NSCLC with exon 20 insertion mutations 2+ line NSCLC with exon 20 insertion mutations frontline				Pivotal Phase 2b 2L+ study enrolled by YE24; Phase 3 1L study actively enrolling	holds US co-development/-commercialization rights with TAIHO ONCOLOGY
CLN-049 FLT3xCD3 T-cell engager	R/R AML, MDS				Clinical update from ongoing Phase 1 study in 2H24	or its subsidiary owns worldwide rights
CLN-418 B7H4x41BB bispecific immune activator	Multiple solid tumors				Clinical update from ongoing Phase 1 study in 2H24	owns U.S. rights
CLN-617 Collagen-binding IL-12 and IL-2 fusion protein	Pan-cancer				Phase 1 study ongoing	or its subsidiary owns worldwide rights

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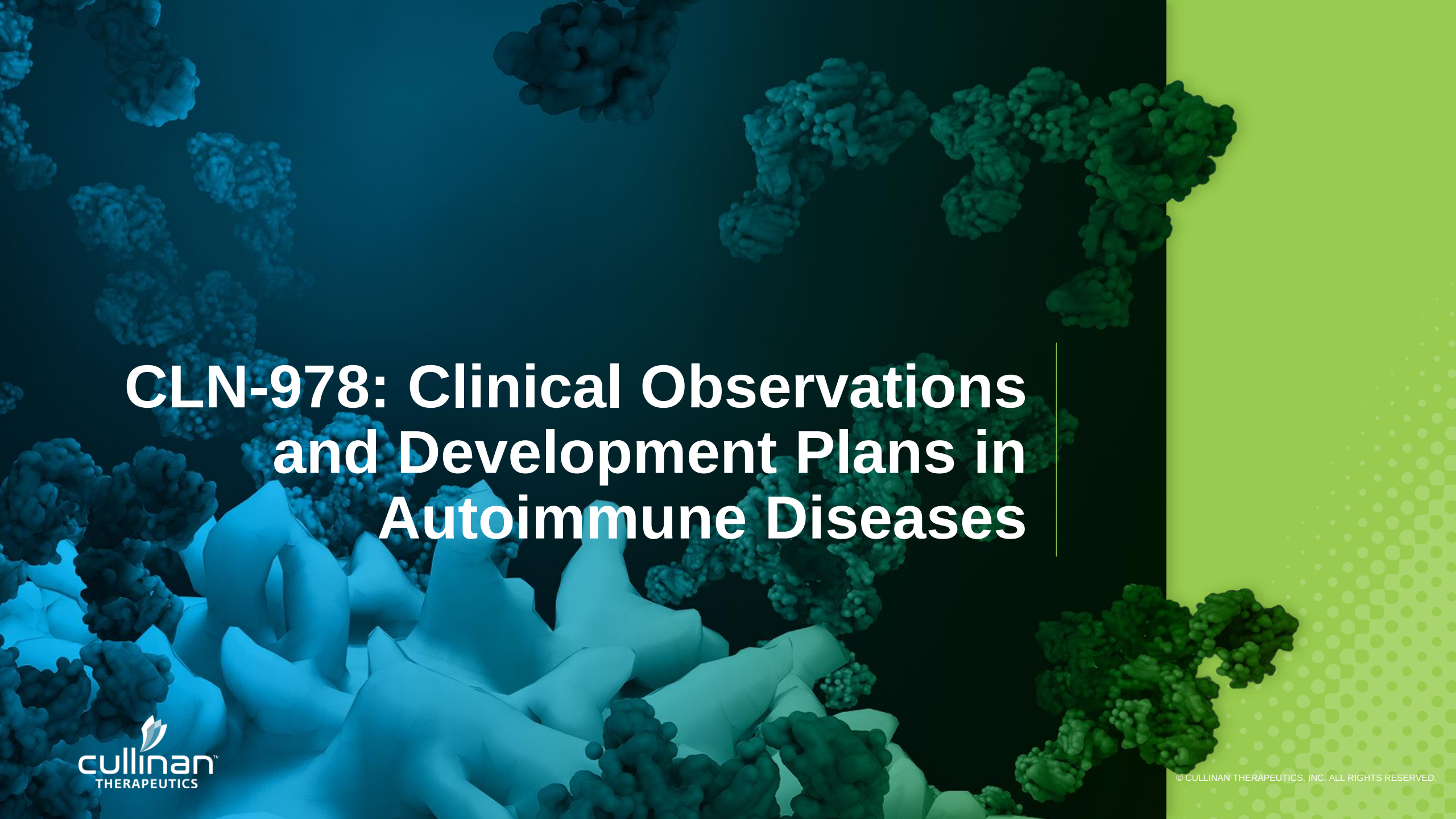
CLN-978: Clinical Observations and Development Plans in Autoimmune Diseases



