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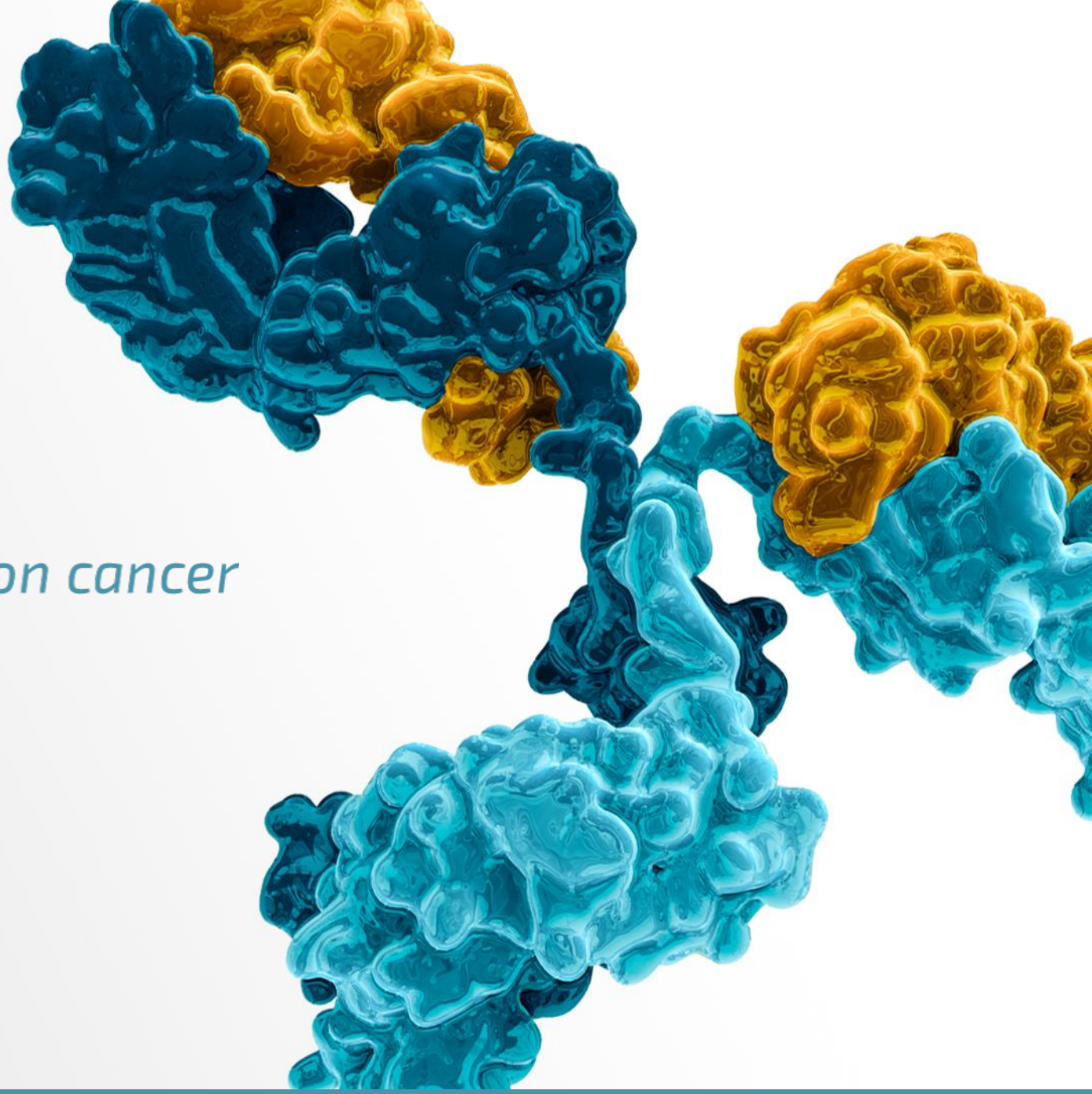
closing in on cancer



Corporate Presentation

September 2022

Disclaimer

This presentation (including any oral commentary that accompanies this presentation) contains forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995. All statements contained in this presentation that do not relate to matters of historical fact should be considered forward-looking statements, including without limitation statements regarding the impact our Biclonics® and Triclonics® platforms can have on cancer, our product candidates' potential to treat certain types of tumors, the timing of regulatory filings and the timing and anticipated data read outs or results from our clinical trials and our collaborations, and anticipated cash runway. These forward-looking statements are based on management's current expectations. These statements are neither promises nor guarantees, but involve known and unknown risks, uncertainties and other important factors 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, including, but not limited to, the following: we have incurred significant losses, are not currently profitable and may never become profitable; our need for additional funding, which may not be available and which may require us to restrict out operations or require us to relinquish rights to our technologies or antibody candidates; potential delays in regulatory approval and impacts of the COVID-19 pandemic, which would impact our ability to commercialize our product candidates and affect our ability to generate revenue; the unproven approach to therapeutic intervention of our Biclonics®, and Triclonics® technology; our limited operating history; economic, political, regulatory and other risks involved with international operations; the lengthy and expensive process of clinical drug development, which has an uncertain outcome; the unpredictable nature of our early stage development efforts for marketable drugs; potential adverse public reaction to the use of cancer therapies; potential delays in enrollment of patients, which could affect the receipt of necessary regulatory approvals; failure to obtain marketing approval internationally; failure to compete successfully against other drug companies; potential competition from other drug companies if we fail to obtain orphan drug designation or maintain orphan drug exclusivity for our products; our reliance on third parties to conduct our clinical trials and perform clinical development and the potential for those third parties to not perform satisfactorily; our reliance on third parties to manufacture our product candidates, which may delay, prevent or impair our development and commercialization efforts; protection of our proprietary technology; our patents being found invalid or unenforceable; potential lawsuits for infringement of third-party intellectual property; our ability to attract and retain key personnel; managing our growth could result in difficulties.

These and other important factors discussed under the caption “Risk Factors” in our in our Annual Report on Form 10-Q for the period ended June 30, 2022 filed on August 8, 2022 with the Securities and Exchange Commission, or SEC, and our other reports filed with the SEC, could cause actual results to differ materially from those indicated by the forward-looking statements made in this presentation. Any such forward-looking statements represent management's estimates as of the date of 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.

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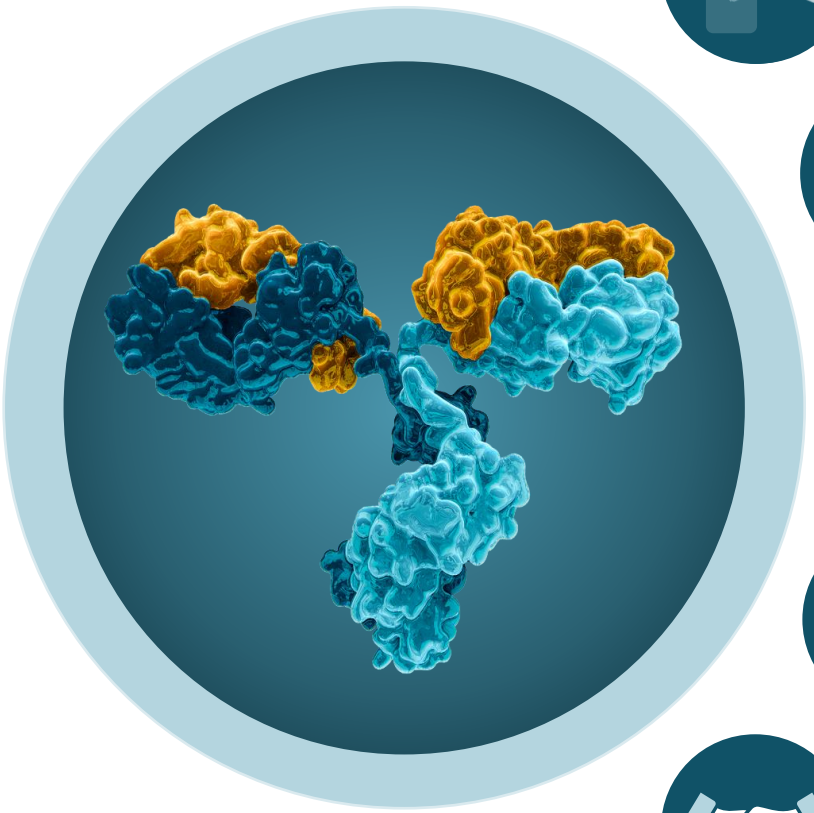
Merus Overview



Oncology-focused Company Developing Multispecific Antibody Therapies

Bispecific and trispecific cancer therapeutic candidates based on the human IgG format

Established Clinical Pipeline

Four clinical stage assets with proof of concept data on Zeno in NRG1-fusion (NRG1+) cancer[1], and early encouraging data on Peto in Head and Neck cancer[2]

Leading Multispecific Antibody (Multiclonics®) Platforms

Common light chain format permits broad high throughput evaluation of Biclonics® and Triclonics®, to develop clinical stage assets with meaningful clinical responses in patients

Near Term Trial Updates and Strong Cash Position Beyond 2024*

Upcoming clinical milestones and program updates planned over the next 12-18 months: Zeno registration-directed program, MCLA-129 initial clinical data, and Peto clinical update

Strategic Collaborations to Unlock Platform Value

Multiple strategic collaborations and license agreements

[1] *Schram et al., ASCO 2022, efficacy data cutoff April 12, 2022 and safety data cutoff January 12, 2022*

[2] *Hollebecque et al., AACR-NCI-EORTC 2021, data cutoff August 9, 2021*

*See August 8, 2022 10-Q noting our belief that our cash, cash equivalents and marketable securities, will fund our operations beyond 2024.

