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

FORM 6-K

REPORT OF FOREIGN ISSUER
PURSUANT TO RULE 13a-16 OR 15d-16
OF THE SECURITIES EXCHANGE ACT OF 1934

For the month of January 2023

(Commission File No. 001-40241)

LAVA Therapeutics N.V.
(Translation of registrant's name into English)

Yalelaan 60
3584 CM Ultrecht, The Netherlands
(Address of principal executive offices)

Indicate by check mark whether the registrant files or will file annual reports under cover Form 20-F or Form 40-F.
Form 20-F ☒ Form 40-F ☐

Indicate by check mark if the registrant is submitting the Form 6-K in paper as permitted by Regulation S-T Rule 101 (b) (1):
Yes ☐ No ☐

Indicate by check mark if the registrant is submitting the Form 6-K in paper as permitted by Regulation S-T Rule 101 (b) (7):
Yes ☐ No ☐

EXHIBIT LIST

Exhibit	Description
99.1	LAVA Therapeutics N.V. Investor Presentation

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, thereto duly authorized.

LAVA Therapeutics, N.V.

(Registrant)

Date: January 9, 2023

By: /s/ Fred Powell

Fred Powell

Chief Financial Officer

LAVA THERAPEUTICS

Gamma delta T cell engagers for the development of next-generation cancer therapeutics

Corporate presentation
January 2023

Legal Disclosure: Forward-looking Statements

This presentation contains statements that constitute forward-looking statements. Many of the forward-looking statements contained in this presentation can be identified by the use of forward-looking words such as “anticipate,” “believe,” “could,” “expect,” “should,” “plan,” “intend,” “estimate” and “potential,” and similar terms and phrases. Forward-looking statements appear in a number of places in this presentation and include, but are not limited to, statements regarding our intent, belief or current expectations. Forward-looking statements are based on our management’s beliefs and assumptions and on information currently available to our management. Such statements are subject to risks and uncertainties, and actual results may differ materially from those expressed or implied in the forward-looking statements due to various important factors. These risk and uncertainties include, among other things, the timing and results of our research and development programs, preclinical studies and clinical trials, including the timing of our clinical trials for LAVA-051 and LAVA-1207, and the submission of INDs or CTAs for our other product candidates; our ability to develop and obtain regulatory approval for and commercialize any of our product candidates; the failure of LAVA’s collaborators to support or advance collaborations or our product candidates; our ability to leverage our initial programs to develop additional product candidates using our Gammabody™ platform; and the risk that positive results in a preclinical study or clinical trial may not be replicated in subsequent trials or success in early stage clinical trials may not be predictive of results in later stage clinical trials. For a discussion of other risks and uncertainties, and other important factors, any of which could cause our actual results to differ from those contained in the forward-looking statements, see the "Risk Factors" section, as well as discussions of potential risks, uncertainties and other important factors, in the filings we make with the Securities and Exchange Commission from time to time.

Because forward-looking statements are inherently subject to risks and uncertainties, some of which cannot be predicted or quantified and some of which are beyond our control, you should not rely on these forward-looking statements as predictions of future events. Although we believe that we have a reasonable basis for each forward-looking statement contained in this presentation, the events and circumstances reflected in our forward-looking statements may not be achieved or occur and actual results could differ materially from those projected in the forward-looking statements. We qualify all of our forward-looking statements by these cautionary statements.

Any forward-looking statements represent the Company’s views only as of the date of this presentation and do not represent its views as of any subsequent date. The Company explicitly disclaims any obligation to update any forward-looking statements. By attending this presentation, you acknowledge and agree that you are cautioned not to place undue reliance on any forward-looking statements, and that you will conduct your own analysis and be solely responsible for forming your own view of the potential future performance of the Company.

Pioneering Next-Generation Cancer Therapeutics

Proprietary Gammabody™ platform

- Bispecific antibody platform to engage Vγ9Vδ2 T cells for highly specific tumor cell killing
  - Leverage the unique quality of Vγ9Vδ2 T cells to selectively kill tumor cells while sparing normal cells
  - Fully modular approach amenable to the use of existing and newly generated antibodies from any platform
  - Gammabody™ combines potent tumor cell killing with no activation of suppressor T cells, low potential for on-target/off-tumor toxicity, and cytokine release syndrome

Clinical stage company

- 2 programs in Phase 1/2a trials
  - LAVA-051 (CD1d), initial data released ASCO and ASH 2022. Additional data expected to be released H1 2023
  - LAVA-1207 (PSMA), first data to be presented at ASCO-GU (Q1 2023). Additional data expected to be released H2 2023

Robust pipeline

- LAVA-1266 (CD123) projected to enter the clinic in the next 2 years and LAVA-1223 (EGFR, licensed to Seagen)
- Multiple additional preclinical programs
  - Includes partnered discovery program with Janssen (J&J)

Solid financials and partnerships

- $142.7M (Q3 2022) in cash and investments; >24 months cash runway
- Collaborations with Janssen (J&J) and Seagen

Gammabody™ Pipeline: Potential in Hematological and Solid Tumor Indications

Candidate	Target	Indication(s)	Discovery	Preclinical	Phase 1	Phase 2	Milestones
LAVA-051	CD1d	MM CLL AML					• Additional data in 1H 2023
LAVA-1207	PSMA	mCRPC					• Phase 1 data Q1 2023 • Additional data 2H 2023
LAVA-1223	EGFR	Solid Tumors			Seagen		• Licensed to Seagen Sept 2022
LAVA-1266	CD123	Hematologic Malignancies					• IND/CTA filing expected in 2024
LAVA-1278	CD40	Hematologic Malignancies					
Janssen Collaboration	undisclosed		janssen				

MM: multiple myeloma
CLL: chronic lymphocytic leukemia
AML: acute myeloid leukemia
PSMA: prostate-specific membrane antigen
EGFR: epidermal growth factor receptor
mCRPC: metastatic castration-resistant prostate cancer

Hematologic malignancy Solid Tumor

Team Led by Experienced Leaders in the Biotech and Pharma Field

Steve Hurly, MSc, MBA
President & CEO

- 25+ years leadership experience in life sciences industry
- Seasoned drug developer and biotech strategist