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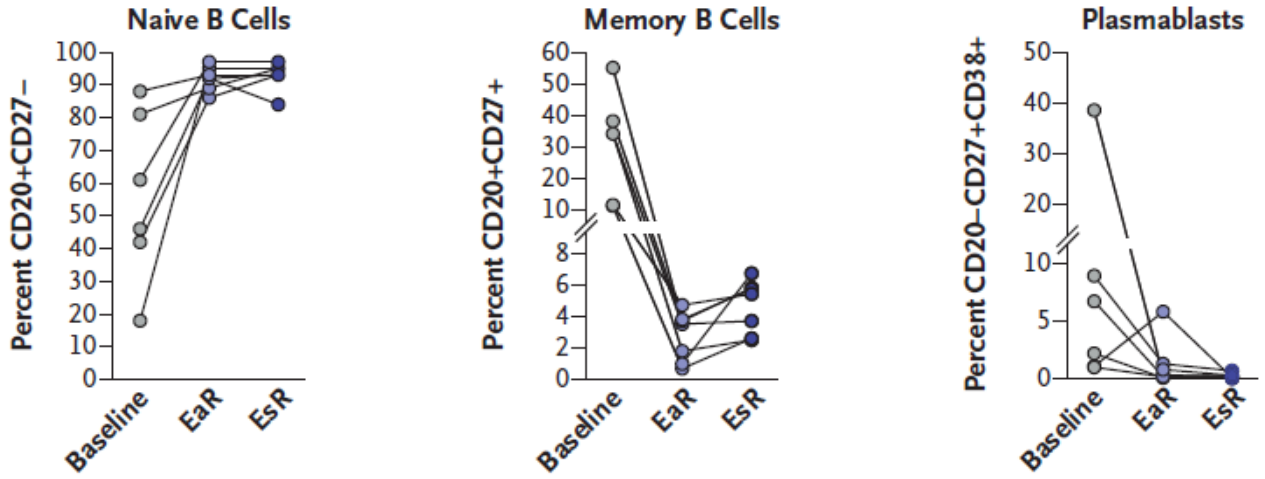
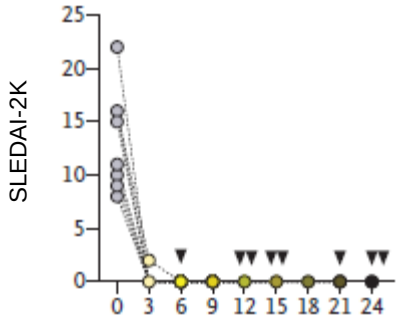
CD19 CAR-T cell therapy generated immune system reset and durable, treatment-free remissions in autoimmune patients

Immune system reset: selected SLE PD data

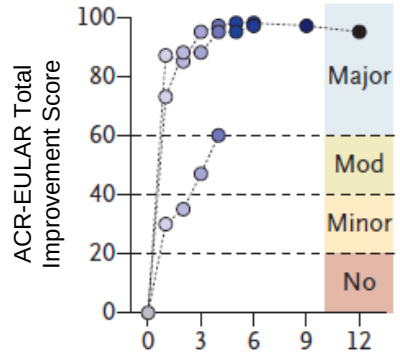


Treatment free remissions in 3 autoimmune disease settings

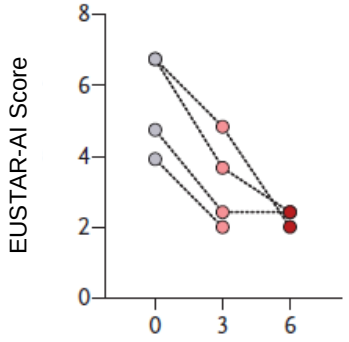
SLE patients (n=8)



IIM patients (n=3)



SSc patients (n=4)



Observations

- Mueller et al. (2024) treated 15 autoimmune patients (SLE, IIM, & SSc)[1] with autologous CD19 CAR T
- SLE and IIM patients had complete resolution of disease symptoms; SSc patients reduced severity of skin and lung disease
- All patients successfully stopped immunosuppressive medication without having relapses or worsening disease
- Supported by deep B cell depletion, followed by immune reset and sustained diminution of autoantibodies
- Sustained drug-free remission are highly unlikely to be induced by lymphodepletion alone (e.g., some pts w/ prior chemotherapy)

Muller, F. et al. NEJM 2024. 1) SLE = Systemic Lupus Erythematosus, IIM = idiopathic inflammatory myositis, SSc = systemic sclerosis

CD19 CAR T was generally well tolerated across autoimmune indications studied; however infectious complications were frequent

Class Specific Adverse Events	Grade 1	Grade 2+
ICANS	1/15	0/15
CRS*	10/15	1/15

*6/15 patient received tocilizumab

- Safety profile generally favorable versus observed safety of CD19 CAR T for ALL or B-NHL
- Infectious complications were common during the 12 months after CD19 CAR T
  - 14/15 patients experienced urinary or respiratory tract infections, including 1 case of pneumonia requiring hospitalization
  - 4/15 experience 2 or more infection episodes
  - Two patients experienced herpes zoster reactivation

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Muller, F. et al. NEJM 2024. CRS = cytokine release syndrome. ICANS = immune effector cell-associated neurotoxicity syndrome
ALL = acute lymphoblastic leukemia

CAR T cell therapy: Promising outcomes but multiple challenges may limit broad uptake in autoimmune diseases

Cell therapy limitations

- Available cellular therapies all require lymphodepleting chemotherapy, which has been associated with increased risk for infection, infertility, and secondary malignancies
- FDA has mandated boxed warnings across approved products highlighting the risk for secondary malignancies related to the CAR T cell therapy itself
- Complex manufacturing processes can introduce treatment delays for patients
- Reimbursement challenges continue to limit provider uptake for existing indications
- Treatment limited to specialized centers certified to provide CAR T cell therapies
- Prohibitive logistical and economic challenges will likely prevent retreatment upon relapse

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TCE and CAR-T cells have comparable efficacy in similar NHL patient populations*

	YESCARTA[1] Axi-cel	**KYMRIAH[2]** Tisa-cel	**LUNSUMIO[3]** Mosunetuzumab
Target/Modality	CD19 CAR T	CD19 CAR T	CD20 TCE (IV)
Study / pt pop	ZUMA-5 Phase 2 R/R FL 3L+	ELARA Phase 3 R/R FL 3L+	GO29781 Phase 2 R/R FL 3L+
ORR / CR (%)	**91 / 60**	**86 / 68**	**80 / 60**
mDOR (months)	Not evaluable	Not evaluable	22.8 mo
Landmarks (if available)	76% at 12 mo 74% at 18 mo	71% at 12 mo	62% at 12 mo 57% at 18 mo

Mosunetuzumab (TCE) achieves similar outcomes to CAR-T cells in heavily pre-treated follicular lymphoma patients with off-the-shelf convenience and no need for lymphodepleting chemotherapy.