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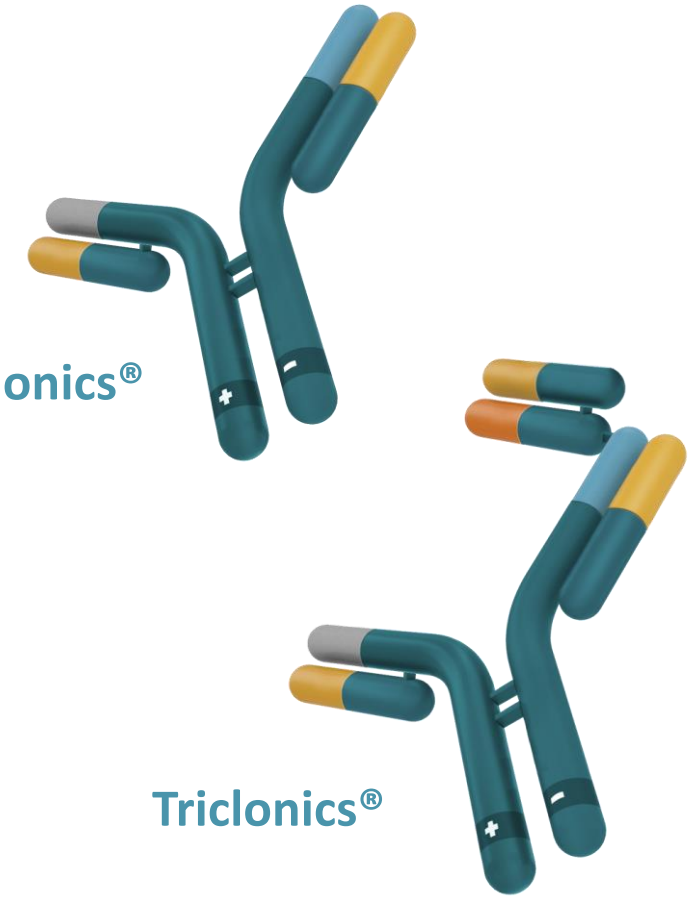
Merus Multiclonics®

Bispecific and Trispecific therapeutic candidates for cancer with broad application for human disease



Large-scale screening

- *To select the best Biclonics® and Triclonics® from up to 1,000s of candidates*

Fully human IgG format

- *Ease of manufacturing*
- *Low immunogenicity risk*
- *Predictable in vivo behavior*
- *Durable, consistent half life*
- *Potential for ADCC enhancement and Fc silencing*

Robust IP portfolio

Patents covering Multiclonics® technology, including

- *Common light chain antibody generation and screening*
- *Dimerization by charge engineering*

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Merus Clinical Pipeline

PROGRAM	BISPECIFIC TARGETS	INDICATION(S)	PRECLINICAL	PHASE 1	PHASE 1/2	STATUS
Zenocutuzumab (Zeno) (MCLA-128)	HER2 x HER3	NRG1+ cancer				• Phase 1/2 registration-directed trial on-going
		with afatinib in NRG1+ NSCLC				
		Castration resistant prostate cancer				
		Other cancers				
Petosemtamab (Peto) (MCLA-158)	LGR5 x EGFR	Solid tumors				• Phase 1 trial ongoing • Clinical update planned 1H23
MCLA-145	CD137 x PD-L1	Solid tumors				• Phase 1 trial ongoing
		with a PD1 inhibitor in solid tumors				
MCLA-129	EGFR x c-MET	Solid tumors	BETTA (China)			• Phase 1/2 trial ongoing • Clinical update planned: EORTC/NCI/AACR 2022
		with osimertinib in NSCLC				
ONO-4685*	PD-1 x CD3	Relapsed/Refractory T Cell Lymphoma; Psoriasis	ONO			• Phase 1 trial ongoing
INCA32459*	LAG3 x PD-1	Not disclosed	Incyte			• Clinical program expected to begin in 2022**

** If commercialized, Merus to receive royalties*
*** Incyte presentation dated August 2, 2022*

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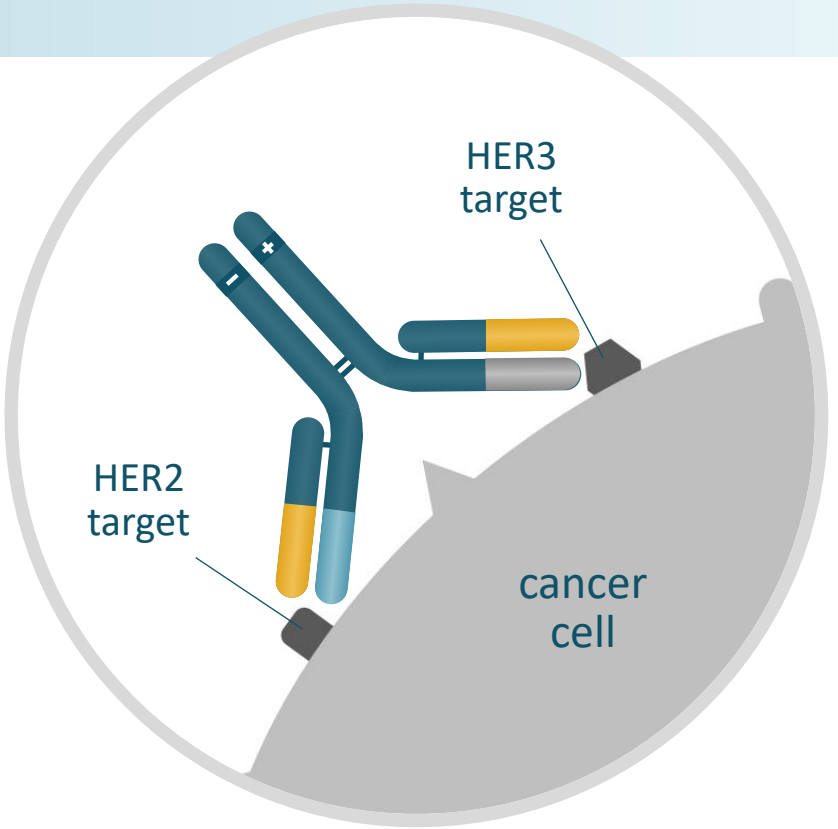
Potential first in class and best in class for NRG1 fusion (NRG1+) cancer

Zenocutuzumab

MCLA-128 or "Zeno"
HER2 x HER3 bispecific

- **NRG1 fusions**
  - Translocations of the Neuregulin-1 (NRG1) gene, also called Heregulin, the ligand for the HER3 receptor. Rare events in solid tumors reported to typically occur in the absence of other cancer driver mutations[1]
  - Reported as associated with poor prognosis[1], lower response rates to standard therapy[2], and shorter overall survival in lung cancer[1,3]
- **Zeno**
  - Biclonics® antibody binds to HER2 and blocks HER3; 100-fold more potent *in vitro* than an anti-HER3 antibody derived from Zeno alone
  - Enrollment with clinical follow up completed to potentially support a Biologics License Application for a tumor agnostic indication in NRG1+ cancer
  - Planned initiation of a clinical trial evaluating Zeno with afatinib for NRG1+ NSCLC, and other non-NRG1+ solid tumors, including CPRC[4]
  - Granted orphan and fast track designation by FDA for pancreatic cancer, and NRG1+ cancer post standard of care, respectively



[1] Chang et al., Clin Cancer Research 2021, [2] Drilon et al., J Clin Oncol 2021, [3] Shin et al., Oncotarget 2016, [4]Castration-resistant prostate cancer

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Zeno DOCK & BLOCK® Mechanism Potently Blocks NRG1 fusions