Ton Adang, PhD
CDO

- Vast experience in drug development
- Extensive experience in product discovery and project management (e.g., KEYTRUDA)

Amy Garabedian, MSc, JD
General Counsel

- Extensive global, diversified legal and team building experience
- Almost 20 years practicing law, including over 15 years in the biotech and pharmaceutical industry

Paul Parren, PhD
EVP

- Industry leader in antibody science and drug development
- Vast experience inventing and developing therapeutic antibodies and technologies, including DARZALEX, RYBREVANT, TEPEZZA, TIVDAK & DuoBody

Fred Powell
CFO

- 20+ years of global CFO/leadership experience in biopharma
- Deep expertise across investor relations, finance, capital markets, operations and information technology

Hans van der Vliet, MD, PhD
CSO

- Inventor of LAVA's gamma delta T cell engager platform
- Medical oncologist, extensive experience in pre-clinical and clinical research

Benjamin Winograd, MD, PhD
CMO

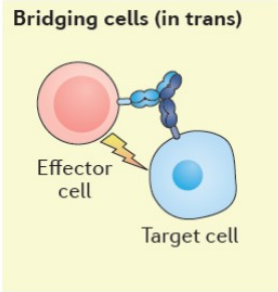
- Longstanding expertise in clinical research and drug development in hematology and oncology
- Instrumental in several registrations, including REVLIMID and POMALYST

LAVA's Proprietary Gammabody™ Platform

Bispecific Gamma Delta T Cell Engagers

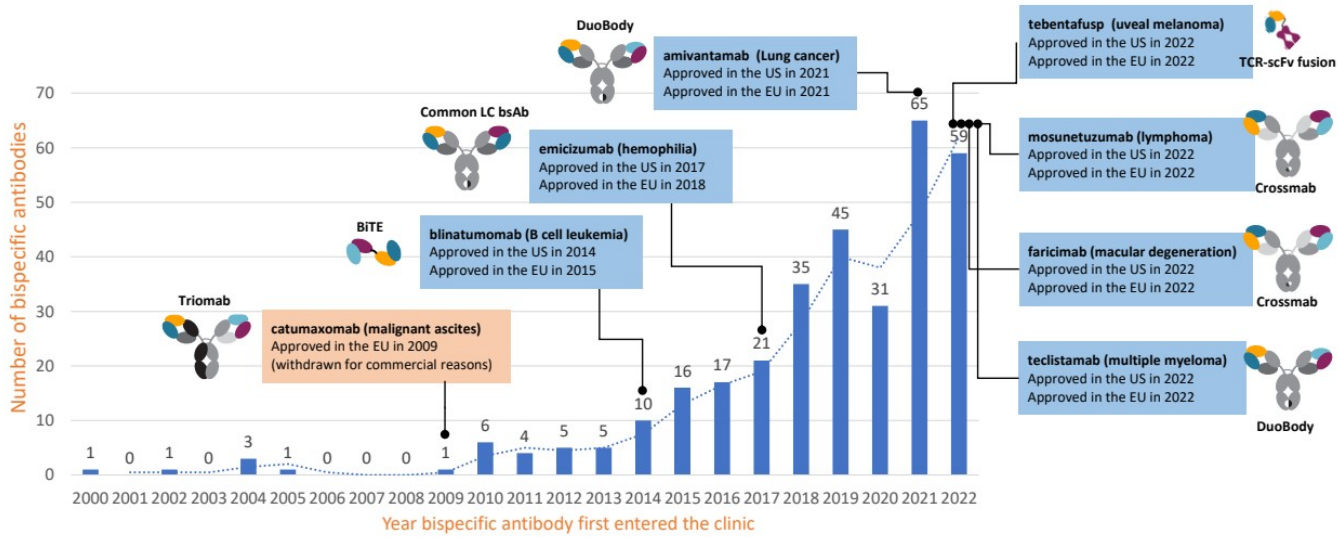
Enthusiasm for Bispecific T Cell Engagers

- High expectations for T cell bi-specific therapies driving significant development
- 200+ bispecific antibodies in the clinic
- Four marketed bispecific T cell engagers and many in the pipeline
  - **blinatumomab** (Blincyto); CD3 x CD19 bsTCE (2014)
  - **tebentafusp** (KIMMTRAK); CD3 x TCR-fusion targeting HLA-A2/gp100 (2022)
  - **mosunetuzumab** (Lunsumio); CD3 x CD20 bsTCE (2022)
  - **teclistamab** (TECVAYLI); CD3 x BCMA bsTCE (2022)
- Several bispecific T cell engagers in late-stage development
  - **epcoritamab**; CD3 x CD20 bsTCE in regulatory review
  - **talquetamab**; CD3 x GPCR5D bsTCE in regulatory review
  - **glofitamab**; CD3 x bivalent CD20 bsTCE in regulatory review
  - **elranatamab**; CD3 x BCMA bsTCE in phase III
  - 80+ bispecific T cell engagers currently in clinical development



Source: The Antibody Society; data as of Jan. 4, 2023
Labrijn, Janmaat, Reichert & Parren, Nature Reviews Drug Discovery, 2019, 18: 585-608

200+ Bispecific Antibodies in the Clinic
7 Approved

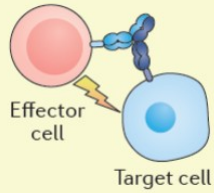


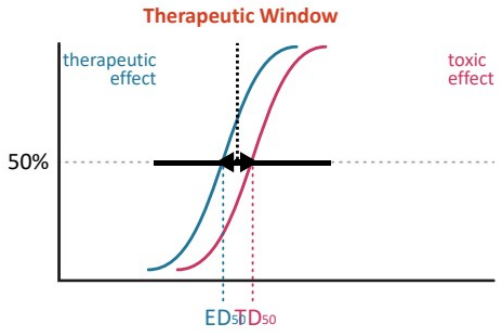
Source: The Antibody Society
Data as of Jan. 4, 2023