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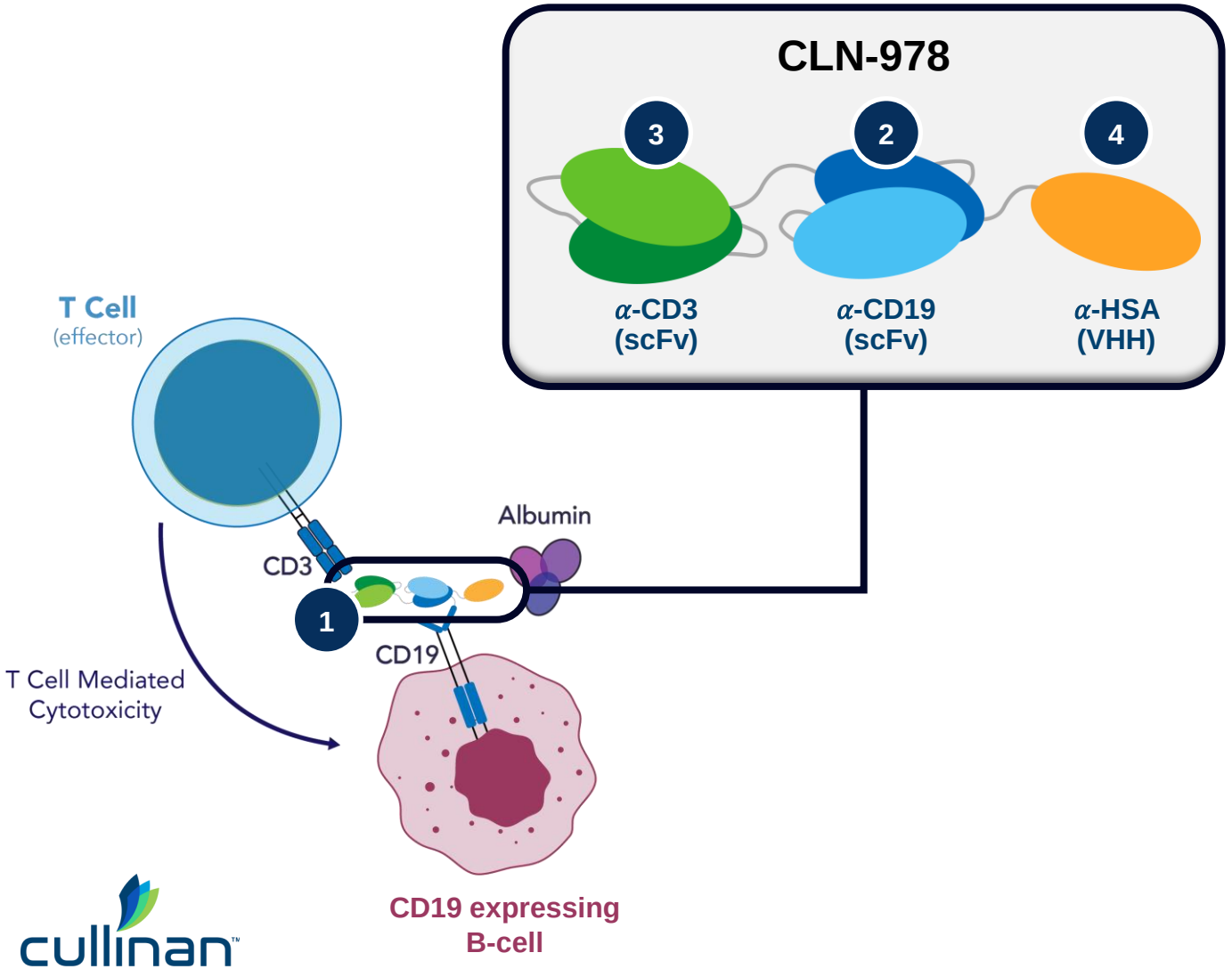
1. FDA package insert.; 2. FDA package insert; Fowler et al; 3. FDA package insert. * head-to –head studies have not been conducted

CLN-978: A novel CD19 T cell engager designed to deliver T cell directed potency with off-the-shelf convenience



1. CLN-978 potently triggers redirected lysis of CD19-expressing target cells *in vitro* and *in vivo*
2. Engineered to achieve very high affinity binding to CD19 to efficiently target B cells expressing very low CD19 levels
3. CD3 vs CD19 relative binding affinity ratio selected for optimal therapeutic index
4. Binding to serum albumin for extension of serum half-life, enabling weekly subcutaneous dosing

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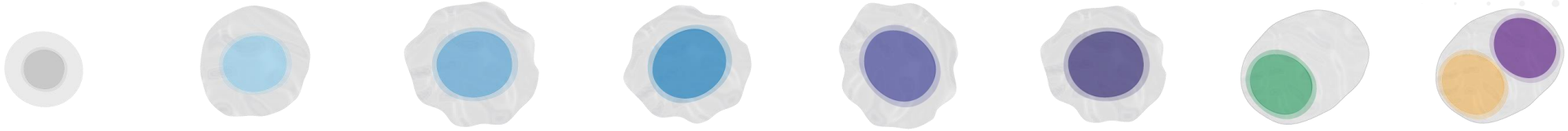
Targeting CD19 achieves broader coverage of the B cell compartment compared to CD20 or BCMA



