UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, D.C. 20549

FORM 8-K

CURRENT REPORT

Pursuant to Section 13 or 15(d) of the Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): September 04, 2025

CULLINAN THERAPEUTICS, INC.

(Exact name of Registrant as Specified in Its Charter)

Delaware	**001-39856**	**81-3879991**
(State or Other Jurisdiction of Incorporation)	**(Commission File Number)**	**(IRS Employer Identification No.)**
One Main Street Suite 1350 Cambridge, Massachusetts		**02142**
(Address of Principal Executive Offices)		**(Zip Code)**

Registrant's Telephone Number, Including Area Code: 617 410-4650

(Former Name or Former Address, if Changed Since Last Report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	**Trading Symbol(s)**	**Name of each exchange on which registered**
Common Stock, $0.0001 par value per share	CGEM	The Nasdaq Global Select Market

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§ 230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§ 240.12b-2 of this chapter).

Emerging growth company ☐

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 7.01 Regulation FD Disclosure.

Cullinan Therapeutics, Inc. (the "Company") updated its corporate presentation, used from time to time in meetings with third parties and posted to its website. A copy of the current presentation is attached as Exhibit 99.1 to this Current Report on Form 8-K.

The information in this Item 7.01, including Exhibit 99.1 attached hereto, shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended, or otherwise subject to the liabilities of that section, nor shall it be deemed incorporated by reference in any filing under the Securities Act of 1933, as amended, except as expressly set forth by specific reference in such filing.

Item 8.01 Other Events.

The Company updated its anticipated timeline of announcing initial clinical data for its CLN-978 program in systemic lupus erythematosus to be in the first half of 2026. The anticipated timeline for the announcement of clinical data for all of the Company's other programs remains unchanged.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

Exhibit No.	Description
99.1	Corporate Presentation
104	Cover page from this Current Report on Form 8-K, formatted in Inline XBRL

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

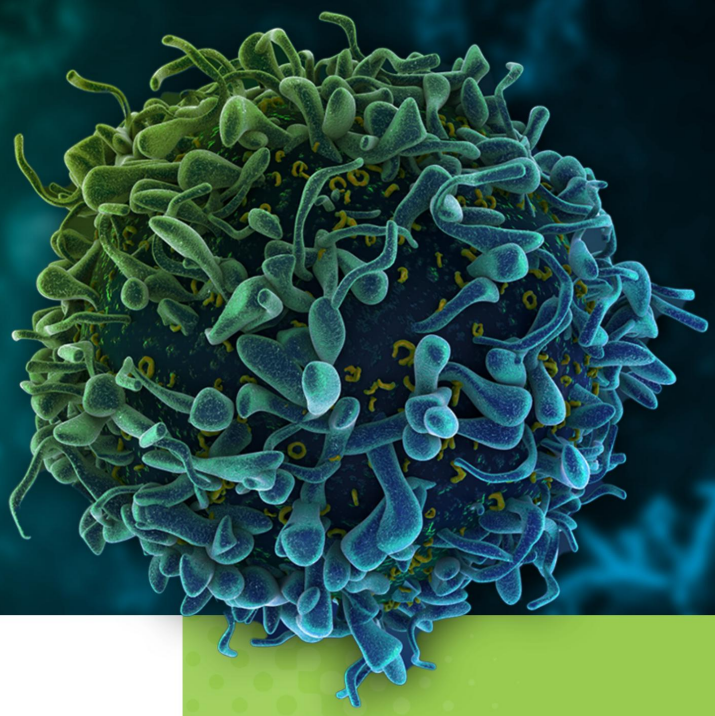
CULLINAN THERAPEUTICS, INC.

Date: September 4, 2025

By: /s/ Mary Kay Fenton
Mary Kay Fenton
Chief Financial Officer

CORPORATE OVERVIEW

September 2025



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Important Notice and Disclaimers

This presentation contains forward-looking statements within the meaning of The Private Securities Litigation Reform Act of 1995. All statements other than statements of historical facts contained in this presentation, including express or implied statements regarding the Company's beliefs and expectations related to: our preclinical and clinical development plans and timelines, the clinical and therapeutic potential of our product candidates, the strategy of our product candidates, our research and development activities, our future financial condition, our future operations and projected costs, prospects and plans of management are forward-looking statements. In some cases, you can identify forward-looking statements by terminology such as "anticipate," "believe," "continue," "estimate," "expect," "intend," "may," "plan," "potential," "predict," "seek," "should," "target," "will" or the negative of these terms or other comparable terminology.

Any forward-looking statements in this presentation are based on management's current expectations and beliefs of future events and are subject to known and unknown risks and uncertainties that may cause our actual results, performance or achievements to be materially different from any future results, performance or achievements expressed or implied by the forward-looking statements. These risks include, but are not limited to, the following: uncertainty regarding the timing and results of regulatory submissions; the risk that any INDs, NDAs or other global regulatory submissions we may file with the United States Food and Drug Administration or other global regulatory agencies are not cleared on our expected timelines, or at all; the success of our clinical trials and preclinical studies; the risks related to our ability to protect and maintain our intellectual property position; the risks related to manufacturing, supply, and distribution of our product candidates; the risk that any one or more of our product candidates, including those that are co-developed, will not be successfully developed and commercialized; the risk that the results of preclinical studies or clinical studies will not be predictive of future results in connection with future studies; the success of any collaboration, partnership, license or similar agreements; and other important risks and uncertainties discussed in our filings with the Securities and Exchange Commission, including under the caption "Risk Factors" in our most recent Annual Report on Form 10-K, Quarterly Report on Form 10-Q and other filings that we make with the SEC from time to time. These risks could cause actual results to differ materially from those indicated by the forward-looking statements made in this presentation. While we may elect to update such forward-looking statements at some point in the future, we disclaim any obligation to do so, even if subsequent events cause our views to change, except to the extent required by law. These forward-looking statements should not be relied upon as representing our views as of any date subsequent to the date of this presentation. Moreover, except as required by law, neither Cullinan nor any other person assumes responsibility for the accuracy and completeness of the forward-looking statements included in this presentation. Any forward-looking statement included in this presentation speaks only as of the date on which it was made.

Certain information contained in this presentation relates to or is based on studies, publications, surveys and other data obtained from third-party sources and our own internal estimates and research. While we believe these third-party sources to be reliable as of the date of this presentation, we have not independently verified, and make no representation as to the adequacy, fairness, accuracy or completeness of, any information obtained from third-party sources. In addition, all of the market data included in this presentation involves a number of assumptions and limitations, and there can be no guarantee as to the accuracy or reliability of such assumptions. Finally, while we believe our own internal research is reliable, such research has not been verified by any independent source.

CULLINAN THERAPEUTICS

Our Mission: Create new standards of care for patients

- We use a unique R&D model of identifying high-impact targets and then applying the best modality to address each target
- We are rigorously advancing only highly-differentiated molecules, yielding a robust portfolio of clinical-stage programs, including three T cell engagers, a core area of development expertise
- Cullinan has a leadership position in the development of T cell engagers for autoimmune diseases with CLN-978 (CD19xCD3) and velinotamig (BCMAxCD3), utilizing a comprehensive potential disease modifying approach through both B cell and plasma cell depletion. CLN-978 is being investigated in systemic lupus erythematosus (SLE), rheumatoid arthritis (RA) and Sjögren's disease (SjD). Genrix Bio is planning a Phase 1 study of velinotamig in China in patients with autoimmune diseases to begin later this year.
- We are simultaneously advancing a diversified pipeline of clinical-stage oncology programs, with multiple catalysts in 2025
- We are well-positioned to execute on strategic goals and advance to a commercial-stage organization, with cash runway into 2028*

As of June 30, 2025

Poised for multiple value-creation opportunities in the near-term

	CLN-978 CD19xCD3 bispecific TCE for SLE, RA, and SjD	Zipalertinib EGFR inhibitor for EGFR ex20ins NSCLC	CLN-049 FTL3xCD3 bispecific T cell engager for AML & MDS	Additional clinical programs
Program highlights	• *First-in-class* potential in autoimmune diseases • Proven modality (TCE) & differentiated profile	• *Best-in-class* potential • Attractive economics incl. $130m milestones + 50/50 U.S. profit share	• *First-in-class* potential • FLT3 is expressed in approximately 80% of AML patients	• **CLN-619:** anti-MICA/B mAb enrolling in Phase 1 disease specific expansion cohorts in NSCLC and Phase 1 study in r/r multiple myeloma • **CLN-617:** IL2/IL12 fusion protein for solid tumors • **Velinotamig:** BCMA x CD3 bispecific T cell engager for autoimmune diseases
Next milestone/ status	• Initial data in SLE in **H1 2026** • Initial data in RA in **H1 2026** • Phase 1 study ongoing in SjD • Reviewing development in additional autoimmune diseases	• Pivotal REZILIENT1 Phase 2b results in 2L+ shared at **ASCO 2025**; updated data at **IASLC 2025 WCLC** • Pending discussions with the U.S. FDA, potential NDA filling by **YE 2025** by Taiho in relapsed EGFR ex20ins NSCLC.	• Phase 1 study ongoing in patients with relapsed/refractory AML or MDS; clinical data in **Q4 2025** • Phase 1 study ongoing in patients with measurable minimal residual disease in AML.	

Cash of $511M* supports operations into 2028

*As of June 30, 2025 (unaudited), includes cash equivalents, investments, and interest receivable


T Cell Engagers in
Autoimmune Diseases
cullinan
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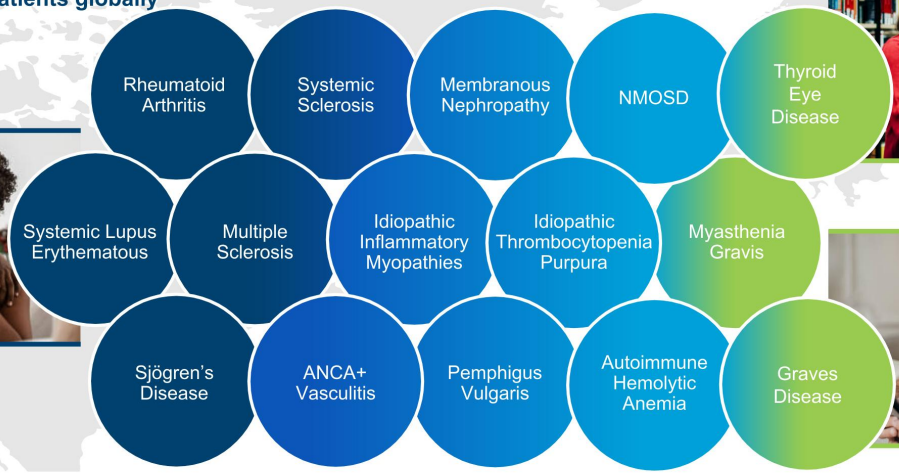
Growing global prevalence of autoimmune diseases underscores need for treatments that deliver durable remissions

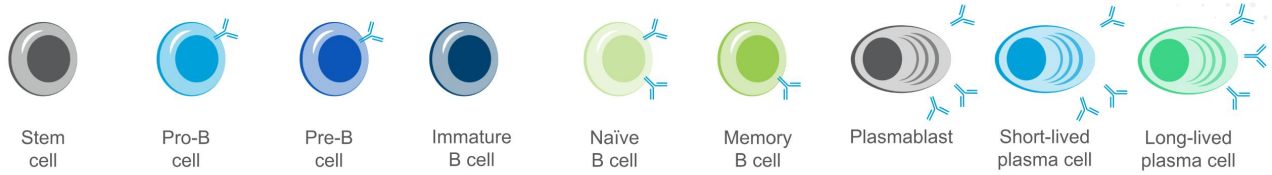
OPPORTUNITY TO ADDRESS AUTOIMMUNE DISEASES THROUGH B CELL AND LONG-LIVED PLASMA CELL DEPLETION