Bispecific T Cell Engagers

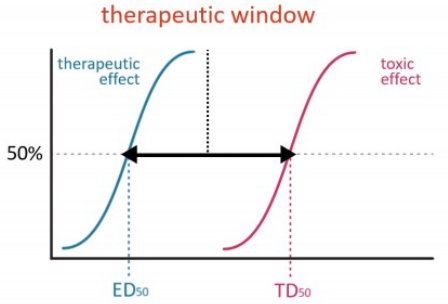
- High expectations for targeted T cell therapies in cancer, **but often:**
  - **Narrow therapeutic window:**
  - **Cytokine Release Syndrome**
  - **On-target/off-tumor**-related **toxicities**
  - Activates **immunosuppressive T cells**
  - **Sporadic efficacy** in **solid tumors**

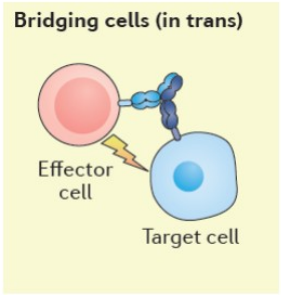
Bridging cells (in trans)





Strategies for Widening the Therapeutic Window





- Selecting 'tumor-specific' targets
- Step-dosing / subcutaneous dosing
- Decreasing affinity for T cells
- Masking/site-specific activation

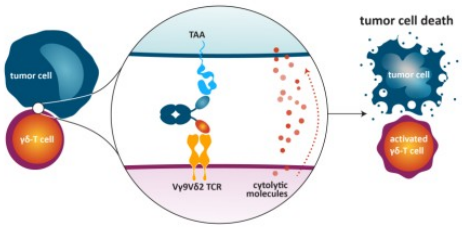
  - Address only narrow target range, and/or
  - Cumbersome, and/or
  - Strongly decrease potency

- **Recruiting alternative effector cells**

Gammabody™ Platform: Bispecific γδ T Cell Engagers

DIFFERENTIAL APPROACH

A versatile bispecific antibody platform for developing novel cancer therapeutics



MECHANISM OF ACTION

LAVA's proprietary bispecific antibodies are designed to:

- Target Vγ9Vδ2 T cells to tumor antigens initiating selective tumor cell killing while sparing normal cells.
- Carry a low potential for on-target/off-tumor toxicity and cytokine release syndrome (CRS).

OFF-THE-SHELF THERAPEUTICS

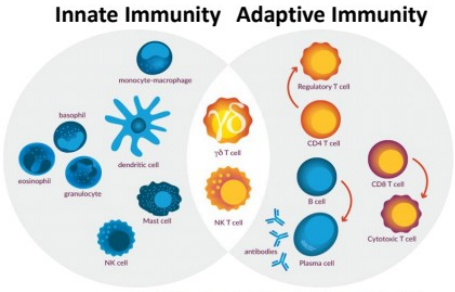
- ✓ Fully modular platform
- ✓ High developability
- ✓ Small size favors tumor penetration
- ✓ Proven quality of antibody products
- ✓ 2 formats in the clinic : bsVHH and bsVHH-Fc

Bispecific γδ T Cell-Engagers Aim to Harness Innate and Adaptive Immunity

Introducing Vγ9Vδ2 T cells

- Largest γδ-T cell subset in blood: (~90-95% of total γδ-T cells)
- Natural ability to recognize and kill tumor cells
- Highly cytotoxic
- Relatively abundant in tumor-infiltrating lymphocytes
- Presence of γδ T cells associated with improved outcomes in cancer patients

- Recognize tumors through phosphoantigen-BTN2A1/3A1 complex
- Consistent proinflammatory cytotoxic effector T cell population
- Does not contain immune-dampening regulatory T cell subsets
- Ability to present antigen and orchestrate immune responses

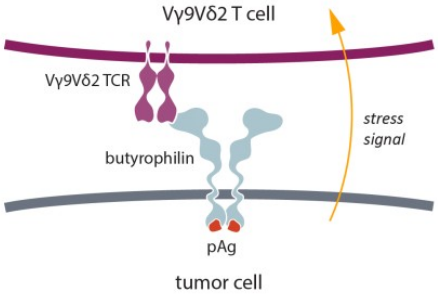


Adapted from Dranoff G., Nature Rev. Cancer 2004; 4: 11-22

Off-the-Shelf Gammabody™ Platform: Enhances Innate Tumor Recognition by Directing Vγ9Vδ2 T Cells to the Cancer Cells

Natural Activation Mechanism

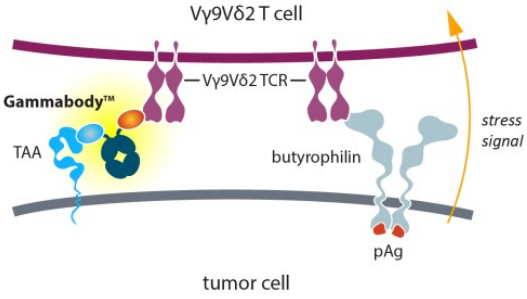
1 Vγ9Vδ2 T cells recognize stress signals TCR interacts with pAg-butyrophilin complex



Gammabody™ Provides Tumor Recognition to Trigger Vγ9Vδ2 T Cell-Mediated Immunity

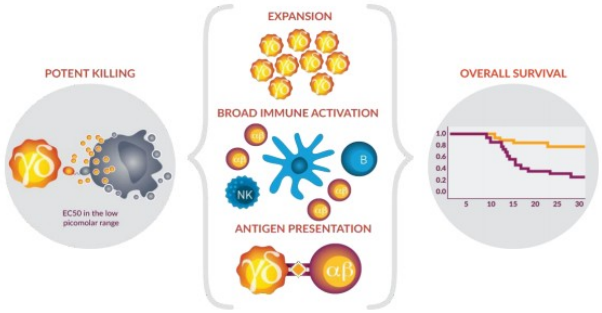
1 Conditionally activate Vγ9Vδ2 T cells upon crosslinking with tumor associated antigen (TAA)

2 Retains recognition of natural stress signals



LAVA's Gammabody™ directs Vγ9Vδ2 T cells to tumors with high affinity to induce direct tumor cell killing and orchestrate an immunological cascade of anti-cancer responses and while retaining tumor selectivity