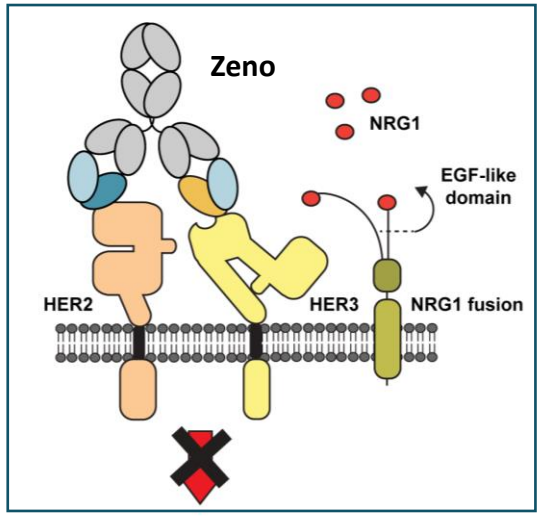
Zeno

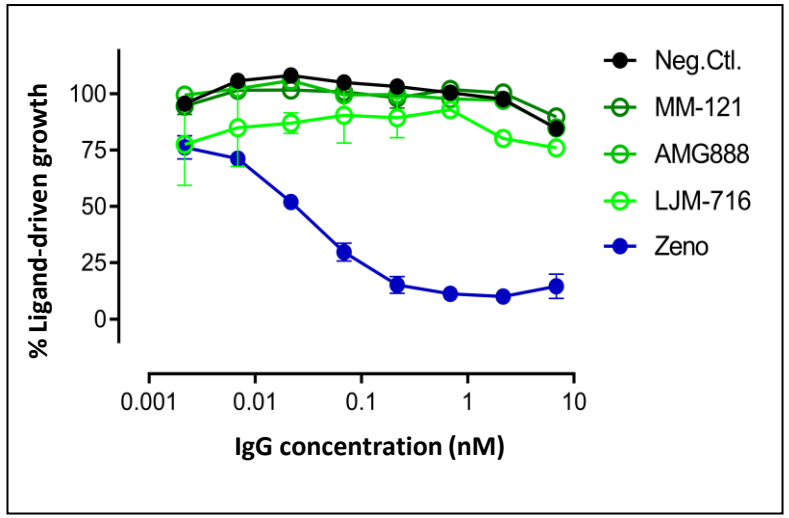
Common light chain bispecific Biclonics® antibody

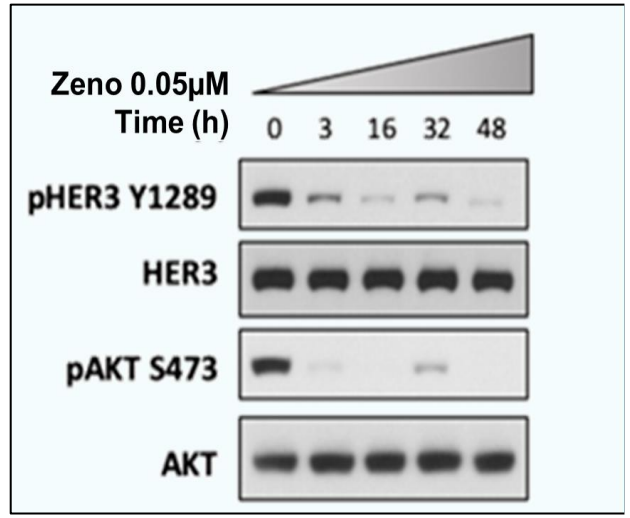
DOCKS
onto the more abundant HER2 protein leads to high local concentration on the cell surface

BLOCKS
- NRG1 fusion interaction with HER3
- HER3 from interacting with HER2
- Growth signals in cells

INDUCES
enhanced ADCC (Antibody-Dependent Cellular Cytotoxicity)







Growth of N87 cells with 12.5 nM HRG and a titration of the indicated antibodies.

Unique-Targeting of NRG1+ Cancer

Shown to Potently Inhibit Growth and NRG1:HER3 Signalling Preclinically

Geuijen et al. Cancer Cell. 2018;33:922-36; Odintsov et al. AACR. 2021; abstract 956; Schram et al., ASCO 2022,

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Oral presentation at ASCO 2022[1]

Efficacy and safety of zenocutuzumab, a HER2 x HER3 bispecific antibody, in advanced *NRG1* fusion-positive (NRG1+) cancer

- **Alison M Schram[1],** Koichi Goto[2], Dong-Wan Kim[3], Patricia Martin-Romano[4], Sai-Hong I Ou[5], Grainne M O'Kane[6], Eileen M O'Reilly[1], Kumiko Umemoto[7], Michaël Duruisseaux[8], Cindy Neuzillet[9], Frans Opdam[10], Jordi Rodon Ahnert[11], Misako Nagasaka[12], Benjamin A Weinberg[13], Teresa Macarulla[14], Andrew K Joe[15], Jim Ford[15], Viktoriya Stalbovskaya[15], Ernesto Wasserman[15], Alexander E Drilon[1]
- **1**. Memorial Sloan Kettering Cancer Center, New York, NY, USA; **2**. National Cancer Center Hospital East, Kashiwa, Japan; **3**. Seoul National University Hospital, Seoul, South Korea; **4**. Gustave Roussy Cancer Campus, Villejuif, France; **5**. Chao Family Comprehensive Cancer Center, University of California Irvine, Orange, CA, USA; **6**. Princess Margaret Cancer Centre, Toronto, ON, Canada; **7**. St. Marianna University School of Medicine, Kawasaki, Japan; **8**. URCOT, Hôpital Louis Pradel, Hospices Civils de Lyon Cancer Institute, Lyon, France; **9**. Curie Institute, Saint-Cloud, France; **10**. Netherlands Cancer Institute, Amsterdam, Netherlands; **11.**The University of Texas MD Anderson Cancer Center, Houston, TX, USA; **12**. Karmanos Cancer Institute, Detroit, MI, USA; **13**. Ruesch Center for the Cure of Gastrointestinal Cancers, Lombardi Comprehensive Cancer Center, Georgetown University Medical Center, Washington, DC, USA; **14**. Hospital Vall d'Hebron, Barcelona, Spain; **15**. Merus NV, Utrecht, Netherlands

[1] *Schram et al., ASCO 2022, efficacy data cutoff April 12, 2022 and safety data cutoff January 12, 2022*

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Schema

Global, Multicenter Zenocutuzumab Development Program

Phase 1/2 global, open-label clinical trial (eNRGy)
+
Early Access Program (EAP)

PDAC
NSCLC
Other solid tumors

Inclusion Criteria

- Locally advanced, unresectable or metastatic solid tumor
- NRG1+ cancer
- Previously treated with or unable to receive standard therapy
- ≥ 18 years-old
- ECOG PS ≤ 2

Treatment Plan

- Zenocutuzumab 750 mg IV Q2W until PD
- Tumor assessment Q8W

Follow-up

Survival follow-up: up to 2 years

Endpoints and Population

Primary endpoint
Overall response rate (ORR) using RECIST v1.1 per investigator

Secondary endpoints
Duration of response, ORR per central review, safety, pharmacokinetics, antidrug antibodies

Primary analysis population
≥ 1 dose of Zeno, opportunity for ≥ 6 months follow-up at cutoff, and met criteria for primary efficacy population

Enrollment and Analysis

Data cutoff date
12-Apr-2022

Enrollment
110 patients
64 sites
17 countries

Primary analysis population
83 patients

27 patients excluded[1]:

- 21 patients < 6 months follow-up[2]
- 2 patients baseline scan > 5 weeks before first dose
- 2 patients with other genetic drivers (*KRAS*)
- 2 patients with prior anti-HER3 inhibitor[2]
- 1 patient with ECOG PS 3

[1] Per protocol/SAP
[2] One patient had 2 reasons for exclusion

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Safety Profile

Zenocutuzumab is well tolerated

Key Takeaways

- Safety profile of 208 patients treated with Zeno at the RP2D[1] in the single agent program
- Low incidence of Grade ≥3 treatment-related AEs
- Low incidence of severe gastrointestinal and skin toxicity, and no clinical cardiotoxicity
- <1% of patients discontinued due to AEs

	AEs Irrespective of Causality (>10%)			Treatment-Related AEs (>10% and all Grade 3-5)		
	ALL GRADES	GRADE 3-4	GRADE 5	ALL GRADES	GRADE 3-4[2]	GRADE 5
Patients with ≥1 AE	**92%**	**36%**	**3%**	**61%**	**5%**	**0.5%**
Diarrhea	32%	2%	-	21%	0.5%	-
Asthenia/fatigue	30%	4%	-	12%	0.5%	-
Nausea	20%	1%	-	10%	0.5%	-
Anemia	19%	3%	-	1%	-	-
Infusion-related reaction[3,4]	15%	1%	0.5%	15%	1%	0.5%[3]
Dyspnea	14%	4%	-	2%	0.5%	-
Vomiting	13%	0.5%	-	4%	-	-
Abdominal pain	12%	1%	-	2%	0.5%	-
Constipation	11%	-	-	2%	-	-
Decreased appetite	10%	0.5%	-	4%	-	-
AST increase	9%	3%	-	2%	0.5%	-
Cough	8%	0.5%	-	1%	0.5%	-
ALT increase	7%	3%	-	1%	0.5%	-
Myalgia	4%	0.5%	-	2%	0.5%	-
Neutropenia	3%	1%	-	2%	0.5%	-
Hypertension	1%	1%	-	0.5%	0.5%	-
Platelet count decrease	1%	0.5%		0.5%	0.5%	-
Hyperuricemia	0.5%	0.5%	-	0.5%	0.5%	-
Lymphadenitis	0.5%	0.5%	-	0.5%	0.5%	-
Hypoxia	0.5%	0.5%	-	0.5%	0.5%	-
Bacteremia	0.5%	0.5%	-	0.5%	0.5%	-