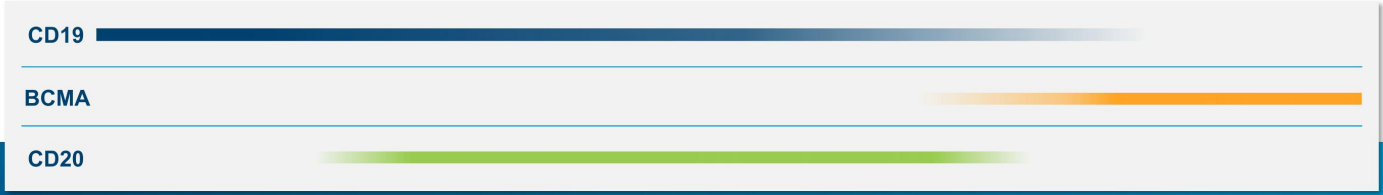
Combined ~41.6M patients globally



Source: SLE: Tian J, Zhang D, Yao X, Huang Y, Lu Q. Global epidemiology of systemic lupus erythematosus: a comprehensive systematic analysis and modelling study. Ann Rheum Dis. Published online October 14, 2022. RA: Rheumatoid arthritis: Key Facts. WHO. Published online June 28, 2023. MG: Dresser L, Wlodarski R, Rezania K, Soliven B. Myasthenia Gravis: Epidemiology, Pathophysiology and Clinical Manifestations. J Clin Med. 2021 May 21;10(11):2235. SS: Tian J, Kang S, Zhang D, Huang Y, Zhao M, Gui X, Yao X, Lu Q. Global, regional, and national incidence and prevalence of systemic sclerosis. Clin Immunol. 2023 Mar;248:109267. MN: Ronco, P., Beck, L., Debiec, H. et al. Membranous nephropathy. Nat Rev Dis Primers 7, 69 (2021). ITP: Affected Populations. Immune Thrombocytopenia National Organization for Rare Disorders. Last updated 2022. MS: MS Prevalence. National MS Society. IIM: Khoo, T., Lilleker, J.B., Thong, B.YH. et al. Epidemiology of the idiopathic inflammatory myopathies. Nat Rev Rheumatol 19, 695–712 (2023). NMOSD: Baghereh S, Afshari-Safavi A, Vaheb S, Kiani M, Ghaffary EM, Barzegar M, Shaygannejad V, Zabeti A, Mirmosayyeb O. Worldwide prevalence of neuromyelitis optica spectrum disorder (NMOSD) and neuromyelitis optica (NMO): a systematic review and meta-analysis. Neurol Sci. 2023 Jun;44(6):1905-1915. AHA: Hansen D.L., Möller S., Andersen K., Gaist D., Frederiksen H. Increasing Incidence and Prevalence of Acquired Hemolytic Anemias in Denmark, 1980–2016. Clin. Epidemiol. 2020;12:497–508. ANCAV: Redondo-Rodriguez R, Mena-Vázquez N, Cabezas-Lucena AM, Manrique-Arija S, Mucientes A, Fernández-Nebro A. Systematic Review and Metaanalysis of Worldwide Incidence and Prevalence of Antineutrophil Cytoplasmic Antibody (ANCA) Associated Vasculitis. J Clin Med. 2022 May 4;11(9):2573. Sjogren's: Epidemiology. Sjogren Syndrome. Medscape. Last updated: March 24, 2023. PV: Rosi-Schumacher M, Baker J, Waris J, Seiffert-Sinha K, Sinha AA. Worldwide epidemiologic factors in pemphigus vulgaris and bullous pemphigoid. Front Immunol. 2023 Apr 25;14:1159351. Graves'/TED: Girgis CM, Champion BL, Wall JR. Current concepts in graves' disease. Ther Adv Endocrinol Metab. 2011;2(3):135-144. doi:10.1177/2042018811408488

Targeting CD19 or BCMA may be central to the disease modification of certain autoimmune diseases





- B cell dysfunction is central to the pathogenesis of many autoimmune diseases, and broadly and deeply depleting these cells by targeting **CD19** appears necessary to affect an immune system reset
- Autoantibodies central to the pathogenesis of other autoimmune diseases are predominantly produced by plasma cells, particularly long-lived plasma cells, so depleting these cells by targeting **BCMA** could potentially improve outcomes[1,2,3]
- **CD20** expression on B cells may not be sufficiently broad enough to induce an immune reset nor does it directly deplete plasma cells

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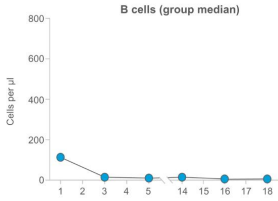
[1] Dong N., Zhang H., Song J., et al. B-cell maturation antigen expression and clinical features of plasmablastic lymphoma. EJHaem. 2024 Jan 18;5(1):285-289. doi: 10.1002/jha2.807. PMID: 38406544; PMCID: PMC10887266.
[2] Pillarisetti K., Powers G., Luistro L., et al. Teclistamab is an active T cell-redirecting bispecific antibody against B-cell maturation antigen for multiple myeloma. Blood Adv. 2020 Sep 22;4(18):4538-4549. doi: 10.1182/bloodadvances.2020002393. PMID: 32956453; PMCID: PMC7509877.
[3] Tian D.S., Qin C., Dong M.H., et al. cell lineage reconstitution underlies CAR-T cell therapeutic efficacy in patients with refractory myasthenia gravis. EMBO Mol Med. 2024 Apr;16(4):966-987. doi: 10.1038/s44321-024-00043-z. Epub 2024 Feb 26. PMID: 38409527; PMCID: PMC11018773

Data show proof of concept for TCEs to meaningfully improve upon standard of care across certain autoimmune diseases

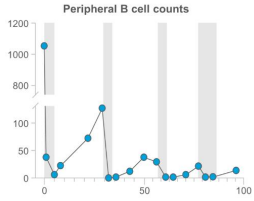
BLINATUMOMAB: CD19-DIRECTED TCE IN RA AND SSc

B cell depletion in RA (n=6)



Bucci L et al. *Nat Med*. 2024.

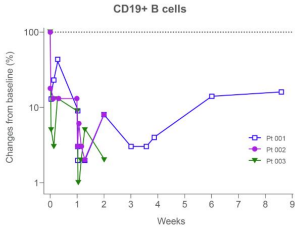
B cell depletion in SSc (n=1)



Subklewe et al. *Eur J Cancer*. 2024.

CD19-DIRECTED TCE IN SLE (N=6)

SLE peripheral blood CD19+ B cell counts (n=3)



- Studied in six severe/refractory patients with SLE
- No CRS/ICANS/infections
- Improved SLEDAI-2K scores within two to eight weeks in three evaluable patients, including one patient with B cell repopulation and no relapse at eight weeks

Mei C et al. Presented at ACR Convergence 2024. Abstract L12.

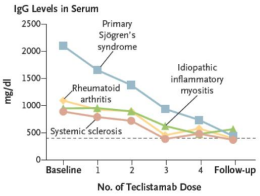
BCMAXCD3 TCE IN RA, SjD, IIM, SSc

Clinical effect, profound decrease in autoantibodies and IgG levels in four patients with autoimmune diseases that were multi-drug resistant (n=4)

Changes in Serum Autoantibodies at Latest Follow-up

	Autoantibody	Baseline	Follow-up
Patient 1 (systemic sclerosis)	ANA — titer	1:3200	1:100
	PM-Scl-75 — RU (normal <15)	30	1
	PM-Scl-100 — RU (normal <15)	35	3
Patient 2 (primary Sjögren's syndrome)	ANA — titer	1:10,000	1:3200
	SS-A/Ro-52 — U/ml (normal <25)	129.7	55.3
	SS-A/Ro-60 — U/ml (normal <25)	189.5	131.3
	SS-B/La — U/ml (normal <25)	222.5	165.2
	PL-7 — RU (normal <15)	122	91
Patient 3 (idiopathic inflammatory myositis)	ANA — U/ml (normal <25)	1:100	1:100
	MDA5 — RU (normal <15)	38	18
Patient 4 (rheumatoid arthritis)	RF — U/ml (normal <25)	507.2	18.4
	CCP2 — U/ml (normal <25)	2.7	0.9
	MCV — U/ml (normal <25)	82.9	3.0

IgG Levels in Serum



Clinical Efficacy Analysis

	Patient 1 (systemic sclerosis)		Patient 2 (primary Sjögren's syndrome)		Patient 3 (idiopathic inflammatory myositis)		Patient 4 (rheumatoid arthritis)	
	Before treatment	After treatment	Before treatment	After treatment	Before treatment	After treatment	Before treatment	After treatment
Visual analogue scale (patient global) — mm	90	50	50	10	50	20	60	40
Modified Rodnan skin score — units	39	24	N/A	N/A	N/A	N/A	N/A	N/A
ESSDAI (Sjögren's syndrome activity) — units	N/A	N/A	34	15	N/A	N/A	N/A	N/A
CDASI (skin inflammation) — units	N/A	N/A	N/A	N/A	22	6	N/A	N/A
MMT-8 (diffusion capacity of lung) — units	N/A	N/A	N/A	N/A	138	150	N/A	N/A
DAS28–CRP (arthritis) — units	N/A	N/A	N/A	N/A	3.6	1.6	5.6	1.9
Health assessment questionnaire — units	2.5	2.1	1.3	1.1	1.8	1.0	3.0	1.6
DLCOSB (single-breath diffusion capacity of lung for carbon monoxide) — percent of predicted	34	33	23	27	44	57	76	72

Hagen M et al. *NEJM*. 2024.

SLE = Systemic Lupus Erythematosus; IIM = Idiopathic Inflammatory Myositis; SSc = Systemic Sclerosis; RA = Rheumatoid Arthritis.

Optimal modality: TCEs provide high therapeutic potential for deep and durable B cell and plasma cell depletion in a convenient, off-the-shelf treatment option

mAbs	TCEs	CAR T
✗ Lower efficacy; limited tissue penetration	✓ High efficacy; B cell depletion in tissue	✓ High efficacy; B cell depletion in tissue
✗ Symptomatic relief	✓ Potentially disease modifying	✓ Potentially disease modifying
✗ Immune modulating	✓ Potential for immune reset	✓ Potential for immune reset
✓ Off-the-shelf and ease of administration	✓ Off-the-shelf and ease of administration	✗ Lengthy manufacturing process tailored to each patient; complex administration
✓ Dosing flexibility	✓ Dosing flexibility to extend remissions	✗ Single dose without opportunity to re-dose
✓ Free from access issues and complex interdisciplinary coordination	✓ Free from access issues and complex interdisciplinary coordination	✗ Can have access issues and involves complex interdisciplinary coordination
✓ Free from lymphodepleting preconditioning regimen	✓ Free from lymphodepleting preconditioning regimen	✗ Requires lymphodepleting preconditioning regimen

T CELL REDIRECTING THERAPIES

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CLN-978
CD19xCD3 bispecific T cell engager
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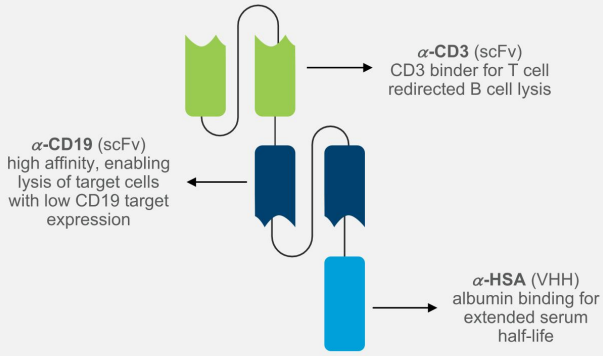
CLN-978: a highly differentiated and potentially best-in-class CD19xCD3 bispecific T cell engager