Cascade of Anti-Cancer Responses – Potential Translation to Favorable Therapeutic Window



In addition to direct tumor cell killing, Vγ9Vδ2 T cells have the potential to orchestrate an immunological cascade response that includes activation of innate and adaptive immune cells in the tumor microenvironment

Efficacy

- Potent killing of cancer cells (EC_{50}s in the low picomolar range)
- No co-activation of immune-suppressive Tregs which dampen antitumor efficacy of cytotoxic T cells
- Orchestrate innate and adaptive immune responses, potentially resulting in potent and durable responses
- Activity against hematologic malignancies and solid tumors, including immunologically "cold" tumors
- Potential for expansion of Vγ9Vδ2 T cells can result in an increased number of anti-tumor Vγ9Vδ2 T cells in the tumor

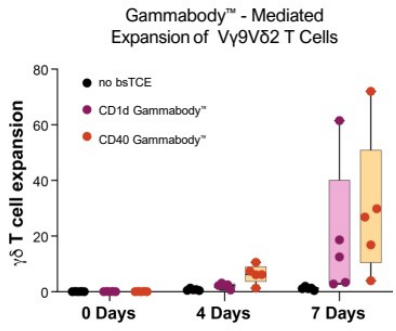
Safety

- Conditional activation with high accuracy
- Greatly reduced potential for cytokine release syndrome (CRS); no evidence of CRS in NHP studies

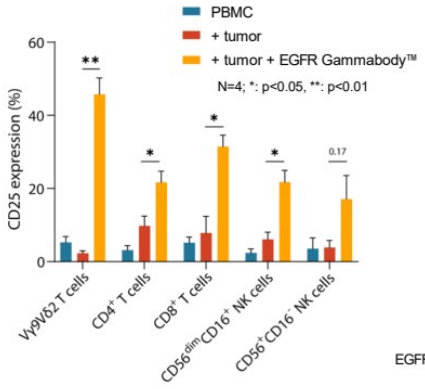
Adapted from Dranoff G, Nature Rev Cancer 2004; 4: 11-22 Kabelitz D et al., Cell Mol Immunol 2020; 17: 925–939

Expansion & Cascade Response Without Treg Activation in Preclinical Models

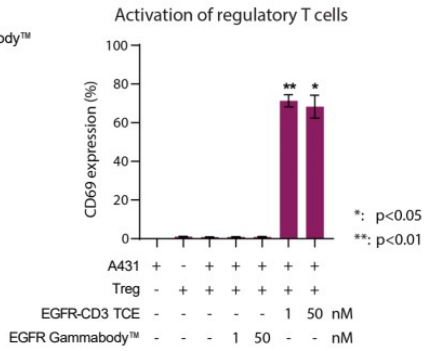
Expansion



Cascade Response



No Treg Activation

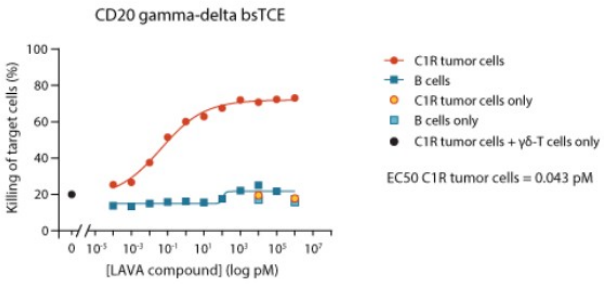


Gammabody™ can induce robust gamma delta T cell expansion and can amplify the anti- tumor immune response via downstream activation of other immune cells while avoiding co-activation of immunosuppressive T cells such as Tregs

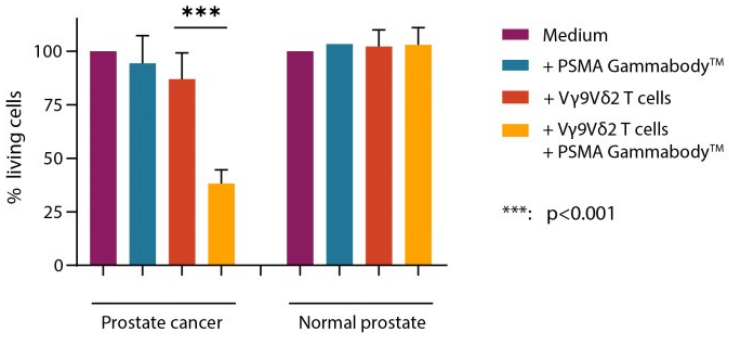
Data on file: LAVA Therapeutics N.V.

Gammabody™ Can Selectively Kill Cancer Cells While Sparing Healthy Cells in Hematologic Malignancy and Solid Tumor models

CD20 Gammabody™ Mediated Killing



PSMA Gammabody™ Mediated Killing



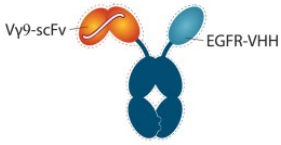
Preferential killing of cancer versus healthy cells demonstrated *in vitro* and *ex vivo*;
May prevent on-target/off-tumor mediated toxicity and allow for targeting of widely expressed tumor associated antigens

Data on file: LAVA Therapeutics N.V.