Safety data cut off:12-Jan-2022
1. 101 patients with 750 mg Q3W; 26 patients with QW; 81 patients with 750 mg Q2W

2. No Grade 4 treatment-related AEs reported
3. One Grade 5 hypersensitivity (previously reported; Alsina et al. ASCO, 2017)
4. Composite term covering preferred terms considered by the investigator to be IRRs occurring within 24 hours of infusion start

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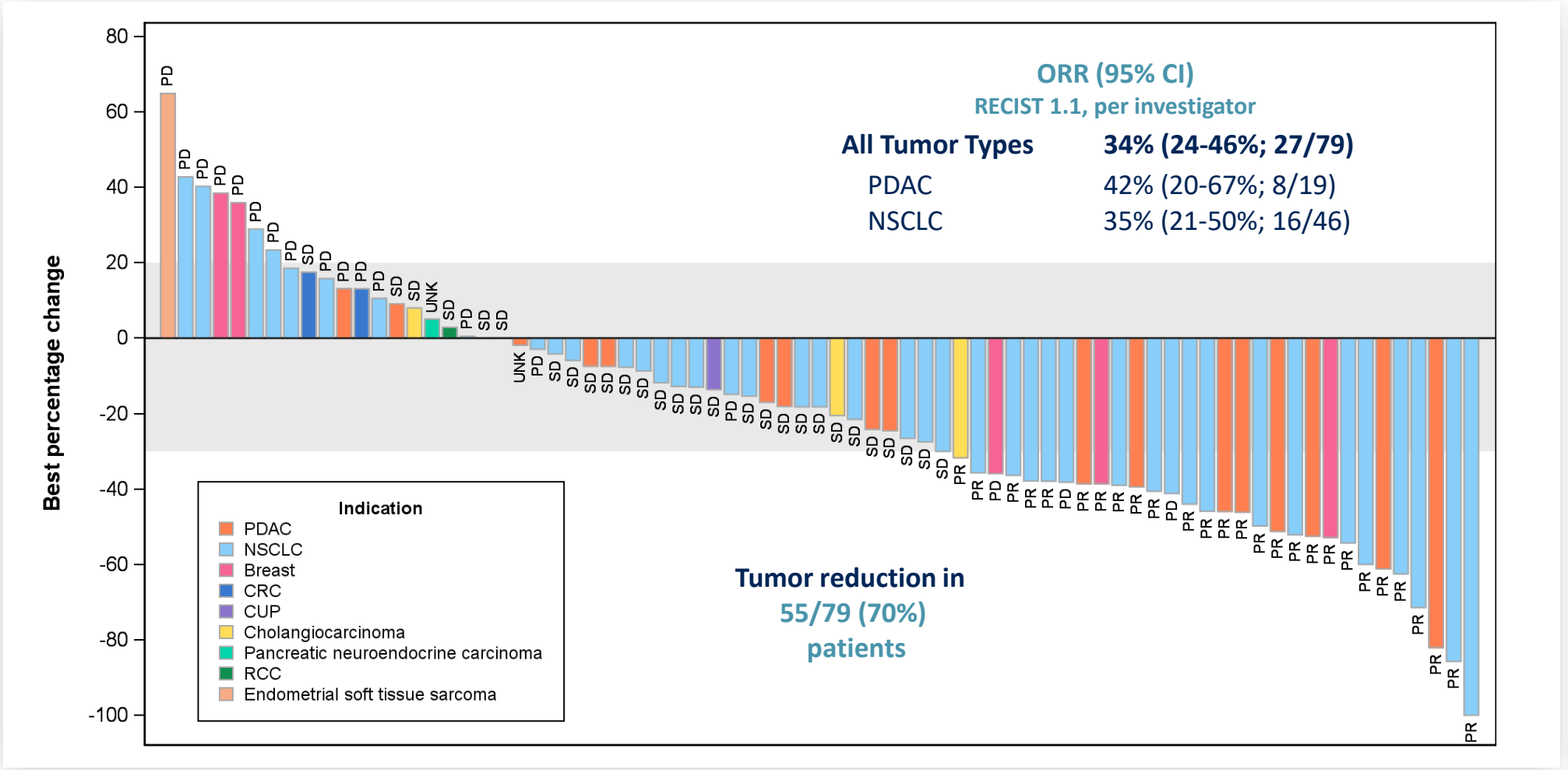
Zenocutuzumab Activity in NRG1+ Cancer

Best Percent Change in Target Lesions from Baseline



Note: The waterfall plot shows data for 75 of 79 patients. Change in tumor size could not be measured for 4 of the 79 patients, 3 due to absence of post baseline assessment (early progression) and one due to incomplete assessment.

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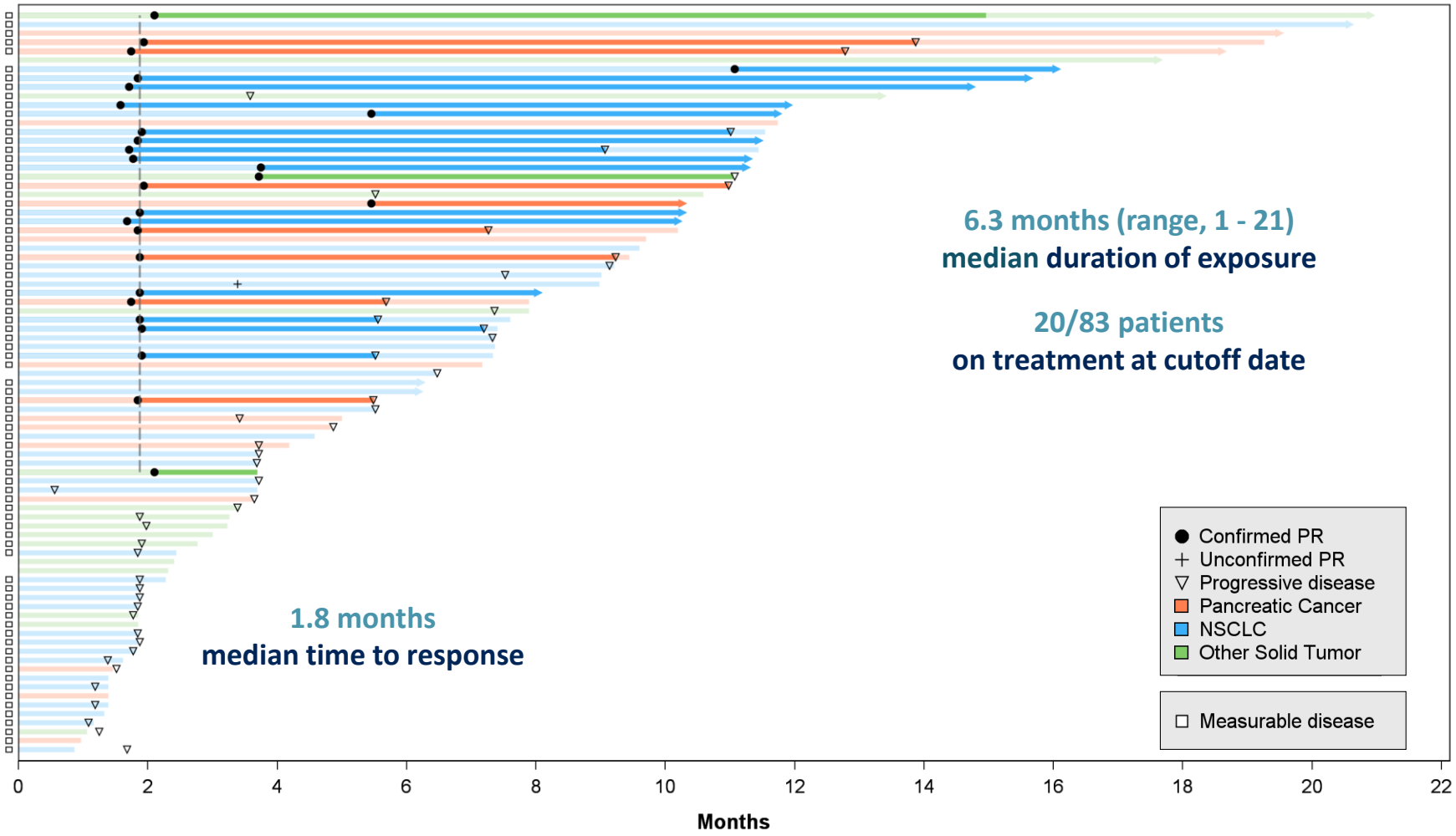
Zenocutuzumab Activity in NRG1+ Cancer