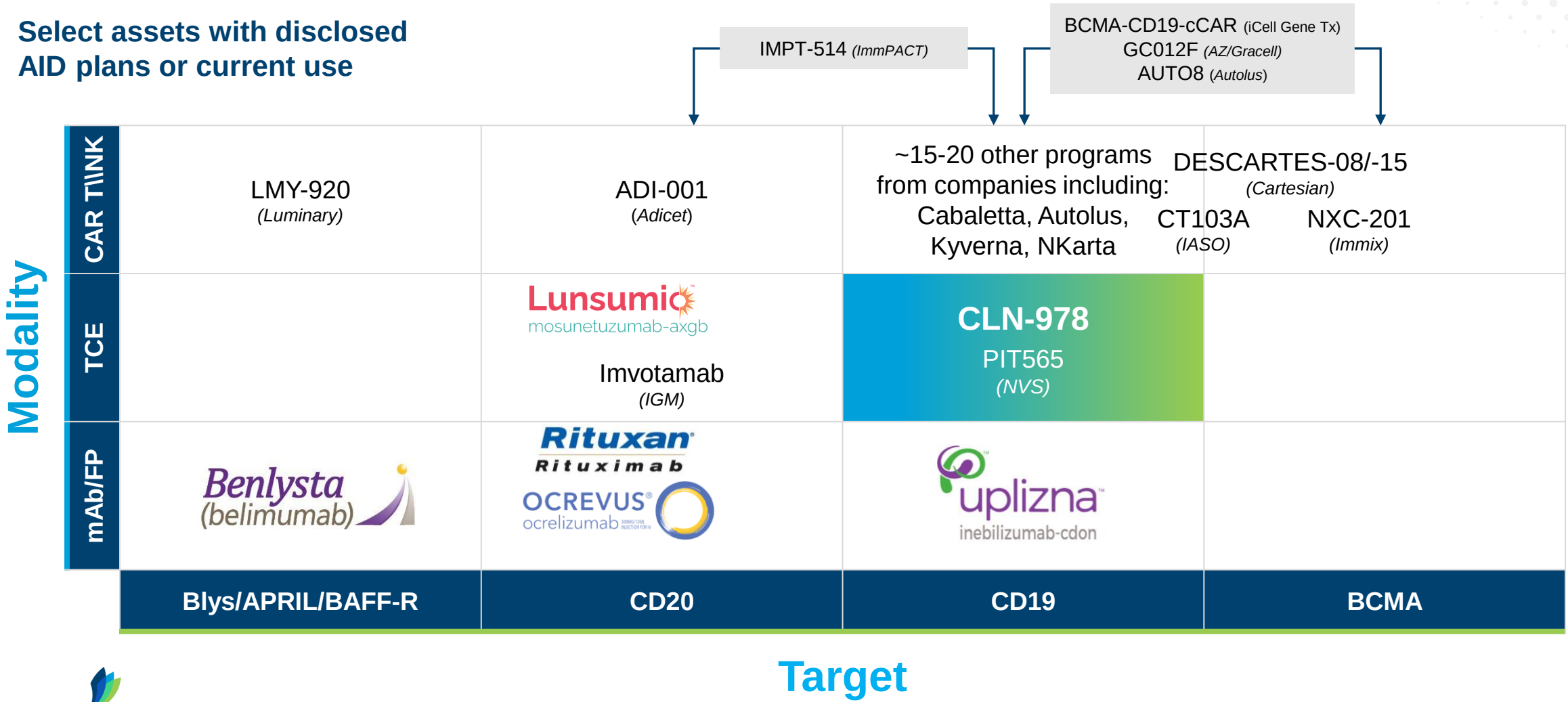
	Stem cell	Pro-B cell	Pre-B cell	Immature B cell	Mature B cell	Memory B Cell	Plasmablast	Plasma cell
CD19								
CD20								
BCMA								

- CD19 directed therapies afford significant potential for deep and broad B cell depletion as is necessary for an immune system reset
- CD20 is not expressed on plasma cells, the cells primarily responsible for autoantibody production[1]
- BCMA-directed therapies deplete long-lived plasma cells and have been associated with significant rates of severe infection[2]

[1] Tedder, T. F. & Engel, P. CD20: a regulator of cell-cycle progression of B lymphocytes. Immunol. Today 15, 450–454 (1994).
[2] Reynolds et al. Blood Advances 2023

Attractive Opportunity for CLN-978 Amongst Autoimmune Therapies Targeting B Cells

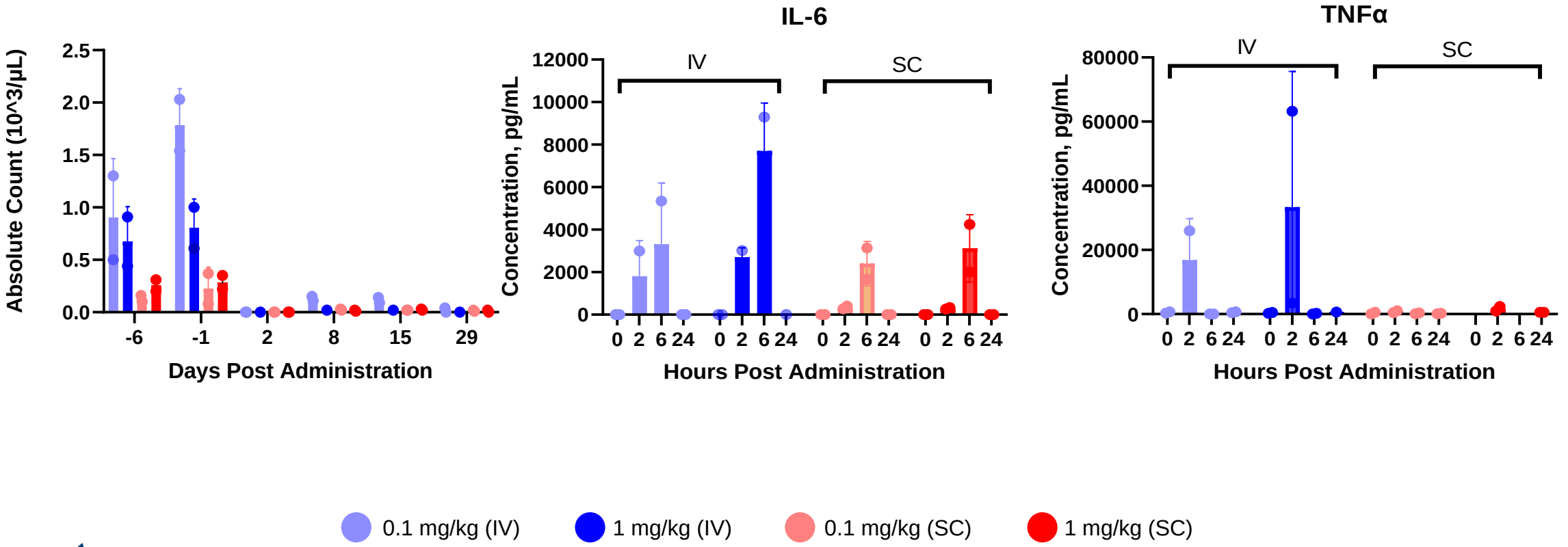
Select assets with disclosed AID plans or current use