Non-Clinical Safety Data Indicate Good Tolerability

- Non-clinical safety studies using Gammabody™ molecules designed for cross-reactivity support the benign safety profile of the platform

- NHP studies completed with Gammabody™ molecules targeting CD1d, CD20 and EGFR
  - CD1d, CD20 targeting surrogate Gammabody™ (without Fc) were dosed up to 10 mg/kg (4 hr infusion, 4 doses, every 2 days) and biweekly at 1 mg/kg for 1 month
  - EGFR targeting surrogate Gammabody™ (without Fc) was dosed up to 10 mg/kg (4 hr infusion, 4 doses, every 2 days)
  - EGFR-targeting surrogate Gammabody™ (Fc-containing) was dosed up to 23 mg/kg (0.5 hr infusion, 4 weekly doses)



- **No signs of cytokine release syndrome, no changes in general health parameters, relevant clinical chemistry, hematology or histopathology observed**

- In stark contrast, EGFR-targeting is severely toxic for first generation bsTCEs
  - NHPs infused with a CD3xEGFR BiTE required euthanasia within 3 days at doses that were 200-fold lower (on a molar basis) compared to an EGFR Gammabody with cell death observed in all tissues expressing EGFR (Lutterbuese et al., PNAS 2010)

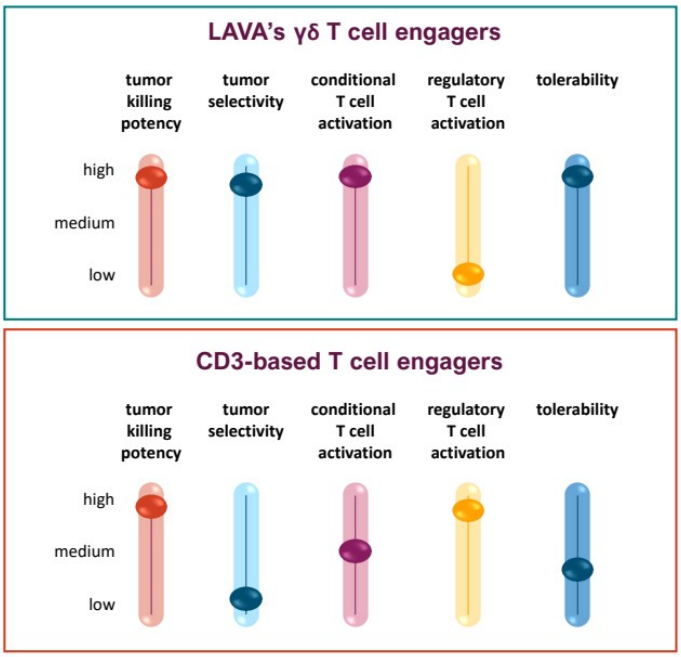
Gammabody™ Platform: A Novel T cell engager approach for cancer therapy

γδ T cell engager platform

- Highly potent (kills at picomolar concentrations)
- Recruits additional immune effector cells by antigen presentation and cascade response
- No activation of regulatory T-cells

- Tumor-cell selective, relative sparing of healthy cells expressing the target
- Low risk for on-target / off tumor toxicity
- Low risk for CRS anticipated
- Potential for a wide therapeutic window

- Applicable to hematological and solid tumor indications (including 'cold' tumors)



Clinical-stage company

LAVA-051

Targets CD1d to Activate Vγ9Vδ2 T Cells and iNKT Cells for the Potential Treatment of CLL, MM & AML

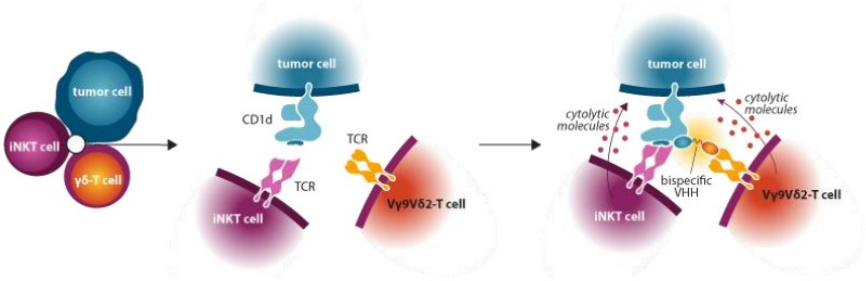
LAVA-051: First-in-Class Gammabody™ Targeting CD1d

Format

- Humanized bispecific single domain antibody (bsVHH) of 27kDa
  - Short plasma half-life, prolonged functional half-life through high-affinity TCR binding

Mechanism of Action

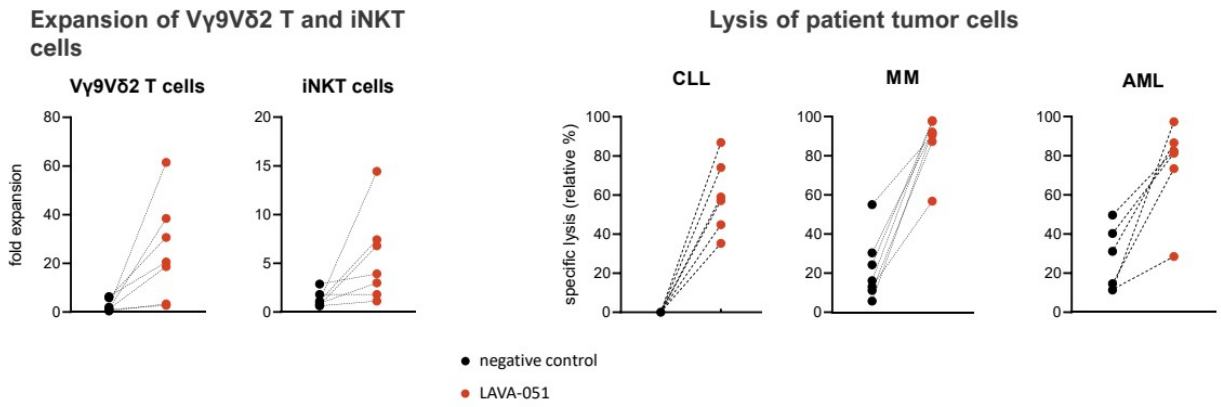
- Engages Vγ9Vδ2 T cells to mediate potent killing of CD1d-expressing tumor cells
  - Activates iNKT cells to mediate killing of CD1d-expressing tumor cells as a secondary mechanism of action
  - CD1d is expressed on tumor cells in CLL, MM and AML
  - Pre-clinical data support mechanism of action, anti-cancer activity, effector cell expansion and tumor selectivity