CLN-978 BISPECIFIC CD19XCD3 T CELL ENGAGER



CLN-978 POTENTIAL ADVANTAGES IN AUTOIMMUNE DISEASES

Therapeutic modality

Predictable PK/PD properties, engaging all subsets of T cells to lyse target cells with variable CD19 expression levels. Dosing flexibility and ability to redose to extend remissions

Optimal target

CD19 best balances the potential for deep and broad B cell depletion (vs CD20) necessary to affect an immune reset while limiting risk for infection (vs BCMA)

Convenience

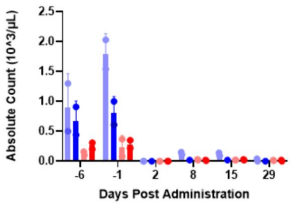
Off-the-shelf availability, subQ administration, lower manufacturing burden, dosing flexibility, potential for outpatient administration

HSA = human serum albumin binding domain

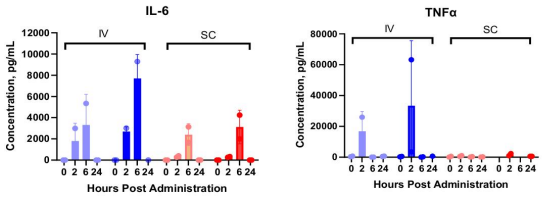
In preclinical studies, subcutaneous dosing of CLN-978 achieved rapid, deep and sustained B cell depletion in blood and tissue with attenuated cytokine release

PRECLINICAL HIGHLIGHTS

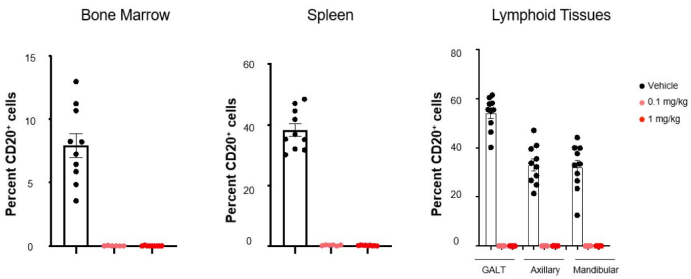
Deep, sustained peripheral B cell depletion after single dose in NHPs



SubQ dosing attenuated cytokine release in NHPs



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

0.1 mg/kg (IV) 1 mg/kg (IV) 0.1 mg/kg (SC) 1 mg/kg (SC)

In a study of B-NHL patients, CLN-978 achieved sustained B cell depletion with promising clinical results

CLINICALLY ACTIVE AT 30µg SC WEEKLY STARTING DOSE FOR NHL PATIENTS

2 of 3 patients demonstrated objective clinical benefit, including a complete response

Class toxicity: max Gr 1 CRS, no ICANS

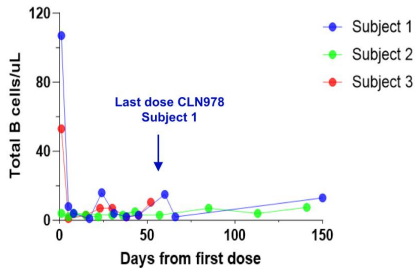
- Subject #3: transient Gr 1 tremor in the context of acute influenza infection during cycle 1; transient (~24h) Gr 2 confusion during cycle 2; neither event associated with CRS/ICANS

Other adverse events were low-grade and/or mechanistically based (e.g., lymphopenia)

Further enrollment discontinued given reprioritization for development in autoimmune diseases

RAPID, DEEP AND SUSTAINED B CELL DEPLETION DEMONSTRATED IN B-NHL PATIENTS

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



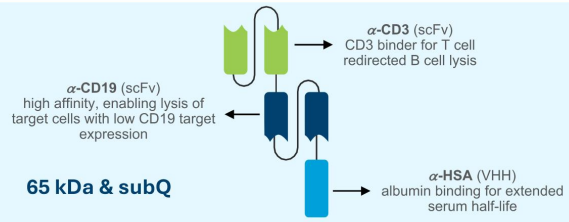
Peripheral blood TBNK flow assay
Data cut-off 20 March 2024

B-NHL = B cell Non-Hodgkin Lymphoma; Gr = grade; CRS = Cytokine Release Syndrome; ICANS = Immune Effector Cell Associated Neurotoxicity Syndrome

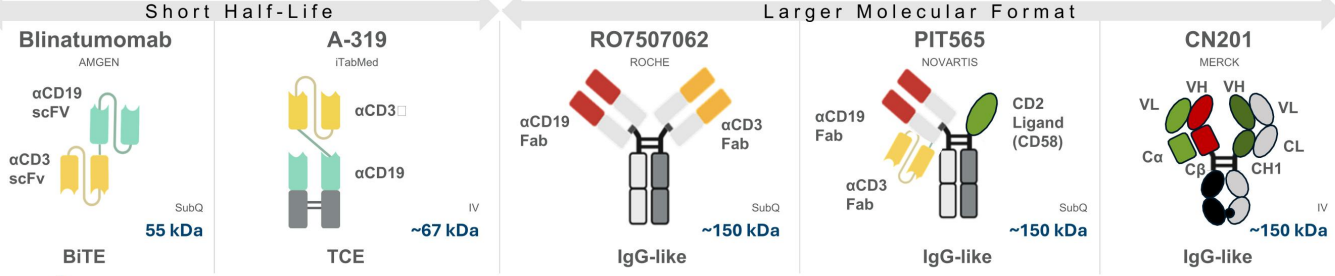
CLN-978 is well differentiated relative to other CD19 TCEs in development*

POTENTIAL ADVANTAGES OF CLN-978

1. Broader B cell lineage depletion due to very high affinity binding to CD19
2. Wider therapeutic index due to “cytokine window” (10X higher potency for B cell depletion relative to cytokine induction)
3. More efficient deep tissue penetration due to smaller size
4. Published clinical data for blinatumomab supports promising potential for a half-life extended bispecific TCE in autoimmune diseases



Short Half-Life | Larger Molecular Format



*The above list of CD19 TCEs in development is indicative only and not exhaustive; molecule size assumptions based on publicly available data
Source: Janeway CA Jr, Travers P, Walport M, et al. Immunobiology: The Immune System in Health and Disease. 5th edition. New York: Garland Science; 2001.

CLN-978-SLE-101 global study design

Study Population

1. SLE meeting 2019 EULAR/ACR criteria
2. One or more of the following SLE autoantibodies:
   - anti-nuclear antibody
   - anti-dsDNA
   - anti-Smith
3. SLEDAI ≥6 at screening
4. Inadequate response to at least one standard immunosuppressant or biologic used for the treatment of SLE

PART A: DOSE ESCALATION

Cohort 4 (*optional; n=3*)
- Day 1: 10 micrograms
- Day 8: 45 micrograms

Cohort 3 *(n=3)*
- Day 1: 10 micrograms
- Day 8: 30 micrograms

Cohort 2 *(n=3)*
- Day 1: 10 micrograms
- Day 8: 20 micrograms

Cohort 1 *(n=3)*
- Day 1: 10 micrograms

PART B: DOSE EXPANSION

Further exploration of 2 or more dosing schedules

Schedule 1 *(n= 3 to 6)*
- Dose level and schedule to be determined by PK/PD findings observed in Part A

Schedule 2 *(n=3 to 6)*
- Dose level and schedule to be determined by PK/PD findings observed in Part A

Objectives

Primary Objective:

Safety and tolerability of CLN-978 for treatment of active SLE

Secondary Objectives:
- PK
- B cell kinetics
- Immunogenicity
- Clinical activity

Global Phase 1 study ongoing in United States, Europe, and Australia

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Clinicaltrials.gov identifier: NCT06613360

Systemic Lupus Erythematosus (SLE): High unmet need in complex disease with few approved therapies, limited efficacy and chronic immunosuppression

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[1], which has a 10-year 30% mortality rate

SLE - SELECT MARKET OPPORTUNITY 2025 ESTIMATE (US, EU5, JP, AU)

430,000 **Diagnosed patients** (18-70 y/o)[2-9]

285,000 **Estimated addressable patients**[10]

193,000 **Estimated moderate/severe patients**[11]

OPPORTUNITY: CURRENT STANDARDS OF CARE DO NOT ROUTINELY INDUCE TREATMENT-FREE REMISSION, MOST PATIENTS REQUIRE LIFELONG IMMUNE SUPPRESSION, TREATING SYMPTOMS WITHOUT MODIFYING COURSE OF DISEASE

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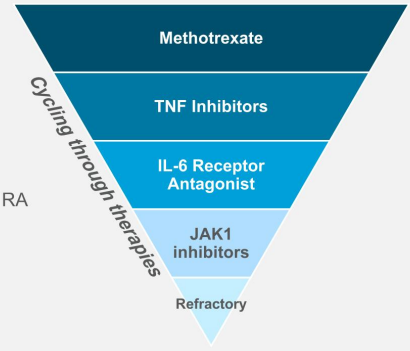
1. Mahajan, A. et al. Lupus. 2020 Aug; 29(9): 1011–1020.
2. US: Izmirly, P. M. et al. (2021b) 'Prevalence of systemic lupus erythematosus in the United States: estimates from a meta-analysis of the centers for disease control and prevention national lupus registries', Arthritis and Rheumatology, 73(6), pp. 991–996. doi: 10.1002/art.41632
3. UK: Rees, F. et al. (2016) 'The incidence and prevalence of systemic lupus erythematosus in the UK, 1999-2012', Annals of the Rheumatic Diseases, 75(1), pp. 136–141. doi: 10.1136/annrheumdis-2014-206334.
4. FR: Arnaud, L. et al. (2014) 'Prevalence and incidence of systemic lupus erythematosus in France: a 2010 nation-wide population-based study', Autoimmunity Reviews, 13(11), pp. 1082–1089. doi: 10.1016/j.autrev.2014.08.034.
5. IT: Tsioni, V. et al. (2015) 'The prevalence and incidence of systemic lupus erythematosus in children and adults: a population-based study in a mountain community in northern Italy', Clinical and Experimental Rheumatology, 33(5), pp. 681–687.
6. DE: Brinks, R. et al. (2014) 'Age-specific prevalence of diagnosed systemic lupus erythematosus in Germany 2002 and projection to 2030', Lupus, 23(13), pp. 1407–1411. doi: 10.1177/0961203314540352.
7. ES: Alonso, M. D. et al. (2011) 'Systemic lupus erythematosus in northwestern Spain: a 20-year epidemiologic study', Medicine, 90(5), pp. 350–358. doi: 10.1097/MD.0b013e31822ed7f7.
8. JP: Bae, E. H. et al. (2020) 'Trend of prevalence and incidence of systemic lupus erythematosus in South Korea, 2005 to 2015: a nationwide population-based study', Korean Journal of Internal Medicine, 35(3), pp. 652–661. doi: 10.3904/kjim.2018.303.
9. AU: Nikpour M, Bridge JA, Richter S. A systematic review of prevalence, disease characteristics and management of systemic lupus erythematosus in Australia: identifying areas of unmet need. Intern Med J. 2014 Dec;44(12a):1170-9. doi: 10.1111/imj.12568. PMID: 25169712.
10. Internal company estimate – Antinuclear Antibody (ANA) positive without Central Nervous System (CNS) patients based on historical systemic therapy treatment rates
11. Internal company estimate - portion of addressable patients who present with moderate or severe SLE

Rheumatoid Arthritis (RA) second indication for CLN-978

No transformational therapies expected from industry RA development pipeline

- Current therapies result in chronic immune suppression, increasing infection risk, especially in elderly patients
- First CD19xCD3 TCE in clinical development for RA