CLN-978 pre-clinical highlights: Subcutaneous dosing achieved rapid, deep and sustained B cell depletion with attenuated cytokine release in NHPs

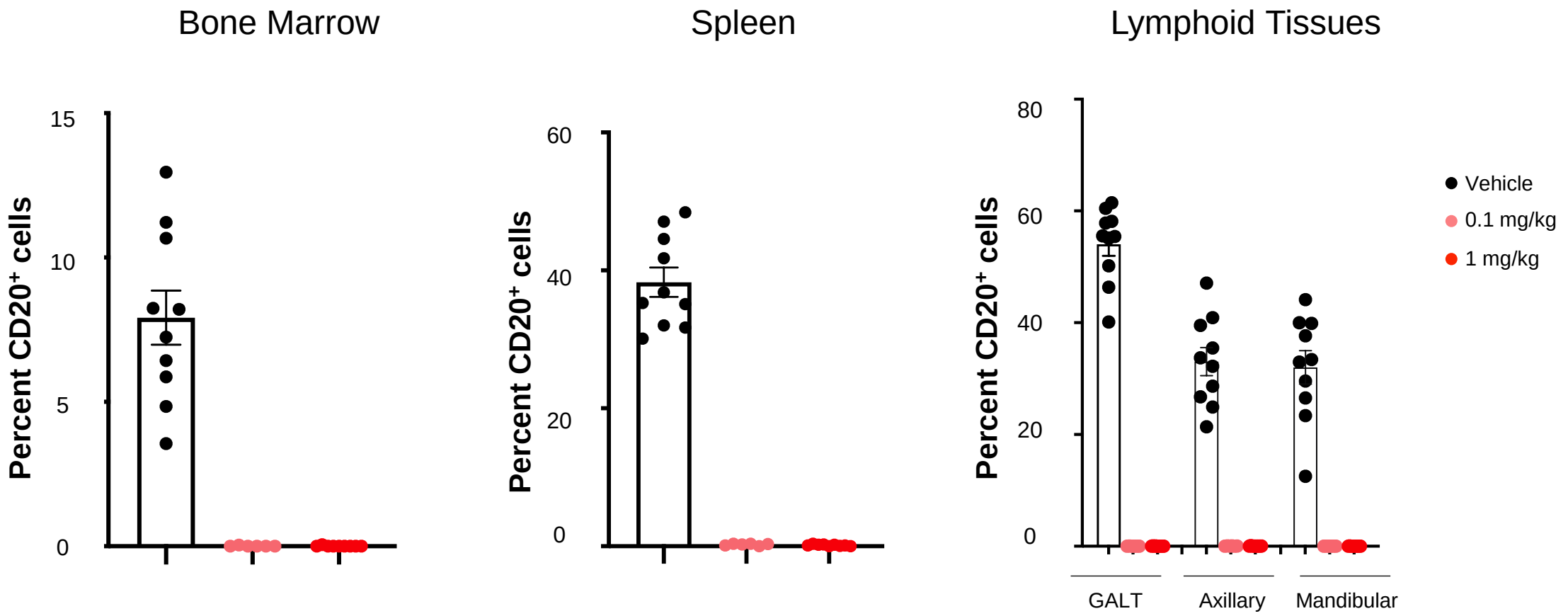
Deep, sustained peripheral B cell depletion after a single dose in NHPs

Subcutaneous dosing attenuated cytokine release in NHPs



NHPs = nonhuman primates

Deep B cell depletion in bone marrow, spleen and lymphoid tissues following SC administration of CLN-978 in cynomolgus monkeys



SC = subcutaneous administration, GALT = gut associated lymphoid tissue

CLN-978 in B-NHL: Clinically active at starting dose with favorable safety

Clinical observations from B-NHL study (as of 5 April 2024)

Disease Characteristics and Efficacy Observations					Treatment Emergent Adverse Events[1]			
ID	**Diagnosis**	**Prior Lines**	**Duration of CLN-978 Treatment**	**Best Response** (Cheson 2014)	Non-Hematological	Hematological	CRS	ICANS
1	DLBCL	3	9 doses	PD	Gr 1 fatigue, injection site reaction, intermittent headaches	Gr 4 lymphopenia[2]	Gr 1 (fever)	None
2	Follicular	3	24 doses (ongoing)	SD	Gr 1 pruritus	Gr 4 lymphopenia	Gr 1 (fever)	None
3	Mantle	3	7 doses	CR	Gr 3 vascular access complication (DVT)[3,4] Gr 2 intermittent restlessness[4]	Gr 3 lymphopenia	None	None

- **All patients treated at starting dose: 30 µg SC weekly**
- **2 of 3 patients demonstrated objective clinical benefit, including a complete response**