Status

- Phase 1/2a clinical trial ongoing in MM, CLL and AML

LAVA-051: Pre-Clinical Data Support Mechanism of Action and Function

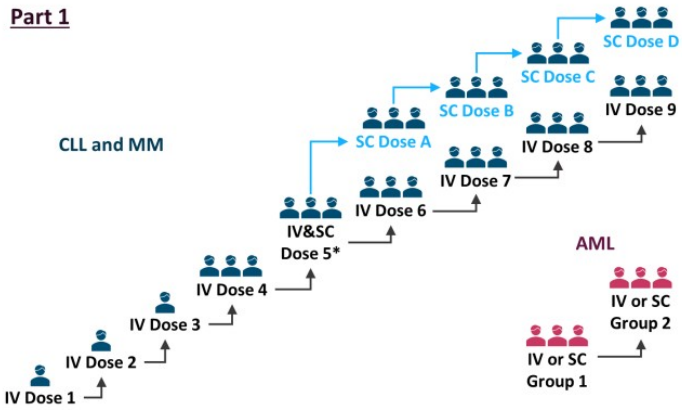


- LAVA-051 triggers expansion of Vγ9Vδ2 T and iNKT cells in the presence of CD1d-positive tumor cells
- LAVA-051 mediates Vγ9Vδ2 T and iNKT cell-mediated cytotoxicity of patient CLL, MM and AML cells

Adapted from Lameris et al., submitted
Data on file: LAVA Therapeutics N.V

LAVA-051 Phase 1/2a in Hematological Malignancies

- Primary objectives: investigate safety and tolerability of LAVA-051 and determine the recommended Phase 2 dose
- Secondary objectives: include evaluation of PK, PD, immunogenicity and preliminary antitumor activity
- LAVA-051 administered as 2-hour infusion (IV), or subcutaneous injection (SC) (day 1, 8 and twice a week thereafter)



* Cohort 5 only: 2nd dose administered SC, remaining doses IV

Clinicaltrials.gov NCT04887259

LAVA-051 – Initial Phase 1 Data - Adverse Events

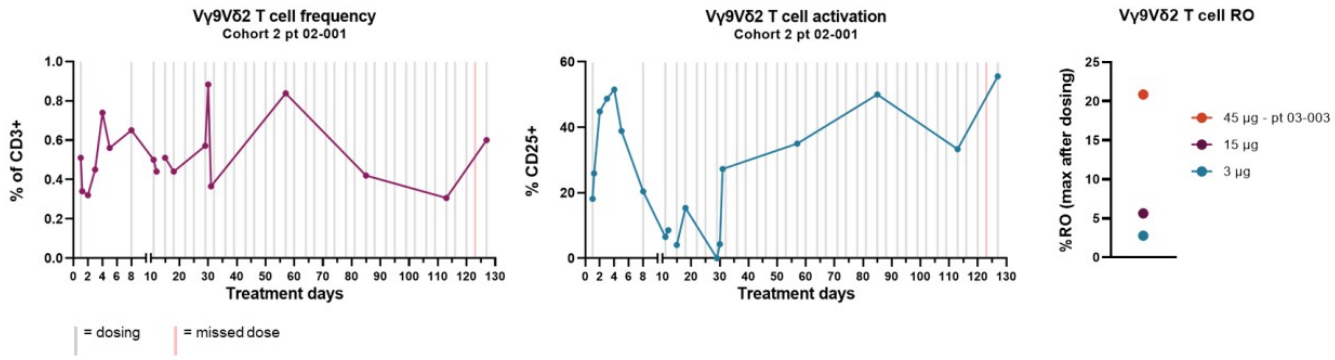
- LAVA-051 has reached a dose of 200 μg (~400x the starting dose) in MM and CLL patients
- Most observed AEs have not been suspected to be related
- Frequency and severity of AEs have not correlated with increasing dose levels
- No CRS and no ICANS (ASTCT) and no clinically relevant increase in the CRS-related cytokine IL-6

(Data cut-off date: 11 Nov 2022)

ASH 2022 abstract #2014

ICANS = Immune Effector Cell Associated Neurotoxicity Syndrome;
DLT = Dose Limiting Toxicity; ASTCT = American Society for Transplantation and Cellular Therapy
Data on file: LAVA Therapeutics N.V

LAVA-051 – Initial Phase 1 Data - Pharmacodynamics

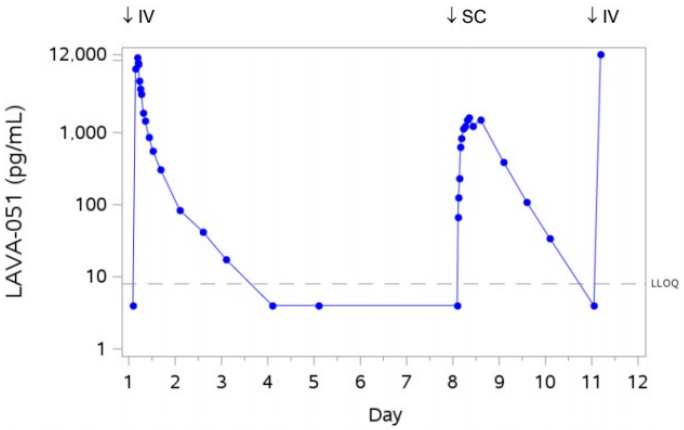


- Pharmacodynamic parameters reflect changes expected for the LAVA-051 mechanism of action
  - Vγ9Vδ2-T cell activation markers (CD25 and CD69) upregulated following dosing
  - Maximum Vγ9Vδ2 T cell receptor occupancy (RO) increased with dose

ASCO 2022 abstract 2577; ASH 2022 abstract #2014
Data on file: LAVA Therapeutics N.V

LAVA-051 - Pharmacokinetics

Pharmacokinetics 1st dose IV, 2nd dose SC patient 32-001 cohort #5



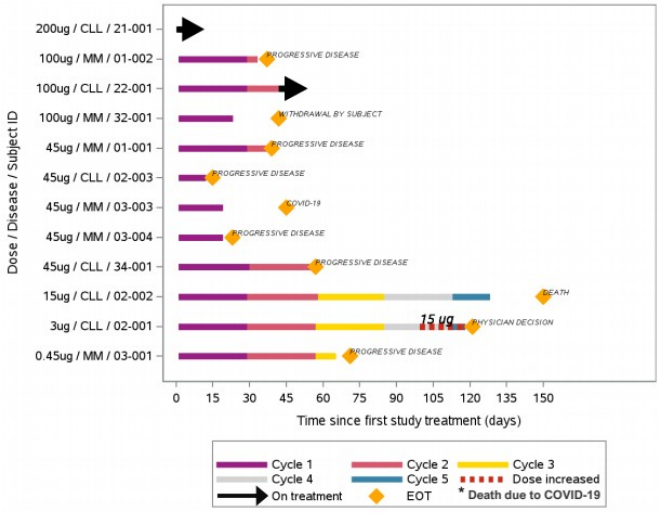
- Linear LAVA-051 pharmacokinetics
- SC bioavailability 74% compared to IV (based on data from Pt 32-001)