CLN-978 company-sponsored clinical trial to be initiated at institutions that pioneered initial POC study of anti-CD19 TCE in RA

FAU Friedrich-Alexander-Universität Erlangen-Nürnberg

Gemelli Fondazione Policlinico Universitario A. Gemelli Università Cattolica del Sacro Cuore


nature medicine

Article https://doi.org/10.1038/s41591-024-02964-1

Bispecific T cell engager therapy for refractory rheumatoid arthritis

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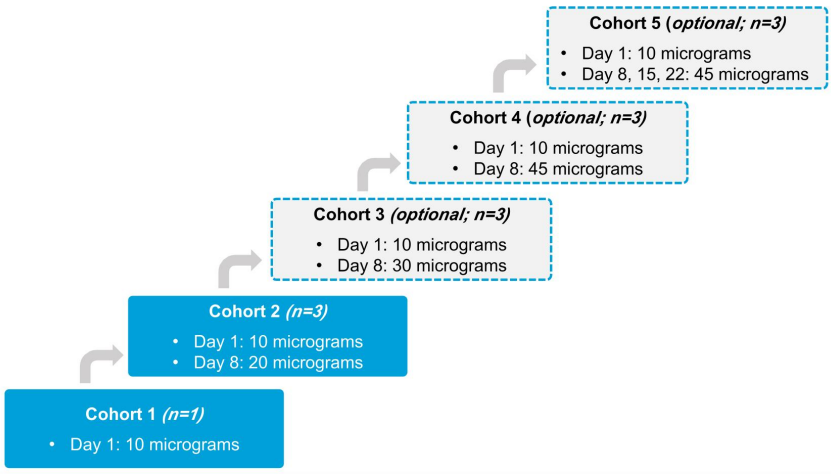
CLN-978-RA-101 initial Phase 1 design

Study Population

1. RA meeting 2010 EULAR/ACR criteria
2. Inadequate response to at least two DMARDS (tsDMARD and/or biologic) after csDMARD treatment
3. DAS28-ESR ≥ 3.2, at least one swollen joint

PHASE 1 DOSE ESCALATION



Objectives

Primary Objective:

Safety and tolerability of CLN-978 for treatment of active RA

Secondary Objectives:

- PK
- B cell kinetics in peripheral blood and tissue biopsies
- Immunogenicity
- Clinical activity

Phase 1 study ongoing in Europe

EUCT number: 2024-519114-31-00

Rheumatoid Arthritis (RA): Large pool of highly refractory patients

HIGH UNMET NEED

- Rheumatoid arthritis (RA) is a chronic inflammatory disease that mainly affects the joints with pathogenesis associated with autoantibodies
- ~77% of RA patients present with moderate or severe disease activity
- Large poly-refractory population with no available treatments
- Current therapies result in chronic immune suppression, increasing infection risk, especially in elderly patients

RA - SELECT MARKET OPPORTUNITY 2025 ESTIMATE (US, EU5, JP, AU)

5,300,000 — **Diagnosed patients** (>=18 years of age)[1-8]

3,386,000 — **Estimated addressable patients**[9]

2,315,000 — **Estimated moderate/severe RA patients**[10]

163,000 — **Estimated number of patients who are multi-drug resistant or poly-refractory**[11]

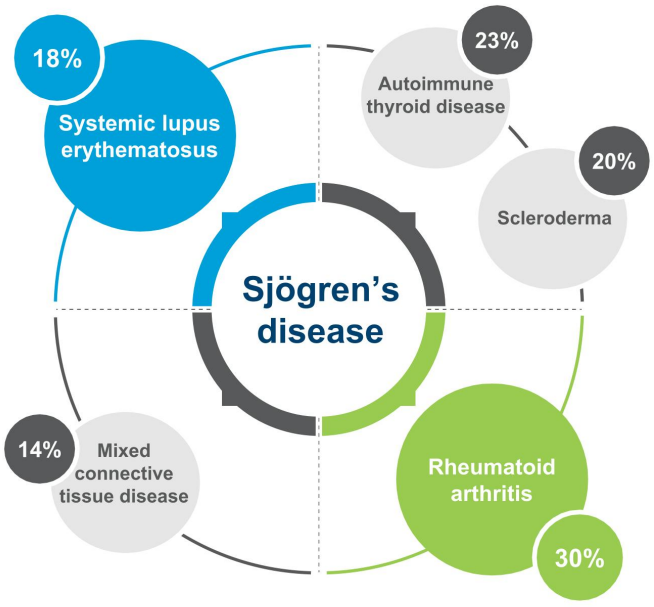
OPPORTUNITY: LARGE GROUP OF RA PATIENTS WHO FAIL AVAILABLE THERAPIES

1. US: Hunter, T. M. et al. (2017) 'Prevalence of rheumatoid arthritis in the United States adult population in healthcare claims databases, 2004–2014', Rheumatology International. Springer Berlin Heidelberg, 37(9), pp. 1551–1557. doi: 10.1007/s00296-017-3726-1.
2. UK: Abhishek, A. et al. (2017) 'Rheumatoid arthritis is getting less frequent-Results of a nationwide population-based cohort study', Rheumatology (United Kingdom), 56(5), pp. 736–744. doi: 10.1093/rheumatology/kew468.
3. FR: Guillemin, F. et al. (2005) 'Prevalence of rheumatoid arthritis in France: 2001', Annals of the Rheumatic Diseases, 64(10), pp. 1427–1430. doi: 10.1136/ard.2004.029199.
4. IT: Rossini, M. et al. (2014) 'Prevalence and incidence of rheumatoid arthritis in Italy', Rheumatology International, 34(5), pp. 659–664. doi: 10.1007/s00296-014-2974-6.
5. DE: Steffen, A. et al. (2017) 'Epidemiologie der rheumatoiden Arthritis in Deutschland – eine Analyse anhand bundes- weiter vertragsärztlicher Abrechnungsdaten', Zentralinstitut für kassenärztliche Versorgung in Deutschland, (17), pp. 1–20. doi: 10.20364/VA-17.08.
6. ES: Fina-Aviles, F. et al. (2016) 'The descriptive epidemiology of rheumatoid arthritis in Catalonia: a retrospective study using routinely collected data', Clinical Rheumatology, 35(3), pp. 751–757. doi: 10.1007/s10067-014-2801-1.
7. JP: Kojima, M. et al. (2019) 'Epidemiological characteristics of rheumatoid arthritis in Japan: Prevalence estimates using a nationwide population-based questionnaire survey', Modern Rheumatology, 7595. doi: 10.1080/14397595.2019.1682776.
8. AU: Ackerman IN, Pratt C, Gorelik A, Liew D. Projected Burden of Osteoarthritis and Rheumatoid Arthritis in Australia: A Population-Level Analysis. Arthritis Care Res (Hoboken). 2018 Jun;70(6):877-883. doi: 10.1002/acr.23414. Epub 2018 Apr 12. PMID: 28898565.
9. Internal company estimate - addressable patients is estimated according to observed rheumatoid factor (RF) or anti-citrullinated protein autoantibodies (ACPA) seropositivity rates and historical systemic therapy treatment rates
10. Internal company estimate – portion of addressable patients who present with moderate to severe rheumatoid arthritis
11. Internal company estimate – portion of moderate to severe patients who are multi-drug resistant or poly-refractory

Third indication for CLN-978: Sjögren's disease (SjD) has significant overlap with SLE, RA and other connective tissue diseases

About half of the time SjD occurs alone, and the other half it occurs in the presence of **another connective tissue disease**.

Signs and symptoms of **SjD frequently overlap with SLE and RA**, and many rheumatologists treat all three diseases.



1. Sjögren's Foundation
2. Clin Rheumatol. 2020 Mar 4;39(6):1899–1905. doi: 10.1007/s10067-020-05004-8
3. Open Access Rheumatol. 2019 Jan 29;11:33–45. doi: 10.2147/OARRR.S167783
4. Case Rep Nephrol Dial. 2024 Mar 22;14(1):48–55. doi: 10.1159/000537873

CLN-978-SjD-101 study design

Study Population

1. SjD meeting 2016 EULAR/ACR criteria with a disease duration ≥ 24 weeks
2. Moderate to severe disease (ESSDAI ≥ 5) at screening
3. Inadequate or loss of response to at least two SOC immunosuppressive or biologic therapies

PART A: DOSE ESCALATION

Cohort 4 (*optional; n=3*)
- Day 1: 10 micrograms
- Day 8: 45 micrograms

Cohort 3 *(n=3)*
- Day 1: 10 micrograms
- Day 8: 30 micrograms

Cohort 2 *(n=3)*
- Day 1: 10 micrograms
- Day 8: 20 micrograms

Cohort 1 *(n=3)*
- Day 1: 10 micrograms

PART B: DOSE EXPANSION

Further exploration of 2 or more dosing schedules

Schedule 1 *(n= 3 to 6)*
- Dose level and schedule to be determined by PK/PD findings observed in Part A

Schedule 2 *(n=3 to 6)*
- Dose level and schedule to be determined by PK/PD findings observed in Part A

Objectives

Primary Objective:

Safety and tolerability of CLN-978 for treatment of patients with active SjD

Secondary Objectives:
- PK
- B cell kinetics in peripheral blood and salivary biopsies
- Immunogenicity
- Clinical activity

Global Phase 1 study ongoing in the U.S. and Europe

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Sjögren's Disease (SjD): Complex and debilitating disease occurring in isolation or in association with other systemic conditions

HIGH UNMET NEED

- SjD is a chronic autoimmune disease characterized by lymphocytic infiltration of the salivary and lacrimal glands
- Autoantibodies play crucial roles in both the diagnosis and prognosis of SjD
- Patients with SjD have an average of five other health conditions, including but not limited to conditions such as GERD, Raynaud's, neuropathy, sinusitis, hypertension, anemia, fibromyalgia, and irritable bowel syndrome[1]

PRIMARY SjD - SELECT MARKET OPPORTUNITY 2025 ESTIMATE (US, EU5, JP, AU)

836,000 **Diagnosed patients** (>=18 years of age)[2-9]

482,000 **Estimated Addressable patients**[10]

148,000 **Estimated moderate/severe patients**[11]

OPPORTUNITY:A PREVALENT B CELL DRIVEN DISEASE WITH NO APPROVED ADVANCED SYSTEMIC THERAPIES

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1. Sjogren's Foundation.
2. US: Maciel, G. et al. (2017) 'FRI0278 Prevalence of primary Sjögren's syndrome in a population-based cohort in the United States', Arthritis Care and Research, 69(10) p. 591.2. doi: 10.1136/annrheumdis-2017-eular.1179.
3. UK: United Kingdom Primary Sjögren's Syndrome Registry (UKPSSR) (2011). National Research Biobank. Available from: https://www.sjogrensregistry.org/for-patients (Accessed: 17 July 2020)
4. FR: Maldini, C. et al. (2014) 'Epidemiology of primary Sjögren's syndrome in a French multiracial/multiethnic area', Arthritis Care and Research, 66(3), pp. 454–463. doi: 10.1002/acr.22115
5. IT: Sardu, C. et al. (2012) 'Population based study of 12 autoimmune diseases in Sardinia, Italy: prevalence and comorbidity', PLoS ONE, 7(3). doi: 10.1371/journal.pone.0032487.
6. DE: Westhoff, G. and Zink, A. (2010) 'Epidemiology of primary Sjögren's syndrome', Journal of Rheumatology, 69(1), pp. 1–9. doi: 10.1007/s00393-009-0518-3
7. ES: Cortes, J. B. et al. (2019) 'Prevalence of Sjögren's syndrome in the community of Madrid', Annals of the Rheumatic Diseases, p. 791.2–792. doi: 10.1136/annrheumdis-2019-eular.3949.
8. JP: Tsuboi, H. et al. (2014) 'Primary and secondary surveys on epidemiology of Sjögren's syndrome in Japan', Modern Rheumatology, 24(3), pp. 464–470. doi: 10.3109/14397595.2013.843765.
9. AU: Lyne SA, Downie-Doyle S, Lester SE, Quinlivan A, Toby Coates P, Gordon TP, Rischmueller M. Primary Sjögren's syndrome in South Australia. Clin Exp Rheumatol. 2020 Jul-Aug;38 Suppl 126(4):57-63. Epub 2020 Sep 15. PMID: 32940213.
10. Internal company estimate – anti-SSA/RO antibodies and/or rheumatoid factor patients based on historical systemic therapy treatment rates
11. Internal company estimate - portion of addressable patients who present with moderate to severe SjD