Time to Response and Time on Therapy



Arrows indicate treatment is ongoing at the cutoff date

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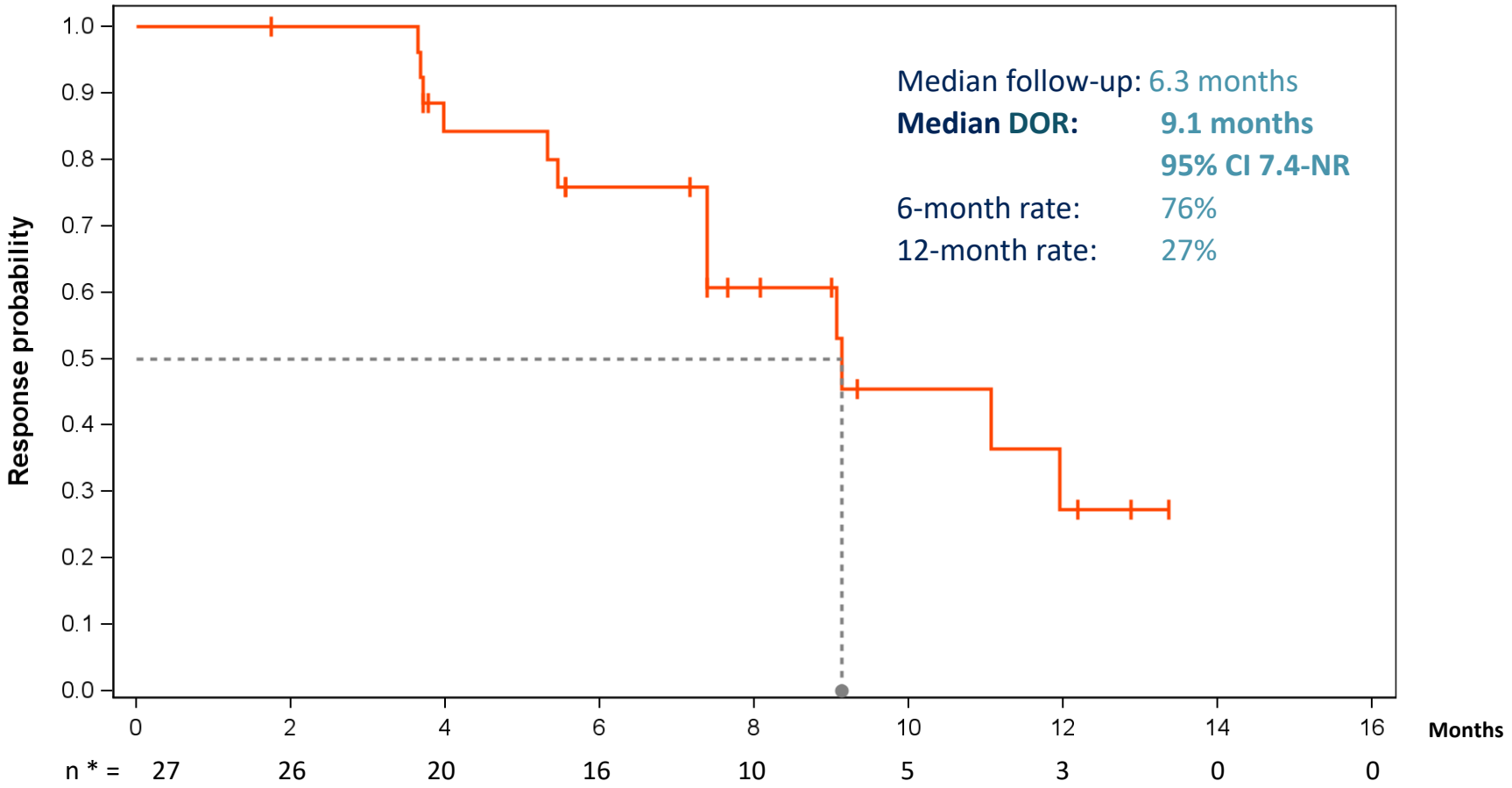
Zenocutuzumab Activity in NRG1+ Cancer

Duration of Response



Note: 20/83 patients were on treatment at cutoff date

* Numbers corrected from the ASCO presentation to reflect correct number of patients at months 2, 6, 8, 10, 14 and 16.

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Zenocutuzumab Conclusions

- **Durable responses in previously treated advanced NRG1+ cancer**
  - ORR 34% (95% CI: 24-46%; n=79)
  - Median DOR 9.1 months (95% CI: 7.4-NR)
  - Antitumor activity across multiple tumor types
- **Extremely well tolerated safety profile**
  - Most adverse events were low grade
  - Very low rate of discontinuations due to toxicity
- **Offers potential new standard of care for patients with NRG1+ cancer**
  - Currently no approved targeted therapy for NRG1+ cancer
  - Great unmet medical need

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Zeno: Continued Progress in NRG1+ Cancer and Beyond

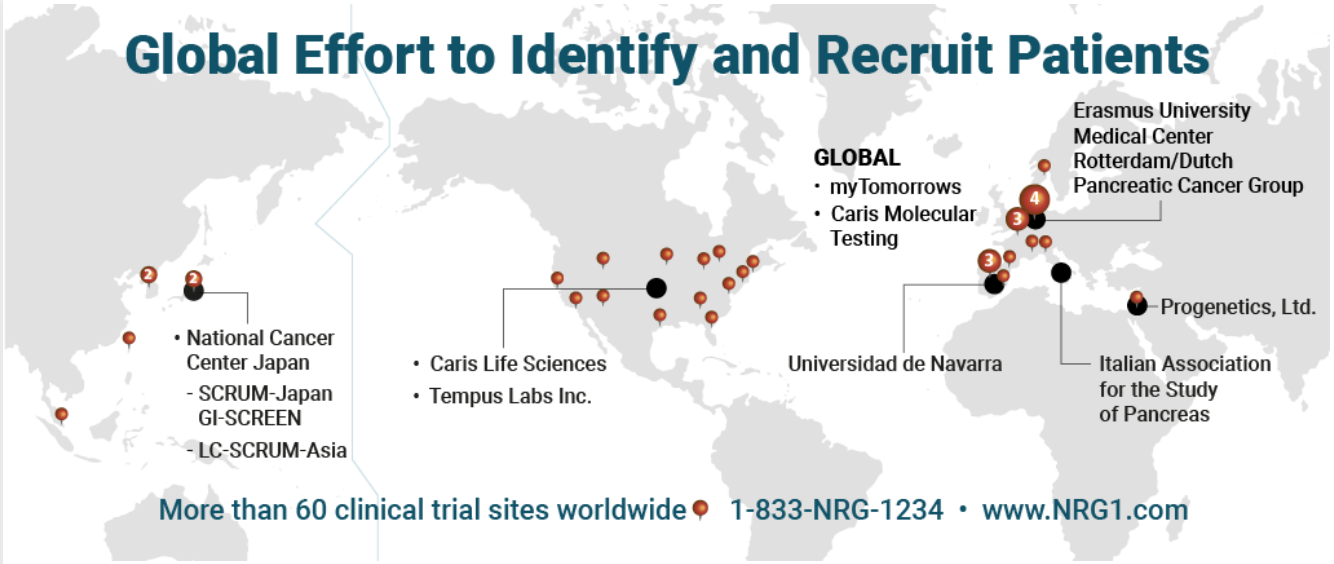
Registration-Directed Clinical Program

- More than 110 patients enrolled as of April 2022
- Obtained alignment with the FDA on registration approach for potential tumor agnostic indication
- Enrollment and follow up completed on an initial cohort of patients we believe may constitute a registrational data set. Enrollment continues, to gather further safety and efficacy data

Broad Zeno Clinical Development Program

- NRG1+ cancer
  - Patient identification and enrollment in the eNRGy trial and early access program (EAP) continue
  - In NSCLC, combination of Zeno with afatinib
- Beyond NRG1+ cancer
  - Castration resistant prostate cancer
- Additional clinical studies in development

Global Effort to Identify and Recruit Patients



eNRGy Clinical Trial

Early Access Program

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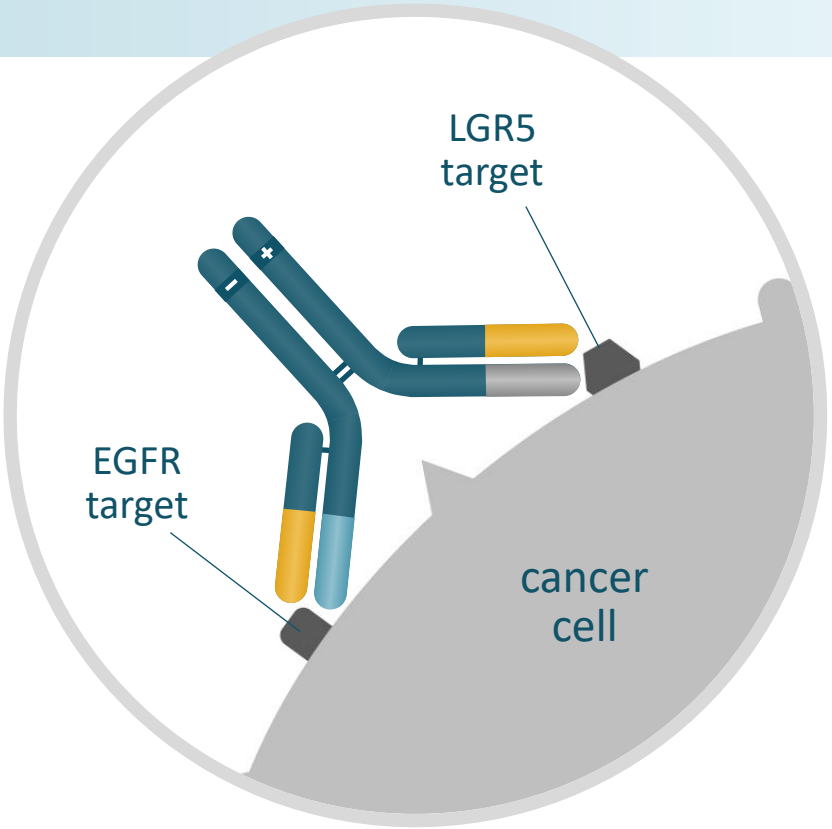
Potential first in class LGR5xEGFR Biclonics® designed to potently block dysregulated signaling and growth in solid tumors