ASH 2022 abstract #2014
Data on file: LAVA Therapeutics N.V

LAVA-051 – Initial Phase 1 Data - Patient Characteristics and Time on Treatment

MM/CLL	6/6
Male/Female	8/4
Median age (range)	69 (59-76)
Prior therapies, median (range) – MM/CLL	4 (3-5) / 5.5 (4-13)

Data cut-off: 11 NOV 2022

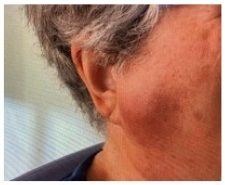


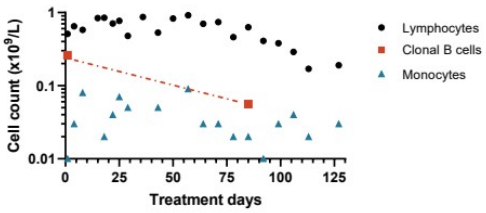
ASH 2022 abstract #2014, corrected
Data on file: LAVA Therapeutics N.V

LAVA-051 – Initial Phase 1 Data - Potential Signs of Activity

CLL

- Patient with R/R CLL (15 μg)
- Temporary enlargement and tenderness of several involved lymph nodes accompanied by grade 2 fever during Cycle 1
  - Resembled a tumor-flare reaction, as reported in CLL with lenalidomide
- **Patient assessed as having stable disease**
- **Percent of clonal B cells in peripheral blood decreased**
- **Numbers of CD1d expressing monocytes remained similar**





MM

- High-risk MM patient (45 μg)
- 4 prior lines of therapy within 6 years from diagnosis
- Refractory to last 3 lines of treatment
- **23% reduction in M-protein**

- Both patients ceased treatment due to COVID

EHA 2022 abstract #1463
R/R = Relapsed/Refractory
Permission for photo obtained
Data on file: LAVA Therapeutics N.V

LAVA-051: Summary of Initial Phase 1 Data Presented

- LAVA-051 is a next-generation bispecific γδ T cell engager designed for a broad therapeutic window
- LAVA-051 has reached a dose of 200 μg (400x the starting dose) in MM and CLL patients
  - Most observed Adverse Events (AEs) have not been suspected to be related to LAVA-051 treatment
  - Frequency and severity of AEs have not correlated with increasing dose levels
  - No Cytokine Release Syndrome (CRS) and no ICANS (ASTCT - criteria)
  - No significant increase in the CRS-related cytokine IL-6
- Linear pharmacokinetics and satisfactory SC bioavailability
- PD parameters reflect changes as expected per Mechanism of Action
- Potential signs of clinical activity
- Trial continuing, including US sites (IND cleared) and evaluation of SC dosing

ICANS: Immune Effector Cell Associated Neurotoxicity Syndrome
ASTCT: American Society for Transplantation and Cellular Therapy
DLT: Dose Limiting Toxicity

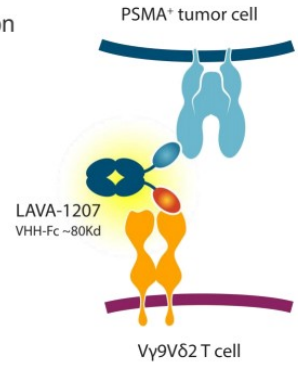
LAVA-1207

Gammabody™ that Activates Vγ9Vδ2 T Cells by Targeting PSMA for the Treatment of mCRPC

LAVA-1207: PSMA-targeting Gammabody™ for Prostate Cancer

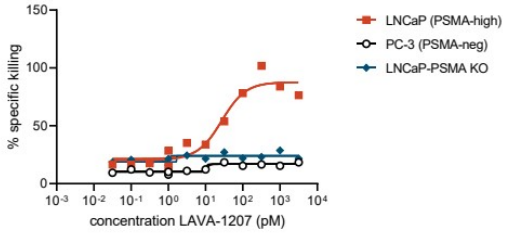
Format

- Contains a Fc domain for extended plasma half-life; silenced to avoid off-target T cell activation
- Small size (compared to regular IgG antibodies) to facilitate tumor penetration



Mechanism of Action

- Specifically directs Vγ9Vδ2 T cells to PSMA-expressing tumor cells
  - PSMA is a well-validated tumor target
- Mediates potent killing of PSMA-positive tumor cells
- Pre-clinical data support mechanism of action, anti-cancer activity & selectivity



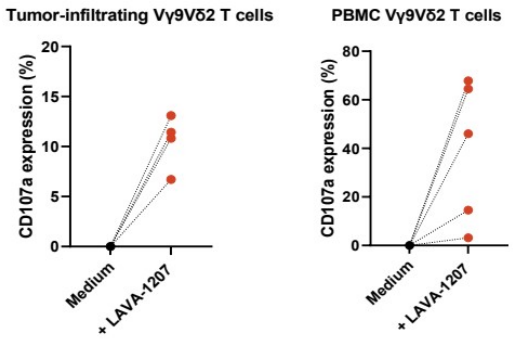
Status

- Phase 1/2a trial in mCRPC; patient recruitment ongoing (NCT05369000)