Velinotamig
BCMAxCD3 bispecific T cell engager
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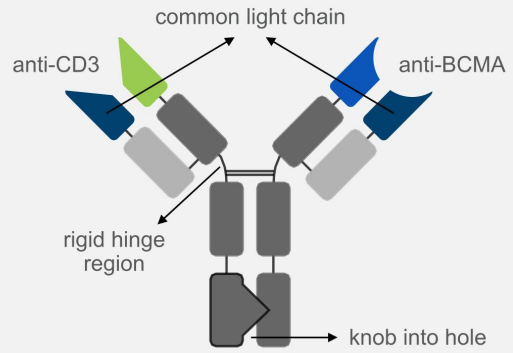
Velinotamig: a BCMAxCD3 bispecific T cell engager with clinical evidence of robust plasma cell depletion

VELINOTAMIG BISPECIFIC BCMAXCD3 T CELL ENGAGER



VELINOTAMIG A HIGHLY SELECTIVE, DIFFERENTIATED MOLECULE

- Binds to the BCMA and CD3 antigens, **redirecting cytotoxic T cells to target BCMA-expressing cells**
- **High affinity for BCMA and lower affinity for CD3:** affinity for BCMA is two orders of magnitude higher than for CD3, promoting recruitment and activation of T cells while minimizing non-specific T cell activation
- A rigid hinge region enhances synapse formation between T cells and target cells to **eliminate BCMA+ plasma cells**
- Similar structure as classic monoclonal antibody and possible lower immunogenicity by using **common light chain; subcutaneous administration (**in development) is feasible

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Velinotamig current clinical development status in China

PLANNED CLINICAL TRIAL IN AUTOIMMUNE DISEASES

- **Genrix Bio** plans to conduct a **Phase 1** study in China in patients with **autoimmune diseases** beginning **later this year**
- **Cullinan expects the data generated to accelerate the global clinical development of velinotamig in autoimmune diseases**
- Following the completion of the Genrix Bio Phase 1 study, **Cullinan will conduct all further development of velinotamig in autoimmune diseases**

COMPLETED/ ONGOING CLINICAL TRIALS IN MULTIPLE MYELOMA

- **GR1803-001:** A (follow-up) completed **Phase 1** study **in patients with relapsed/refractory multiple myeloma**
- **GR1803-002:** A **Phase 2** pivotal study **in patients with relapsed/refractory multiple myeloma**
- **GR1803-003:** A **Phase 2** pivotal study **in patients with relapsed/refractory multiple myeloma with extramedullary lesions**
- **Velinotamig** received **Breakthrough Therapy Designation** by the Center for Drug Evaluation (CDE) for the treatment of relapsed/refractory multiple myeloma

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Velinotamig has demonstrated meaningful efficacy in late-line patients with relapsed/refractory multiple myeloma: Phase 1 results at RP2D target dose

- **Higher ORR** rate observed relative to approved BCMA TCEs
- **Higher ORR** in patients with extramedullary disease **(EMD), a particularly poor prognosis subset of MM patients**
- Similar **CR and ≥VGPR rate** versus the majority of approved BCMA TCEs, despite a **larger proportion of EMD patients**

		Approved molecules[b] (in U.S. and or EU)		
	Velinotamig[a] (n=48)	**Elranatamab[1,2] (n=97)**	**Teclistamab[3,4,5] (n=110)**	**Linvoseltamab[6] (n=117)**
ORR (sCR + CR + VGPR + PR)	85.4%	57.7%	61.8%	70.9%
≥VGPR (sCR + CR + VGPR)	66.7%	51.5%	57.3%	63.2%
CR (sCR + CR)	33.3%	25.8%	28.2%	49.6%
MRD-neg rate (in all patients)	39.6%	*n/a*	26.7%	*n/a*
Proportion of EMD patients in study	50.0%	34.0%	25.5%	16.2%
ORR in EMD	79.2%	36.4%	35.7%	52.6%

a. Data from trial: *Phase 1 clinical study of the safety, pharmacokinetics, immunogenicity and preliminary efficacy of single and multiple administrations of GR1803 (velinotamig) in patients with relapsed/refractory multiple myeloma.* Velinotamig data as of July 31, 2024 cutoff date; includes patients who have received the target dose of 180 ug/kg, which was explored with (n=25) and without a step-up priming regimen (n=23). The recommended Phase 2 dose (RP2D) for further development includes a step-up priming regimen.
b. Data provided for context only; direct comparisons between molecules can not be made in the absence of head-to-head clinical trials.

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1. https://labeling.pfizer.com/ShowLabeling.aspx?id=19669#ID_b883b50f-6abb-40b6-ab24-68b9b6e74ac3
2. Lesokhin AM, Tomasson MH, Arnulf B, et al. Elranatamab in relapsed or refractory multiple myeloma: phase 2 MagnetisMM-3 trial results. Nat Med. 2023;29:2259-2267. doi:10.1038/s41591-023-02528-9
3. https://www.janssenlabels.com/package-insert/product-monograph/prescribing-information/TECVAYLI-pi.pdf
4. https://www.jnjmedicalconnect.com/products/tecvayli/medical-content/tecvayli-use-in-patients-with-extramedullary-disease
5. Moreau P, Garfall A, van de Donk C, et al. Teclistamab in relapsed or refractory multiple myeloma. N Engl J Med. 2022;387:495-505
6. Naresh Bumma et al. Linvoseltamab for Treatment of Relapsed/Refractory Multiple Myeloma. JCO 42, 2702-2712(2024). DOI:10.1200/JCO.24.01008

Overview of velinotamig safety data at RP2D target dose

	Any grade	≥Grade3
	Velinotamig (n=48)*	
Cytokine release syndrome	89.6%	6.3%
Infection	81.3%	50.0%
ICANS	0%	0%
Neutrophil count decreased	89.6%	64.6%
Platelet count decreased	79.2%	39.6%
White blood cell count decreased	89.6%	35.4%
Lymphocyte count decreased	62.5%	60.4%

- Opportunities to further mitigate **CRS** are being implemented:
  - Alternative **step-up dosing** regimen
  - Introduction of **subcutaneous formulation**
- **No ICANS** of any grade at the RP2D
- Augmented supportive care measures to reduce the risk of infection will be implemented in autoimmune studies

*Data from trial: *Phase 1 clinical study of the safety, pharmacokinetics, immunogenicity and preliminary efficacy of single and multiple administrations of GR1803 (velinotamig) in patients with relapsed/refractory multiple myeloma.* Velinotamig data as of July 31, 2024 cutoff date; includes patients who have received the target dose of 180 ug/kg, which was explored with (n=25) and without a step-up priming regimen (n=23). The recommended phase 2 dose (RP2D) for further development includes a step-up priming regimen.

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ZIPALERTINIB
(CLN-081/TAS6417)
EGFRex20ins inhibitor
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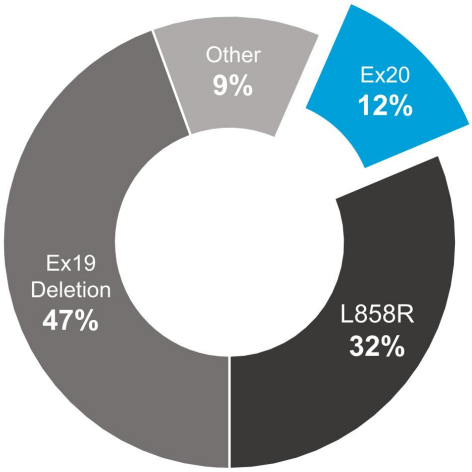
Patients with insertions at exon 20 make up the largest unmet need segment of the lung cancer population with EGFR mutations

EGFR MUTATED NSCLC[1]



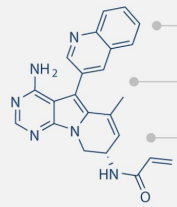
U.S. EXON 20 INCIDENCE

U.S. lung cancer incidence[2]:	~235,000
NSCLC[1]:	80%-85%
Exon 20[3-5]:	1.5%-2.5% of NSCLC ~3,000 to ~5,000 patients

References 1. Riess JW et al. J Thorac Oncol 2018. **2.** American Lung Association (2025) **3.** Riess JW et al. J Thorac Oncol 2018. **4.** Zhang YL et al. Oncotarget 2016. **5.** Burnett H et al. PLoS ONE 2021.

Zipalertinib (CLN-081/TAS6417): Selective EGFR inhibitor with best-in-class potential for NSCLC patients with exon20 mutations

ZIPALERTINIB: UNIQUE DESIGN PROPERTIES



- **Distinct chemical scaffold**
- ***HER2*-sparing**
- **High selectivity to mutant vs WT EGFR**

PARTNERSHIP WITH TAIHO ONCOLOGY

- **Zipalertinib is designed as a next generation, irreversible EGFR inhibitor for the treatment of a genetically defined subset of patients with non-small cell lung cancer – EGFR exon20ins NSCLC.**
- **The FDA granted Breakthrough Therapy designation to zipalertinib in 2022 for the treatment of patients with locally advanced or metastatic NSCLC harboring EGFRex20ins mutations who have previously received platinum-based systemic chemotherapy.**

ZIPALERTINIB HISTORICAL TIMELINE

Zipalertinib licensed from Taiho Oncology – FEB 2019	**Entered into co-development / co-commercialization with Taiho Oncology** – MAY 2022	**REZILIENT2 and REZILIENT3 trials initiated** – AUG 2023	**REZILIENT1 Ph. 2b pivotal study met primary endpoint** – JAN 2025	
REZILIENT1 Phase 1/2a initial PoC (ASCO 2021) – JUNE 2021	**REZILIENT1 Phase 1/2a full results (ASCO 2022)** – JUNE 2022	**REZILIENT1 Ph. 2b pivotal study initiated** – NOV 2022	**REZILIENT 1 initial Module C results** – JUNE & SEP 2024	**REZILIENT1 results (ASCO 2025)** – JUNE 2025

ASCO 2025 data update: **REZILIENT1** Phase 2b trial results

REZILIENT1[1]
NCT04036682

Pivotal Phase 2b
(met primary endpoint)

- Prior chemo* only
- Prior chemo* + amivantamab +/- other exon20ins directed therapy

Data presented at ASCO 2025

**platinum-based*

Primary endpoint: ORR + DOR

REZILIENT2[2]

Phase 2 Parallel Cohort Study

- Active brain mets (+/- prior treatment)
- 1st Line ex20 (zipalertinib monotherapy)
- Non-exon20ins uncommon (PACC+) EGFRm (prior systemic therapy)

Primary endpoint: ORR

REZILIENT3[3]

1L Randomized Phase 3
(initiated Q3 2023)

R 1:1
N=~300**

- Zipalertinib + pemetrexed + carboplatin or cisplatin
- Placebo + pemetrexed + carboplatin or cisplatin