MCLA-158

Petosemtamab or "Peto"
LGR5 x EGFR bispecific

- Binds to EGFR and LGR5, a cancer-stem cell antigen
- Blocks growth in WNT-dysregulated tumor models including Ras^{mut}
- Modifications to enhance ADCC
- Phase 1 trial ongoing; clinical update planned for 1H23
- Early evidence of clinical activity in advanced Head & Neck Squamous Cell Carcinoma (HNSCC) reported at AACR-NCI-EORTC 2021*



* Source: Hollebecque https://merus.nl/wp-content/uploads/2021/10/P185_MCLA-158-HNSCC_virtual-poster_10Sep21_2.pdf

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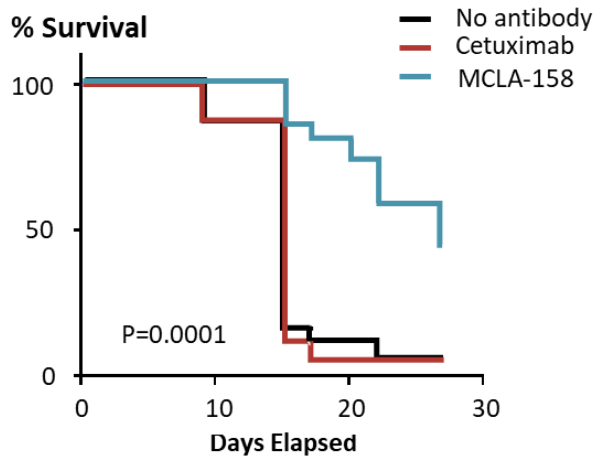
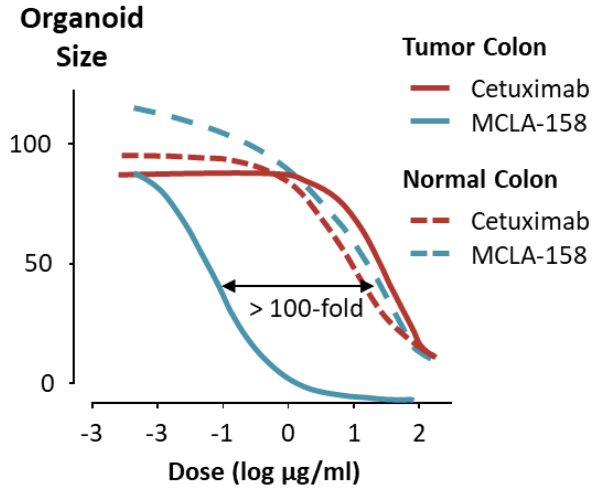
Peto — Novel Target and Innovative MoA

*Superior Growth Inhibition and Selectivity of Tumor Versus Healthy Tissue**

Superior **ACTIVITY** compared to Cetuximab In CRC models

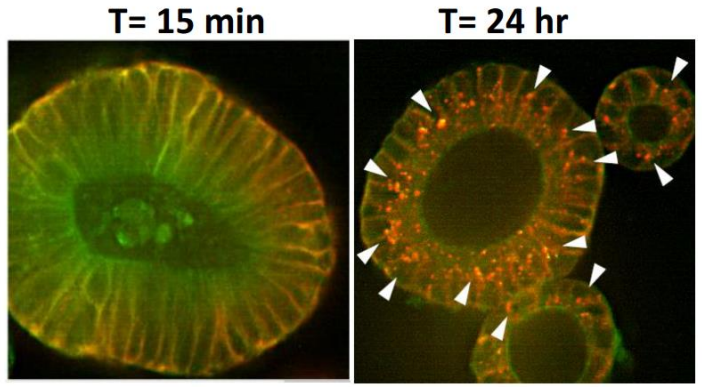


Superior **SELECTIVITY** for tumor-derived organoids



- *Activity observed in xenograft models resistant to treatment with Cetuximab*
- *MCLA-158 discriminated between organoids from tumor and healthy tissue*

*Induces EGFR internalization and degradation***



P18T colorectal cancer organoids

- *After 24h exposure, MCLA-158 (red) is localized intracellularly and overall EGFR expression (green) is strongly reduced*

*Source: Rob C. Roovers (ASCO 2017 Poster Presentation) https://merus.nl/app/uploads/2019/02/MCLA-158-poster-AACR2017.pdf

** Source: Guillem Argilés (ASCO GI 2021 Poster Presentation) https://merus.nl/wp-content/uploads/2021/01/MCLA-158_ASCO_GI_final.pdf

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Updated data from AACR-NCI-EORTC 2021

Phase 1 Cohort Expansion in Head and Neck Squamous Cell Carcinoma

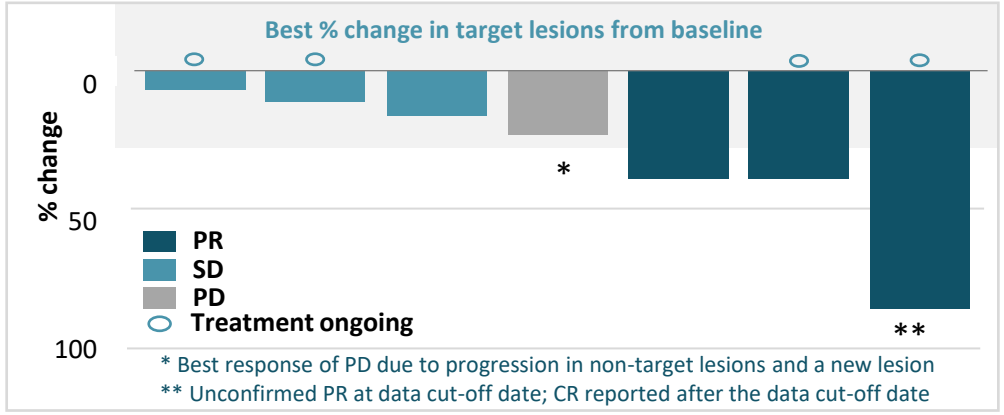
Peto Enrollment and Interim Analysis

- Cohort expansion at RP2D
  - HNSCC (interim data presented at ENA)
  - Gastric/Esophageal (not yet presented)
- Data cut-off date: 09-Aug-2021
- Enrollment: 10 patients, 7 evaluable for efficacy
  - Three patients recently enrolled excluded from interim analysis (first dose <8 weeks from data cut-off date)

HNSCC Patient Characteristics (N=10)	
Age (years), median (range)	65 (50-77)
Male / female	9 (90%) / 1 (10%)
ECOG PS 0 / 1	4 (40%) / 6 (60%)
Squamous cell carcinoma histology	10 (100%)
EGFR IHC score 2+ / 3+ (n=5)	1 (20%) / 4 (80%)
N lines prior therapy, median (range)	2 (1-3)
• Platinum-based chemotherapy	10 (100%)
• PD-(L)1 inhibitor	9 (90%)
• Cetuximab	0%

Early Clinical Activity in HNSCC

- Three of 7 patients achieved partial response
- All 7 patients experienced tumor shrinkage



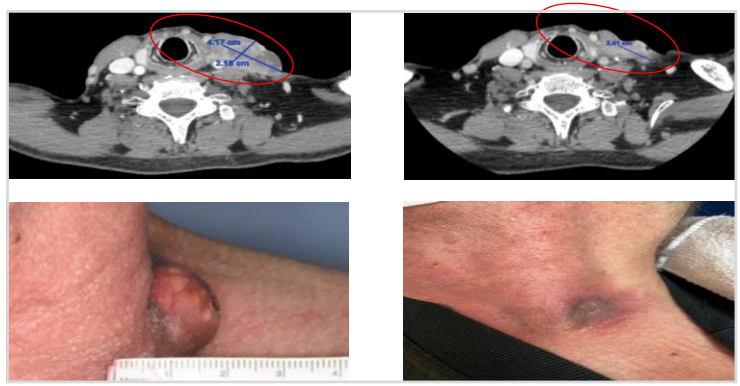
Clinical response in 67-year-old male patient