- **Class toxicity: max Gr 1 CRS, no ICANS**

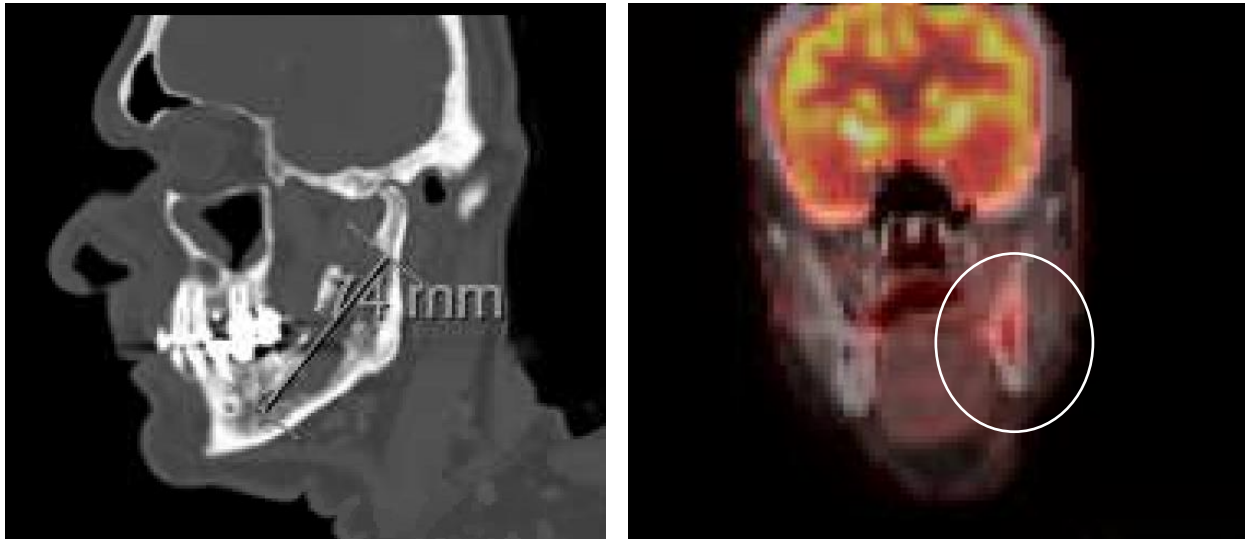
  Subject #3: transient Gr 1 tremor in the context of acute influenza infection during cycle 1; transient (~24h) Gr 2 confusion during cycle 2; neither event associated with CRS/ICANS
- **Other adverse events were low-grade and/or mechanistically based (e.g., lymphopenia)**

Further enrollment discontinued given reprioritization for development in autoimmune diseases

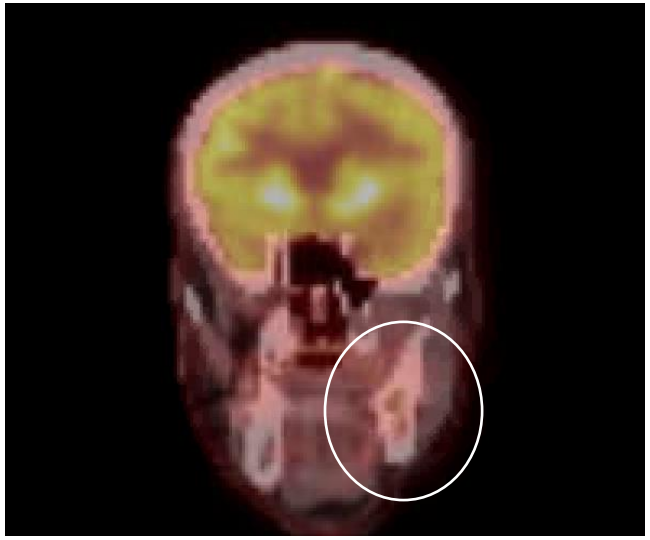
[1]Highest grade events reported per category; [2]Transient (<96h) lymphopenia following the first dose only based on mechanism of action (B cell depletion + transient T cell margination); [3]DVT = deep venous thrombosis, patient with prior history venous thromboembolic disease; [4]Investigator assessed unrelated to CLN-978; DLBCL = Diffuse Large B Cell Lymphoma; Gr = grade; CRS = Cytokine Release Syndrome; ICANS = Immune Effector Cell Associated Neurotoxicity Syndrome; PD = progressive disease; SD = stable disease, CR = complete response

Subject #3 case study: 68-year-old patient with mantle cell lymphoma

Baseline PET-CT



After 7 doses of CLN-978



- 7.4 x 1.7 cm mass in left mandible and adjacent musculature was palpable on physical exam at baseline
- Investigator reported mass was no longer appreciated on exam 96 hours following the first dose of CLN-978
- Repeat PET-CT after 7 doses of CLN-978 consistent with complete metabolic response