Primary endpoint: PFS

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Clinicaltrials.gov identifiers: [1]NCT04036682, [2]NCT05967689 and 3NCT05973773; * includes both approved and investigational exon20 therapies ** following 6-12 patient safety lead in. PACC = P-loop and αC-helix

REZILIENT1: heavily pre-treated patient population, many with brain metastases, relapsed after chemotherapy +/- amivantamab

Characteristic	Primary efficacy population (N=176)	Platinum-based chemotherapy without ex20ins-targeted therapy (n=125)	Prior amivantamab ± other ex20ins-target therapy (n=51)[a]
Median number of prior systemic regimens, No. (range)	**2 (1, 7)**	**1 (1, 6)**	**3 (1, 7)**
Prior chemotherapy, No. (%)	173 (98)	125 (100)	48 (94)
Prior anti–PD-(L)1, No. (%)	84 (48)	62 (50)	22 (43)
Prior targeted therapy, No. (%)	87 (49)	36 (29)	51 (100)
Amivantamab	52 (30)	0	51 (100)
Mobocertinib	17 (10)	0	17 (33)
Bevacizumab	21 (12)	14 (11)	7 (14)
Osimertinib	16 (9)	12 (10)	4 (8)
BLU-451	3 (2)	0	3 (6)
Cetuximab	4 (2)	0	4 (3)
Poziotinib	2 (1)	0	2 (4)
Sunvozertinib	1 (1)	0	1 (2)
Other[a]	21 (12)	17 (14)	4 (8)
Prior brain radiation, No. (%)	**23 (13)**	**16 (13)**	**7 (14)**
Brain metastasis untreated, No. (%)	**45 (26)**	**28 (22)**	**17 (33)**

[a]Includes first/second generation EGFR tyrosine kinase inhibitors, ALK inhibitors, CDK4/6 inhibitors, NTRK/ROS1 inhibitors, angiokinase inhibitors. ALK, anaplastic lymphoma kinase; EGFR, epidermal growth factor receptor; ex20ins, exon 20 insertions; PD-(L)1, programmed death-(ligand) 1.

REZILIENT1: Zipalertinib demonstrated meaningful clinical efficacy, including in patients previously treated with amivantamab

Outcome	Primary efficacy population (N=176)	Platinum-based chemotherapy without ex20ins-targeted therapy (n=125)	Prior amivantamab ± other ex20ins-target therapy (n=51)[a]
BOR, No. (%)[b]			
CR	1 (1)	0	1 (2)
PR	61 (35)	50 (40)	11 (22)
Unconfirmed PR[c]	7 (4)	6 (5)	1 (2)
SD	88 (50)	55 (44)	33 (65)
PD	11 (6)	8 (6)	3 (6)
Not evaluable[d]	8 (5)	6 (5)	0
Confirmed ORR, No. (%) [95% CI][e]	**62 (35) [28–43]**	**50 (40) [31–49]**	**12 (24) [13–38]**
DCR, No. (%) [95% CI][f]	157 (89) [84–93]	111 (89) [82–94]	46 (90) [79–97]
CBR, No. (%) [95% CI][g]	113 (64) [57–71]	85 (68) [59–76]	28 (55) [40–69]
Median time to response, days (range)	44 (31–295)	44 (39–232)	44 (39–232)
Median DOR, months (95% CI)	**8.8 (8.3–12.7)**	**8.8 (8.3–12.7)**	**8.5 (4.2–14.8)**

Patients were evaluable for response if they had received at least one dose of zipalertinib and had at least one post-dose tumor assessment or had discontinued prior to the first efficacy assessment due to clinical disease progression or toxicity. [a]Including 30 patients who received prior amivantamab without and 21 patients with other ex20ins-targeted therapy. [b]Response confirmed ≥4 weeks after response first noted. [c]Patients had PR but confirmatory scan had not yet been performed. [d]No post-baseline imaging. [e]Proportion of patients with confirmed CR or PR. [f]Proportion of patients with CR, PR, or SD. [g]Proportion of patients with CR, PR, or with SD lasting ≥24 weeks.

BOR, best overall response; CBR, clinical benefit rate; CI, confidence interval; CR, complete response; DCR, disease control rate; DOR, duration of response; ex20ins, exon 20 insertions; ICR, independent central review; ORR, objective response rate; PD, progressive disease; PR, partial response; SD, stable disease.

REZILIENT1: Durable clinical benefit observed in patients relapsing after prior treatment with amivantamab

Despite the approval of amivantamab for EGFR ex20ins NSCLC, patients can still face poor outcomes after exhausting a range of prior therapies

Zipalertinib demonstrated clinically meaningful efficacy in the post-amivantamab setting, a significant and growing unmet need

Outcome	Prior amivantamab without other ex20ins-targeted therapy (n=30)	Prior amivantamab and other ex20ins-targeted therapy (n=21)	Total (n=51)
BOR, No. (%)[a]			
CR	1 (3)	0	1 (2)
PR	8 (27)	3 (14)	11 (22)
Unconfirmed PR[b]	1 (3)	0	1 (2)
SD	19 (63)	14 (67)	33 (65)
PD	0	3 (14)	3 (6)
Confirmed ORR, No. (%) [95% CI][c]	**9 (30) [15–49]**	**3 (14) [3–36]**	**12 (24) [13–38]**
DCR, No. (%) [95% CI][d]	29 (97) [83–100]	17 (81) [58–95]	46 (90) [79–97]
CBR, No. (%) [95% CI][e]	18 (60) [41–77]	10 (48) [26–70]	28 (55) [40–69]
Median time to response, days (range)	43 (39–232)	98 (40–103)	44 (39–232)
Median DOR, months (95% CI)	**14.7 (4.2–NE)**	**4.2 (3.9–NE)**	**8.5 (4.2–14.8)**

Patients were evaluable for response if they had received at least one dose of zipalertinib and had at least one post-dose tumor assessment or had discontinued prior to the first efficacy assessment due to clinical disease progression or toxicity. [a]Response confirmed ≥4 weeks after response first noted. [b]Patients had PR but confirmatory scan had not yet been performed. [c]Proportion of patients with confirmed CR or PR. [d]Proportion of patients with CR, PR, or SD. [e]Proportion of patients with CR, PR, or with SD lasting ≥24 weeks. BOR, best overall response; CBR, clinical benefit rate; CI, confidence interval; CR, complete response; DCR, disease control rate; DOR, duration of response; ex20ins, exon 20 insertions; ICR, independent central review; NE, not evaluable; ORR, objective response rate; PD, progressive disease; PR, partial response; SD, stable disease.

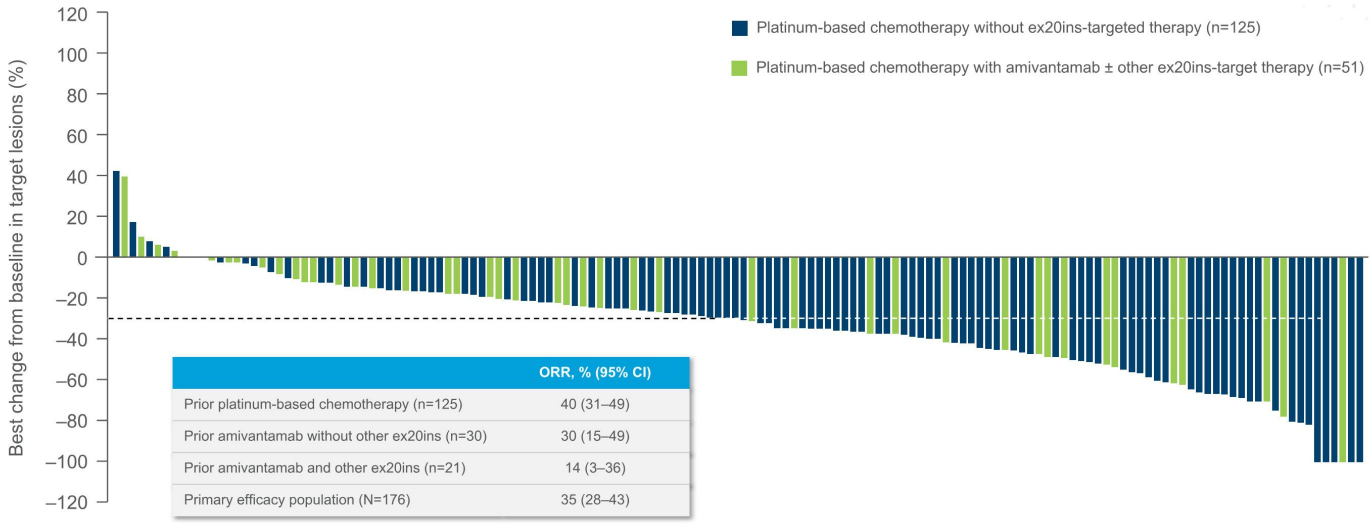
REZILIENT1: Efficacy per ICR in patients with brain metastases

Results provide preliminary evidence supporting the activity of zipalertinib in the high-risk patient population with brain metastases

Outcome	Primary efficacy population (N=176)	Patients with brain metastases[a] (n=68)
BOR, No. (%)[b]		
CR	1 (1)	1 (2)
PR	61 (35)	20 (29)
Unconfirmed PR[c]	7 (4)	2 (3)
SD	88 (50)	37 (54)
PD	11 (6)	5 (7)
Not evaluable[d]	8 (5)	3 (4)
Confirmed ORR, No. (%) [95% CI][e]	62 (35) [28–43]	21 (31) [20–43]
DCR, No. (%) [95% CI][f]	157 (89) [84–93]	60 (88) [78–95]
CBR, No. (%) [95% CI][g]	113 (64) [57–71]	38 (56) [43–68]
Median time to response, days (range)	**44 (31–295)**	**98 (35–232)**

Patients were evaluable for response if they had received at least one dose of zipalertinib and had at least one post-dose tumor assessment or had discontinued prior to the first efficacy assessment due to clinical disease progression or toxicity. [a]Patients with brain metastases included 44 patients with prior platinum-based chemotherapy without ex20ins-targeted therapy and 24 who received prior amivantamab with (9 patients) or without (15 patients) other ex20ins-targeted therapy. [b]Response confirmed ≥4 weeks after response first noted. [c]Patients had PR but confirmatory scan had not yet been performed. [d]No post-baseline imaging. [e]Proportion of patients with confirmed CR or PR. [f]Proportion of patients with CR, PR, or SD. [g]Proportion of patients with CR, PR, or with SD lasting ≥24 weeks.
BOR, best overall response; CBR, clinical benefit rate; CI, confidence interval; CR, complete response; DCR, disease control rate; DOR, duration of response; ex20ins, exon 20 insertions; ICR, independent central review; ORR, objective response rate; PD, progressive disease; PR, partial response; SD, stable disease.