Lesion: larynx

MCLA-158 cycles: 6+

Best response: PRc (-41%)

Prior treatment: platinum +paclitaxel + durvalumab



Investigator review, efficacy data cutoff Aug. 9, 2021, refer to Hollebecque https://merus.nl/wp-content/uploads/2021/10/P185_MCLA-158-HNSCC_virtual-poster_10Sep21_2.pdf

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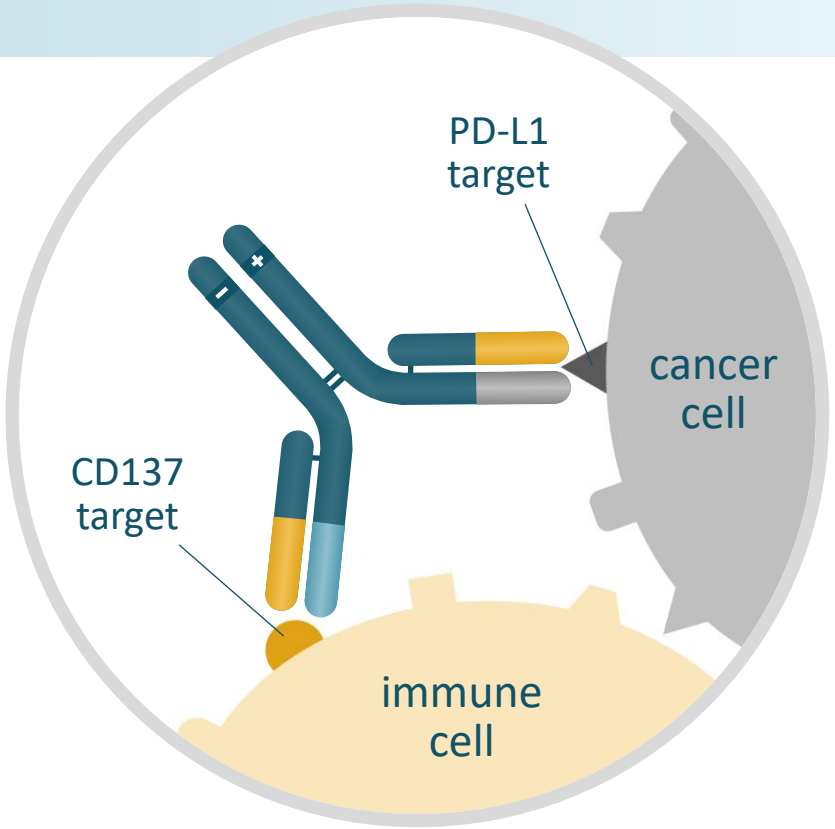
Designed to recruit and activate tumor infiltrating T-cells

MCLA-145

PD-L1 x CD137 bispecific

- Binds to PD-L1 on tumor cells and CD137 (4-1BB), a potent activator of tumor infiltrating T-cells, on activated T-cells
- Targets PD-L1 positive cells in the tumor and blocks the PD-1/PD-L1 inhibitory signal
- Potential in a variety of solid tumors
- Global phase 1 trial ongoing
- Clinical update presented at ESMO Immuno-Oncology Congress 2021



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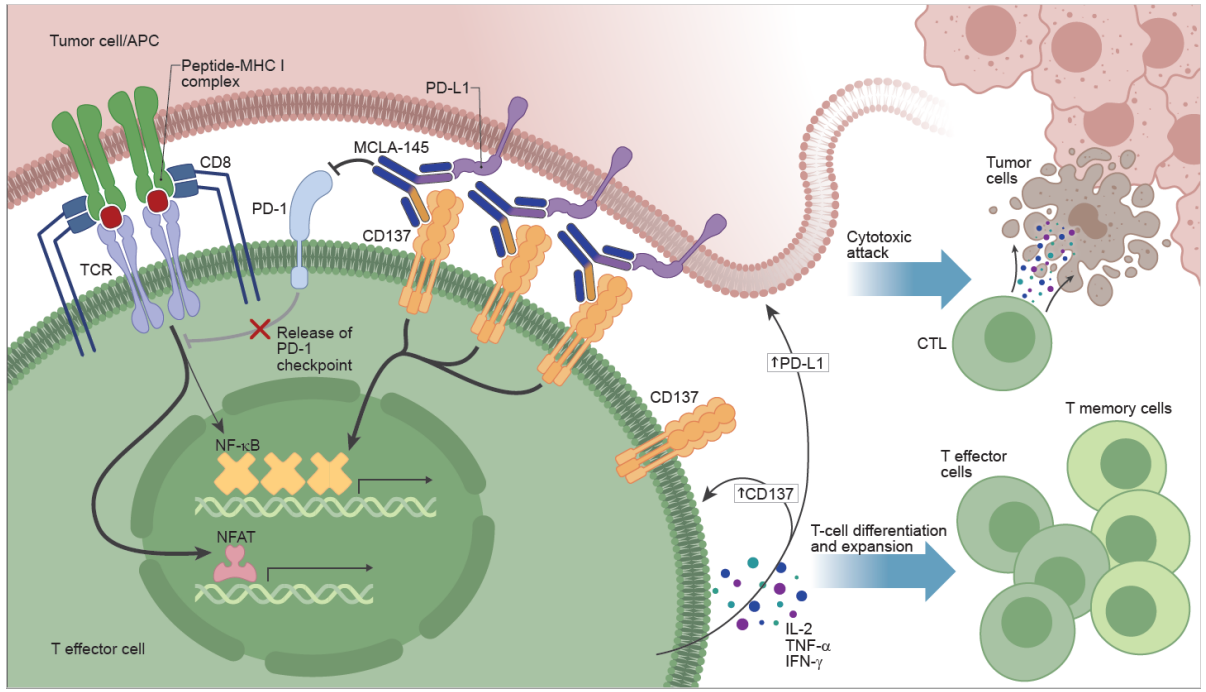
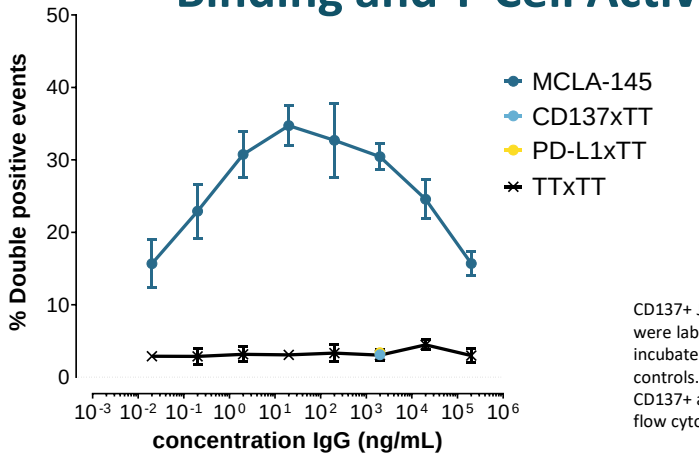
MCLA-145 — Targets PD-L1 Positive Tumor Cells

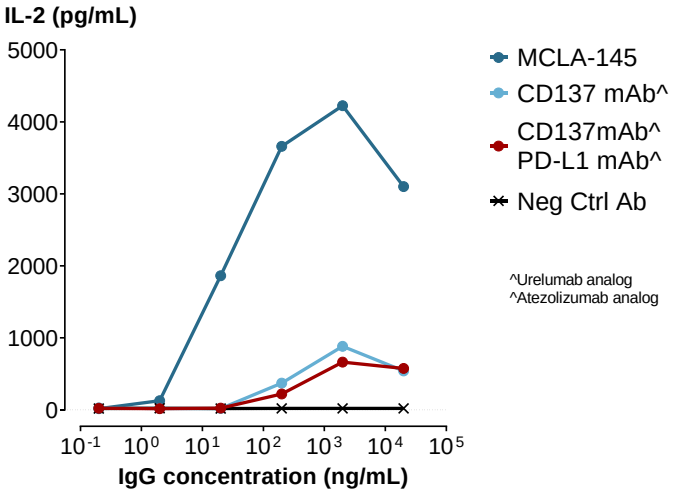
Mechanism of Action



Binding and T-Cell Activation



CD137+ Jurkat cells and PD-L1+ CHO cells were labeled with different dyes and co-incubated in the presence of MCLA-145 or controls. Complex formation between CD137+ and PD-L1+ cells was analyzed by flow cytometry



^Urelumab analog
^Atezolizumab analog

Source: Geuijen https://merus.nl/app/uploads/2019/04/IC171-19E-AACR19-Mayes-MCLA-145-MoA-Poster_MT06_for-approval-032019.pdf
Source: Prenen https://merus.nl/wp-content/uploads/2021/12/MCLA-145-Poster-ESMO-IO_12.3.21_Final.pdf