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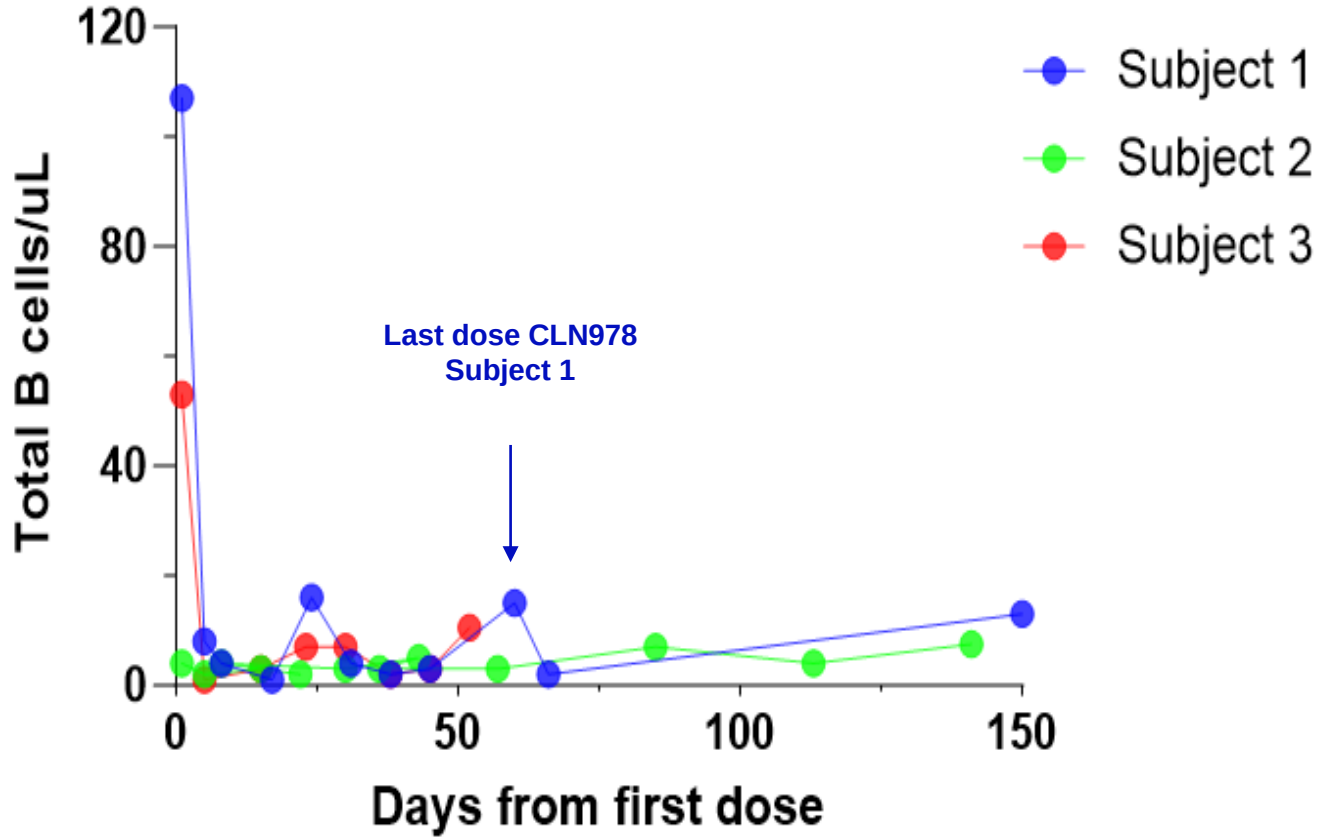
Rapid, deep and sustained B cell depletion was demonstrated in B-NHL patients following CLN-978 dosing

Peripheral blood TBNK flow assay

Data cut-off 20 March 2024



- **Rapid, deep and sustained B cell depletion was demonstrated in 2 of 2 subjects with measurable B cells at baseline**
- **All patients treated at the starting dose level of 30 ug SC weekly**

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CLN-978-SLE-001 planned study design

Study Population

1. SLE patients
2. One or more of the following SLE autoantibodies:
   - anti-nucleosome
   - anti-dsDNA
   - anti-Smith
3. SLEDAI-2K ≥ 8
4. No CNS disease

PART A: DOSE ESCALATION

PLANNED DESIGN FEATURES

- Step-wise escalation to determine target dose for further development
- Incorporation of step-up dosing to minimize risk for cytokine release syndrome and neurotoxicity
- Standard pre-medication including corticosteroids
- In-patient monitoring for 48 hours

PART B: DOSE EXPANSION

PLANNED DESIGN FEATURES

- Exploration of 2 or more dosing schedules in a larger number of SLE patients

Objectives

Primary Objective:

Safety of CLN-978 for treatment of active SLE

Secondary Objectives:

- PK
- B cell kinetics
- Immunogenicity
- Preliminary efficacy

IND submission planned 3Q:24

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Systemic Lupus Erythematosus (SLE)[1]: High Unmet Need Creates a Compelling Market Opportunity

High Unmet Need

- Systemic disease characterized by autoantibodies produced by B cells, leading to multiple affected organ systems (renal, CNS, cardiovascular, respiratory, skin)
  - Largely impacts young, women of color
- ~40% of SLE patients develop Lupus Nephritis[6], which has a 10-year 30% mortality rate
- Current standards of care do not routinely induce treatment-free remission
  - Most patients require lifelong immune suppression, treating symptoms without modifying course of disease

U.S. SLE Market Opportunity – 2024 Estimate

163,000 — **Diagnosed patients** (18-70 y/o)

122,000 — **Addressable patients[2]**

~85,000 — **Estimated moderate/severe patients on available therapies[3]**

$11b+ — **Estimated moderate/severe opportunity with transformative therapies[4]**

~$1.5b — **Estimated U.S. 2023 revenue from currently available therapies[5]**

1. Includes Lupus Nephritis (LN)
2. Antinuclear Antibody (ANA) positive without Central Nervous System (CNS) treated patients
3. Global Data 2023 Estimate – includes moderate or severe patients treated with immunosuppressive agents and or biologic agents, such as Benlysta (belimumab), Saphnelo (anifrolumab) and Rituxan (rituximab)
4. Internal estimates based on total diagnosed moderate/severe patient population (18-70 y/o).
5. Company filings – includes revenue for Benlysta (belimumab) and Saphnelo (anifrolumab).
6. Mahajan, A. et al. Lupus. 2020 Aug; 29(9): 1011–1020.