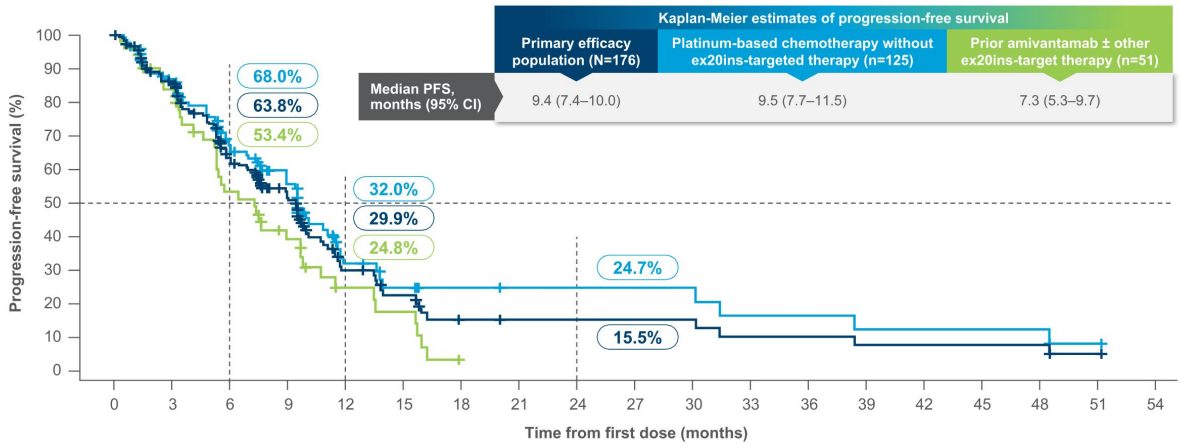
REZILIENT1: Confirmed ORR of 35.2% in the primary efficacy population; best change from baseline of target lesions



	ORR, % (95% CI)
Prior platinum-based chemotherapy (n=125)	40 (31–49)
Prior amivantamab without other ex20ins (n=30)	30 (15–49)
Prior amivantamab and other ex20ins (n=21)	14 (3–36)
Primary efficacy population (N=176)	35 (28–43)

CI, confidence interval; ex20ins, exon 20 insertions; ORR, objective response rate.

REZILIENT1: Zipalertinib shows median progression-free survival (PFS) of 9.4 months per ICR in primary efficacy population



No. at risk																			
Total	176	144	95	57	22	15	7	6	6	6	6	4	4	3	3	3	3	1	0
Platinum-based chemotherapy only	125	103	71	42	15	10	7	6	6	6	6	4	4	3	3	3	3	1	0
Prior amivantamab ± other ex20ins	51	41	24	15	7	5	0	0	0	0	0	0	0	0	0	0	0	0	0

Progression-free survival was defined as the time between the day of the first dose of zipalertinib and the first documentation of progressive disease or death, whichever occurred earlier. CI, confidence interval; ex20ins, exon 20 insertions; ICR, independent central review; PFS, progression-free survival.

REZILIENT1: Most common treatment-related adverse events

Most common treatment-related adverse events were paronychia, rash, dermatitis acneiform, dry skin, and diarrhea

Any-grade TRAEs reported in ≥10% of patients, No. (%)	Any grade	Grade 3
Paronychia	94 (38.5)	0
Rash	74 (30.3)	6 (2.5)
Dermatitis acneiform	60 (24.6)	1 (0.4)
Dry skin	60 (24.6)	0
Diarrhea	53 (21.7)	5 (2.0)
Stomatitis	49 (20.1)	4 (1.6)
Anemia	48 (19.7)	17 (7.0)
Pruritus	44 (18.0)	1 (0.4)
Nausea	35 (14.3)	2 (0.8)
Rash maculopapular	34 (13.9)	3 (1.2)
Fatigue	29 (11.9)	0

- Anemia was the most common grade 3 TRAE
- Other grade ≥3 TRAEs reported in ≥5 patients included pneumonitis and rash (6 patients [2.5%] each), and alanine aminotransferase increased, diarrhea, and platelet count decreased (5 patients [2.0%] each)
- Twelve patients (4.9%) had treatment-related pneumonitis, 5 of whom had received prior immunotherapy
  - Grade 1, n=3; grade 2, n=3; grade 3, n=5; grade 5, n=1

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TRAE, treatment-related adverse event.

REZILIENT1: Pivotal Phase 2b study in 2L+ met the primary endpoint of ORR and DOR, demonstrated a manageable safety profile for zipalertinib

REZILIENT1[1]

Pivotal Phase 2b (met primary endpoint)

- **Prior platinum-based chemo only**
- **Prior platinum-based chemo + amivantamab +/- other exon20ins directed therapy**

Primary endpoint: ORR and DOR

Data

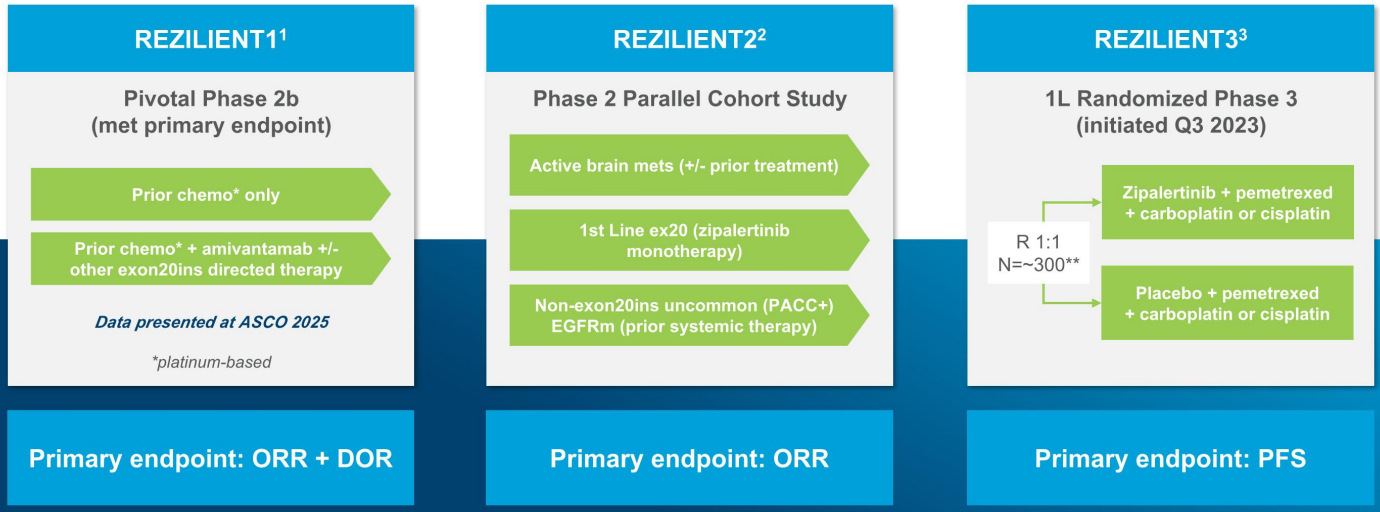
- REZILIENT1 pivotal Phase 2b study in 2L+ **met its primary endpoint of overall response rate** and **duration of response**
- Zipalertinib demonstrated **clinically meaningful efficacy**
- Zipalertinib demonstrated a **manageable safety profile**, **consistent with previously reported data**

Next steps

- These findings support zipalertinib as a **potential treatment option** for patients with EGFR ex20ins-mutant NSCLC after progression on platinum-based chemotherapy, including in the post-amivantamab setting, a significant and **growing unmet need**
- Updated efficacy and safety data in patients previously treated with amivantamab to be shared at the **IASLC 2025 WCLC in September 2025**
- Pending discussions with the U.S. Food and Drug Administration, potential NDA filling in by **YE 2025** by Taiho in relapsed EGFR ex20ins NSCLC.
- Randomized **REZILIENT3 Phase 3 frontline study ongoing;** complete enrollment expected in **H1 2026**

Clinicaltrials.gov identifiers: [1]NCT04036682, [2]NCT05967689 and 3NCT05973773; * includes both approved and investigational exon20 therapies
** following 6-12 patient safety lead in. PACC = P-loop and αC-helix

REZILIENT program: broad development of zipalertinib across multiple studies and indications in collaboration with Taiho Oncology



Primary endpoint: ORR + DOR

Primary endpoint: ORR

Primary endpoint: PFS

Clinicaltrials.gov identifiers: [1]NCT04036682, [2]NCT05967689 and 3NCT05973773; * includes both approved and investigational exon20 therapies ** following 6-12 patient safety lead in. PACC = P-loop and αC-helix

Potential best-in-class profile of zipalertinib creates opportunity to address large unmet need left by currently approved therapies

Despite the approval of amivantamab, an unmet need remains for well-tolerated oral targeted therapies with durable clinical benefit

	Zipalertinib	Amivantamab[1, 2]
Efficacy in patients treated with platinum-based chemotherapy	**40%**	**40%**
Median duration of response **Median PFS**	**8.8 months** **9.5 months**	**11.1 months** **8.3 months**
History of brain metastases	**35%**	**22%**
Route of administration	• Oral • 100mg twice daily	• IV infusion • Weekly for 5 weeks (split dose over 2 days, 1st week) • Then every 2 weeks • Premedicate with antihistamines and antipyretics for all doses and IV glucocorticoids during week 1
Select AEs (All / Grade 3+)	• Rash (30% / 3%) • Diarrhea (22% / 2%) • Anemia (20% / 7%) • ILD/Pneumonitis (5% / 2%)	• Rash (84% / 4%) • Diarrhea (16% / 3%) • Infusion reactions (64% / 3%) • ILD/Pneumonitis (3% / 1%) • Ocular toxicity (1% / -)

Data provided for context only; direct comparisons between molecules can not be made in the absence of head-to-head clinical trials.

1. https://www.janssenlabels.com/package-insert/product-monograph/prescribing-information/RYBREVANT-pi.pdf
2. Park K, Haura EB, Leighl NB, et al. Amivantamab in EGFR Exon 20 Insertion-Mutated Non-Small-Cell Lung Cancer Progressing on Platinum Chemotherapy: Initial Results From the CHRYSALIS Phase I Study. J Clin Oncol. 2021;39(30):3391-3402. doi:10.1200/JCO.21.00662

Taiho zipalertinib collaboration provides Cullinan with financial and strategic benefits

Upfront Payment
$275 million to Cullinan received in 2022 in exchange for providing 50% of U.S. and 100% of ex-U.S. rights to Taiho[1]

Milestone Payments
Cullinan is eligible to receive up to $130 million in payments for EGFR exon 20 NSCLC U.S. regulatory milestones

Collaboration
Taiho and Cullinan entered into a U.S. co-development and co-commercialization agreement, providing Cullinan with a co-promote option

Profit Sharing
Parties will share 50/50 U.S. development costs and potential profits

1. Excludes rights to Japan, which were already held by Taiho, and rights to Greater China, which were previously licensed to Zai Labs

REZILIENT program: upcoming milestones and next steps

REZILIENT1 Update

Updated efficacy and safety data in patients previously treated with amivantamab to be shared at the **IASLC 2025 WCLC in September 2025**

REZILIENT3 status update

Expected to complete enrollment in **H1 2026**

Initial results from REZILIENT2 Cohort C to be shared at ESMO Congress 2025 in October 2025

Initial results from Cohort C of REZILIENT2, which is exploring **zipalertinib monotherapy in patients with active brain metastases** who may or may not have had prior treatment for advanced disease

Initial results from REZILIENT2 Cohort D to be shared at IASLC 2025 WCLC in September 2025

Initial results from Cohort D of REZILIENT3, which is **exploring zipalertinib monotherapy in patients with uncommon EGFR mutations, such as PACC**, who may or may not have had prior systemic therapy

NDA Submission in 2L+

Pending discussions with the U.S. Food and Drug Administration, potential NDA filling by **YE 2025** by Taiho in relapsed EGFR ex20ins NSCLC


CLN-049
FLT3xCD3 bispecific T cell Engager
cullinan
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Significant unmet need in adult AML

- The only curative therapy is intensive chemotherapy +/- stem cell transplantation
- Curative therapy remains out of reach for most AML patients: 85% patients >60 years old are ineligible for intensive chemotherapy
- Recent treatment advancements have not significantly improved the likelihood of cure for the majority of AML patients
- **A significant unmet need remains for –**
  - a broadly applicable therapy that can produce high rates of durable response
  - eradication of measurable residual disease (MRD) that portends relapse even when patients meet clinical criteria for complete remission

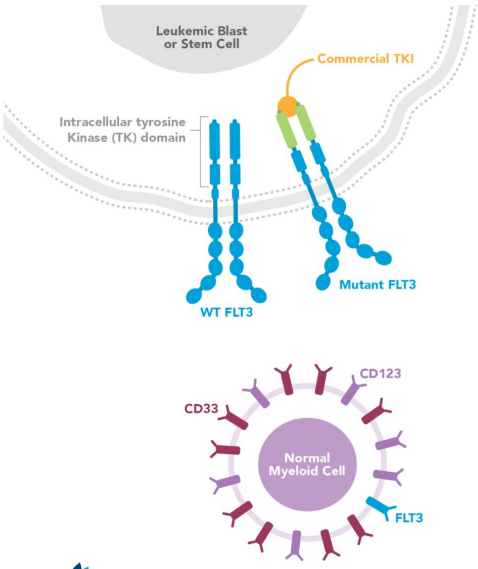
US AML Incidence **22,010**[1]

Average age at diagnosis **68**[1]

5-year survival **10% or less** in relapsed setting[2]

References 1. American Cancer Society (2025) **2.** DeWolf and Tallman. Blood 2020.