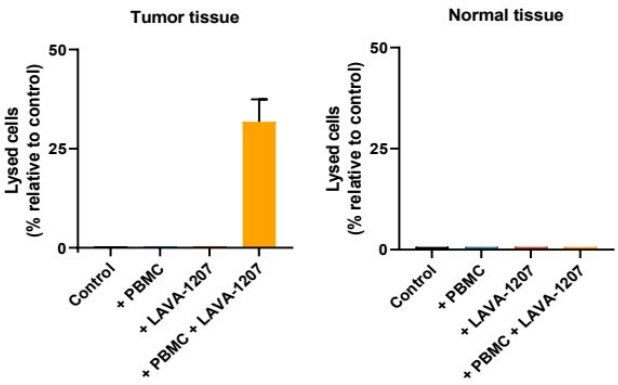
Data on file: LAVA Therapeutics N.V

LAVA-1207: Preclinical Data Support Activity and Selectivity in Patient Samples

Vγ9Vδ2 T cell degranulation



Preferential lysis of prostate tumor cells

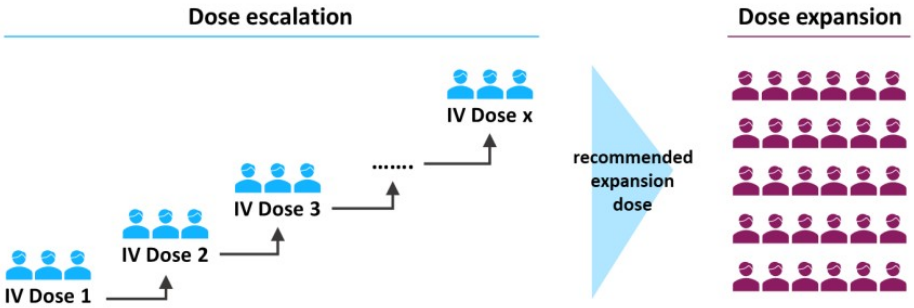


- LAVA-1207 triggers activation of autologous Vγ9Vδ2 T cells in the presence of patient-derived tumor cells
- LAVA-1207 induces selective tumor cell lysis

Data on file: LAVA Therapeutics N.V

LAVA-1207 Phase 1/2a in mCRPC

- Primary objective: investigate safety and tolerability of LAVA-1207 and determine recommended dose and schedule based on optimal biological dose
- Secondary objectives include evaluation of PK, PD, immunogenicity and preliminary antitumor activity
- LAVA-1207 administered as IV infusion, every 2 weeks



Clinicaltrial.gov NCT05369000

LAVA-1223 – Licensed to Seagen

Gammabody™ for the treatment of EGFR-expressing solid tumors

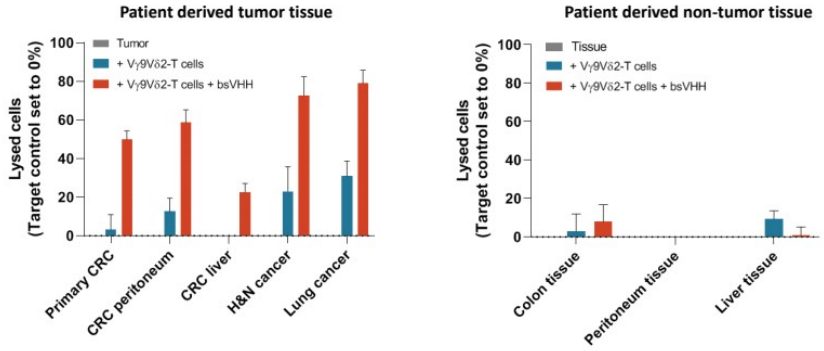
LAVA-1223: EGFR-Targeting Gammabody™

Format

- Gammabody™ format containing a silenced Fc domain

Mechanism of Action

- Induces preferential lysis of EGFR-expressing tumor cells while relatively sparing EGFR-expressing normal cells



Status

- Exclusive worldwide license agreement with Seagen Inc.
- Seagen to develop and commercialize LAVA-1223, potential for milestones of up to approximately $650 million and royalties

King et al., submitted
Data on file: LAVA Therapeutics N.V

LAVA-1266

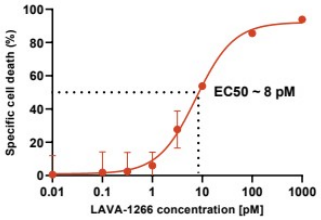
CD123 Targeting Gammabody™ for the Treatment of Hematologic Malignancies

LAVA-1266: CD123-Targeting Gammabody™
In Development for Treating Hematological Malignancies

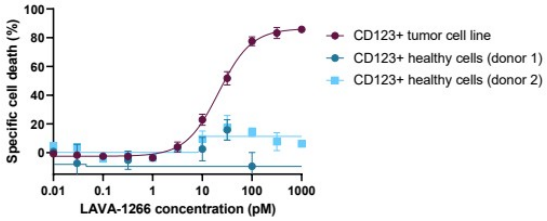
Mechanism of Action

- Induces preferential lysis of CD123-expressing tumor cells while relatively sparing CD123-expressing normal cells
  - CD123 is overexpressed in a wide range of hematological malignancies

Potent lysis of primary AML cells



Preferential lysis of tumor cells



Status

- CTA/IND enabling studies ongoing; filing anticipated in 2024

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Milestones

Gammabody™ Pipeline: Potential in Hematological and Solid Tumor Indications

Candidate	Target	Indication(s)	Discovery	Preclinical	Phase 1	Phase 2	Milestones
LAVA-051	CD1d	MM CLL AML					• Additional data in 1H 2023
LAVA-1207	PSMA	mCRPC					• Phase 1 data Q1 2023 • Additional data 2H 2023
LAVA-1223	EGFR	Solid Tumors			Seagen		• Licensed to Seagen Sept 2022
LAVA-1266	CD123	Hematologic Malignancies					• IND/CTA filing expected in 2024
LAVA-1278	CD40	Hematologic Malignancies					
Janssen Collaboration	undisclosed		janssen				

MM: multiple myeloma
CLL: chronic lymphocytic leukemia
AML: acute myeloid leukemia
PSMA: prostate-specific membrane antigen
EGFR: epidermal growth factor receptor
mCRPC: metastatic castration-resistant prostate cancer

Hematologic malignancy Solid Tumor

LAVA THERAPEUTICS