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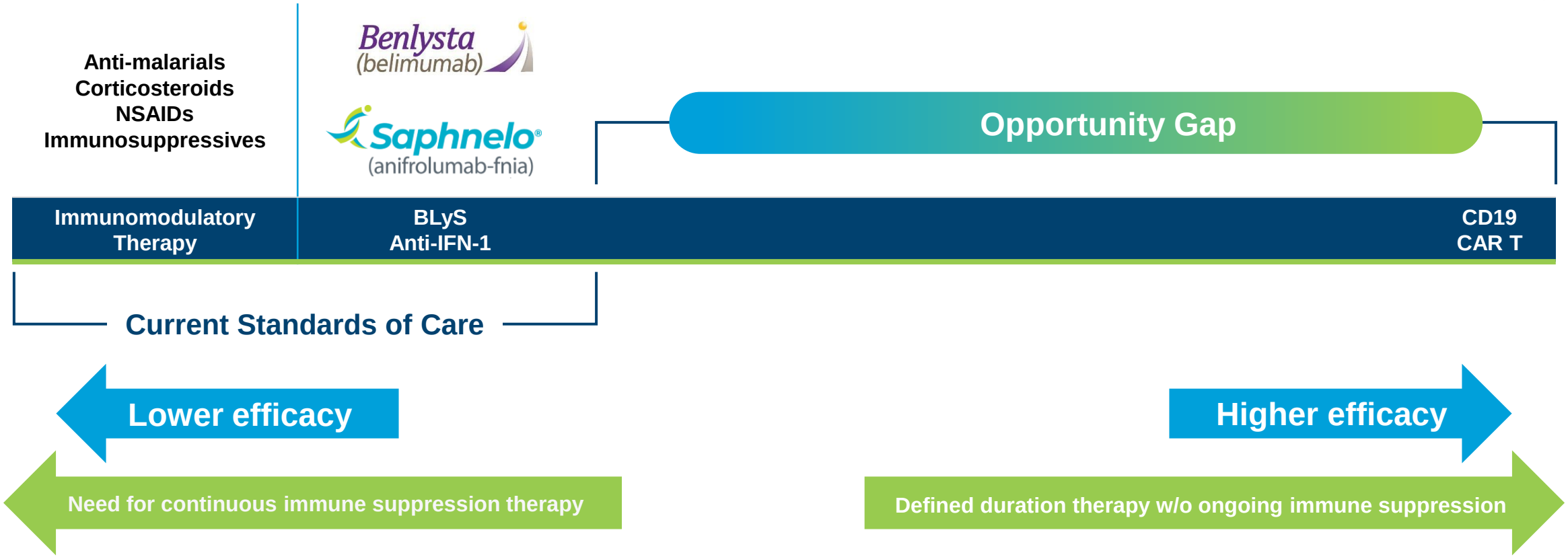
Due to CAR T cell therapy limitations, opportunity exists for CLN-978 across a range of outcomes in SLE



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Substantial number of addressable autoimmune diseases

Opportunity to Address Multiple B Cell Mediated Autoimmune Diseases

<50,000+ U.S. Patients	50,000 - 500,000 U.S. Patients	500,000+ U.S. Patients
NMOSD	SLE	Multiple Sclerosis
Pemphigus vulgaris	Myasthenia gravis	Rheumatoid arthritis
Autoimmune hemolytic anemia	Systemic sclerosis	
Idiopathic thrombocytopenia purpura	Idiopathic inflammatory myopathies	
	Sjogren's	
	ANCA+ vasculitis	
	Membranous nephropathy	

Source: Patient prevalence data from GlobalData, NIH.

NMOSD: Wright, S.K., Wassmer, E., Vincent, A. BBA- Biomem. 2021; PV: Kasperkiewicz, M. et al. Nat Rev Disease Primers 2017., Razzaque Ahmed, A. et al. Experimental Dermatology 2016; wAIHA: Berentsen & Barcellini, N Egl J Med. 2021; ITP: Zufferey, A., Kapur, R. & Semple, J. W.J. Clin. Med. 2017, Al-Samkari,H. et aal. Blood Adv. 2020; SLE: Muller, F. et al. NEJM 2024; MN: Dantas, M. et al. Brazilian J. Nephrol. 2023, Rojas-Rivera, Je.E., et al. Kidney Int. Reports 2023; MG: Gilhus, N.E. et al. Nat. Rev. Disease Primers. 2019; Sjogren's: Bayetto, K. & Logan, R.M. Aust. Dent. Jour. 2010; ANCA vasculitis: Nakazawa, D. et al., Nature Rev. Rheumatol. 2018; SSC: Hoppner, J. et al. Front. Immunol. 2023., Ebata, S. et al. Rheumatol. (United Kingdom) 2022; IIM: Khoo,T. et al. Nat. Rev. Rheumatol. 2023; RA: Volkov, M., van Schie, K.A. & van der Woude, D. Immunol. Rev. 2020.

CLN-978 program: Summary and next steps

- CLN-978 development refocused exclusively on autoimmune diseases
- CD19 is the optimal target for autoimmune diseases
- CLN-978 represents a potential first in class opportunity with the following differentiated benefits:
  - Off-the-shelf convenience and subcutaneous delivery
  - Potential disease-modifying treatment with a differentiated safety profile
  - Flexible modality allowing for repeat dosing as needed
- In B-NHL patients, CLN-978 at the starting dose, has demonstrated:
  - Rapid, deep and sustained B cell depletion
  - Clinical activity including a complete response
  - Favorable safety profile at a clinically active dose level
- SLE is the first indication with an IND submission planned for 3Q 2024
- CLN-978 has the potential to address high unmet need and drive significant value as a disease-modifying treatment across a broad range of autoimmune diseases

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Q&A

Nadim Ahmed
Chief Executive Officer

Jeff Jones, MD, MPH, MBA
Chief Medical Officer

Patrick Baeuerle, PhD
Chief Scientific Advisor

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