FLT3: An optimal target for AML immunotherapy



1. **Validation**: FLT3 plays a key role in promoting leukemic cell proliferation and survival. Tyrosine kinase inhibitors (XOSPATA®, RYDAPT®) treat 20-30% of AML by targeting only mutant FLT3

2. **Potential for treatment of broad AML population:** Targeting the extracellular domain of FLT3 could address ~80% of AML patients that express FLT3 on the cell surface, either wildtype or mutant forms

3. **Promising therapeutic potential:** FLT3 is expressed on leukemic stem cells as well as blast cells, which may increase response durability. Since FLT3 is an oncogenic driver, target loss is unlikely

4. **Potential for reduced toxicity risk:** FLT3 expression is very low on most mature normal myeloid cells compared to other frequently used targets for T cell engagers in AML like CD123 and CD33. FLT3 expression is very low on normal pluripotent stem cells

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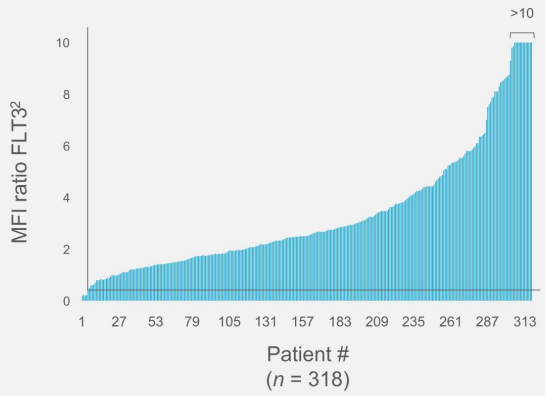
Compelling biological rationale for FLT3 targeted therapeutic approach in AML

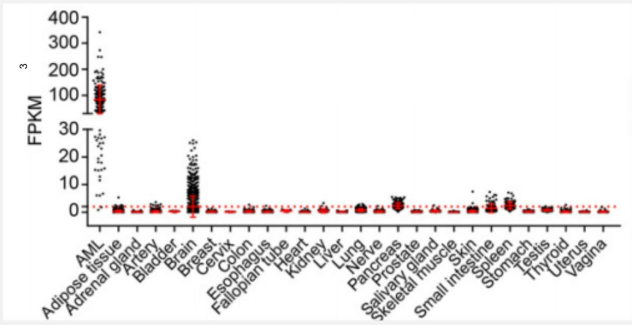
Most AML patients express FLT3 on blast cell surface[1]

Florescence Activated Cell Sorting (FACS) Analysis



Limited expression of FLT3 mRNA in normal tissues[1]

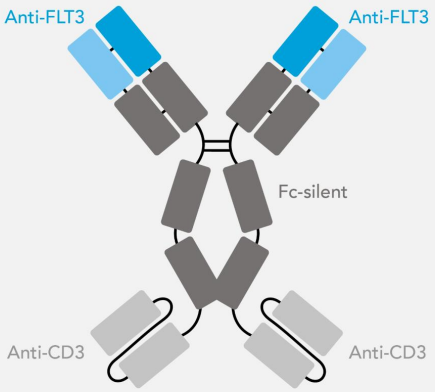
RNA



References 1. Brauchle B et al. Mol Cancer Ther 2020.
MFI = mean florescence intensity; FPKM = Fragments per kilobase of transcript per million mapped reads

CLN-049: A novel bispecific T cell engager targeting FLT3 in Phase 1 for r/r AML or MDS and in Phase 1 for MRD in AML

CLN-049 DESIGN



CLN-049'S POTENTIAL DIFFERENTIATION

Wide therapeutic window

- Two FLT3-binding Fab arms allow for higher avidity binding to AML cells, potentially increasing efficacy
- Two CD3 binding single-chain Fv domains are functionally monovalent to avoid aberrant T cell activation, potentially enhancing safety profile
- Silenced Fc domain avoids T cell activation by Fc-gamma receptor positive cells

Convenient dosing with low immunogenicity risk

- Half-life extended via Fc domain
- Humanized FLT3 and CD3 binding domains

Ease of manufacturing

- Symmetric IgG backbone is highly stable and enables high yield
- Format avoids aggregation risk that is sometimes present in other molecules (e.g. BiTEs)

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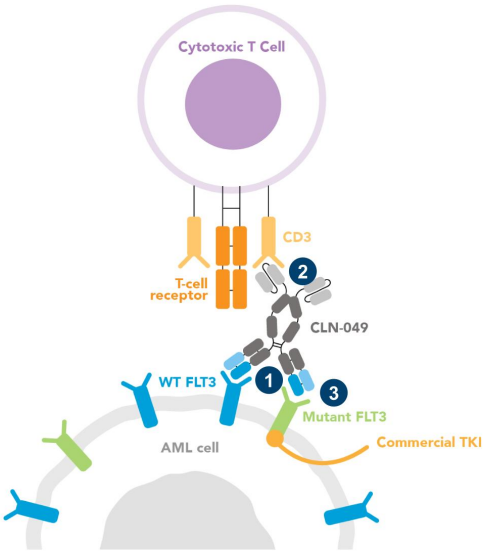
Statements based on preclinical data

CLN-049 mechanism potentially allows for broad FLT3 dependent AML blast killing

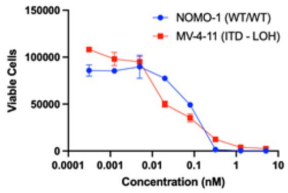


1. Redirects lysis of AML cells expressing mutant or wildtype FLT3
2. Functionally monovalent CD3 binding domains prevent T cell activation in absence of target cells
3. Two FLT3 binding domains drives potent elimination of AML blasts even at low FLT3 expression levels

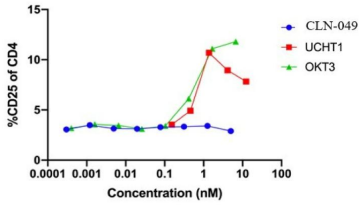
Statements based on preclinical data

CLN-049 preclinical data supports mechanism of FLT3-dependent T cell activation and broad AML cell elimination resulting in improved survival

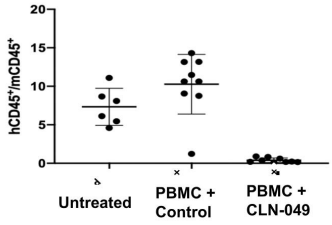
1 Potent redirected lysis of AML cell lines with wildtype or mutant FLT3



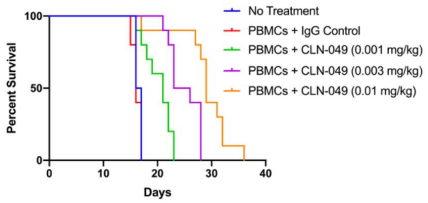
2 No T cell activation by CLN-049 in the absence of target cells



3 Potent elimination of patient-derived AML blasts by CLN-049 in mice



4 Increased survival of MOLM13 leukemia bearing mice at very low doses of CLN-049



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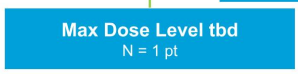
CLN-049 Phase 1 multiple ascending dose design in r/r AML or MDS

Intravenous dosing module ongoing

RELAPSED/REFRACTORY AML AND HYPOMETHYLATING AGENT (HMA)-R/R MDS

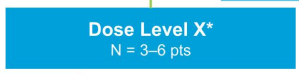
Accelerated Titration

Single Patient Cohort Escalation



Standard Titration

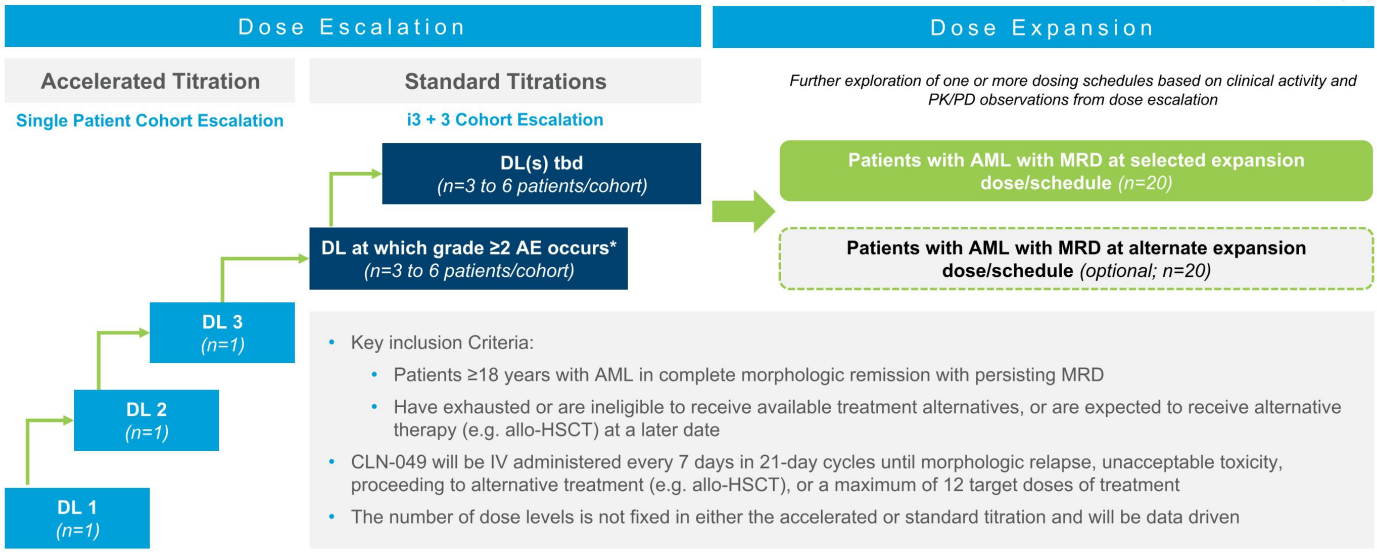
3 + 3 Cohort Escalation



- *Flip to standard titration for Treatment-Related AE (TRAE) Grade >= 2
- Flexibility to initiate step-use dosing for cytokine release syndrome (CRS) Grade >= 2
- The number of dose levels is not fixed in either the accelerated or standard titration and will be data driven

ClinicalTrials.gov Identifier: NCT05143996

CLN-049 Phase 1 study design in measurable minimal residual disease in AML



- Key inclusion Criteria:
  - Patients ≥18 years with AML in complete morphologic remission with persisting MRD
  - Have exhausted or are ineligible to receive available treatment alternatives, or are expected to receive alternative therapy (e.g. allo-HSCT) at a later date
- CLN-049 will be IV administered every 7 days in 21-day cycles until morphologic relapse, unacceptable toxicity, proceeding to alternative treatment (e.g. allo-HSCT), or a maximum of 12 target doses of treatment
- The number of dose levels is not fixed in either the accelerated or standard titration and will be data driven

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