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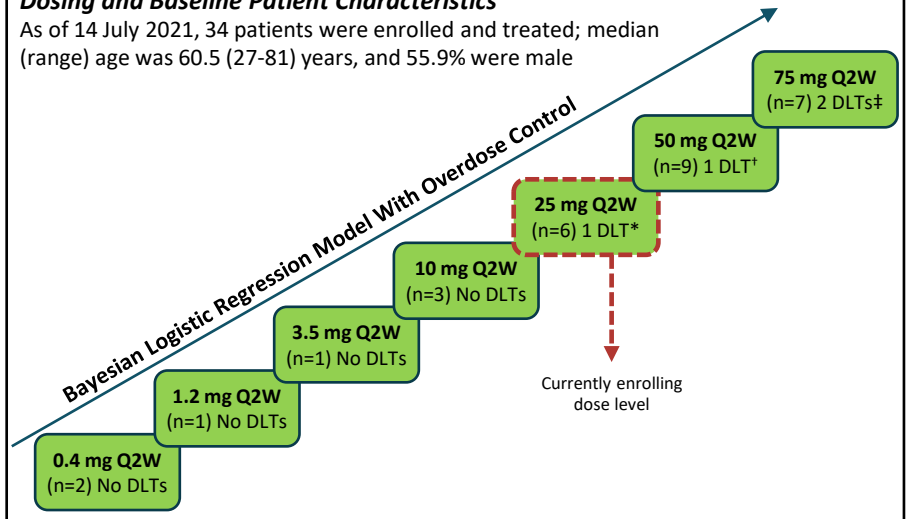
Data from ESMO-IO 2021

Phase 1 Clinical Trial

Dosing and Baseline Patient Characteristics

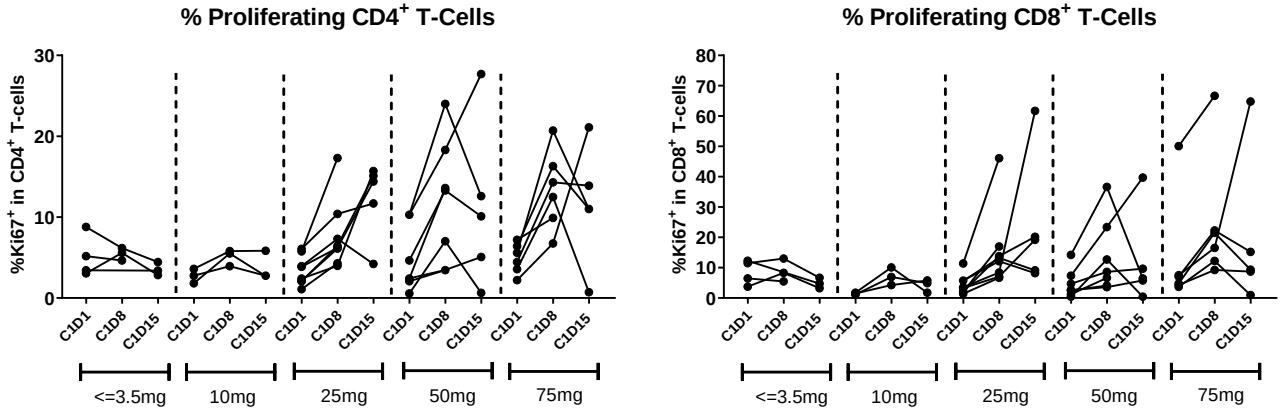
As of 14 July 2021, 34 patients were enrolled and treated; median (range) age was 60.5 (27-81) years, and 55.9% were male



ALT, alanine aminotransferase; AST, aspartate aminotransferase; DLT, dose-limiting toxicity (DLT period is defined as 28 days from the first infusion); Q2W, every 14 days.
Intermediate dose levels from 25 to 75 mg have been explored in 5 additional patients not included in the figure above.
*Febrile neutropenia. †Myositis. ‡ALT/AST increased, febrile neutropenia

Patient Characteristics (N=34)	
Age (years), median (range)	60.5 (27-81)
Male / female	19 (56%) / 15 (44%)
ECOG PS 0 / 1	16 (47%) / 18 (53%)
PDL-1 expression on tumor cells	
• Unknown/unevaluable	16 (47%)
• 0% / ≥1%	11 (32%) / 7 (21%)
PDL-1 expression on tumor assoc. immune cells	
• Unknown/unevaluable	16 (47%)
• 0% / ≥1%	4 (12%) / 14 (41%)

Peripheral Blood T cell activation



C, cycle; D, day.
* 25 mg data includes patients from the intermediate dosing cohort.
Horizontal lines=Geometric mean per dose level.
Limited data for IFNγ fold change due to levels below detection limit for 13/33 patients.

Conclusions

- Thirty-four patients have been treated with MCLA-145 at dose levels from 0.4 – 75 mg q2W
- AEs are consistent with the MOA and can be managed with drug interruption and/or steroids in some patients
- Preliminary evidence of antitumor activity has been observed at doses ≥ 25 mg
- Peripheral blood T cell activation has been observed
- Further evaluation of optimal dose in PD-L1+ tumors is planned. Full blockade of PDL1 may be required

https://merus.nl/wp-content/uploads/2021/12/MCLA-145-Poster-ESMO-IO_12.3.21_Final.pdf

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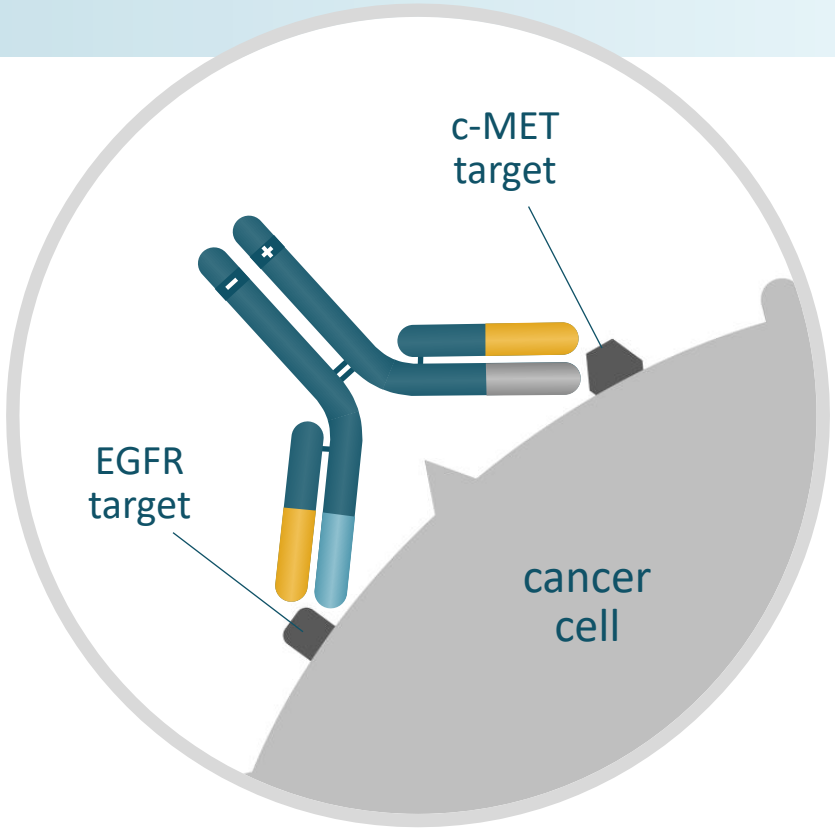
Designed to target lung cancer and other solid tumors expressing EGFR and c-MET

MCLA-129

c-MET x EGFR Bispecific



- Targets both EGFR and c-MET on cancer cells
- Modifications to enhance ADCC
- Significant opportunity in lung cancer and other solid tumors
- Phase 1/2 trial ongoing; clinical update planned for EORTC/NCI/AACR 2022
- Expansion cohorts, including in combination with osimertinimab, a third generation EGFR-TKI, to begin in 2H22

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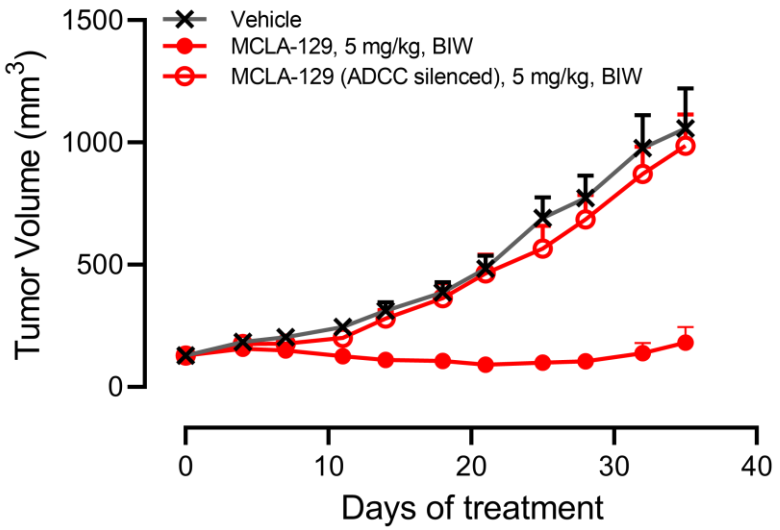
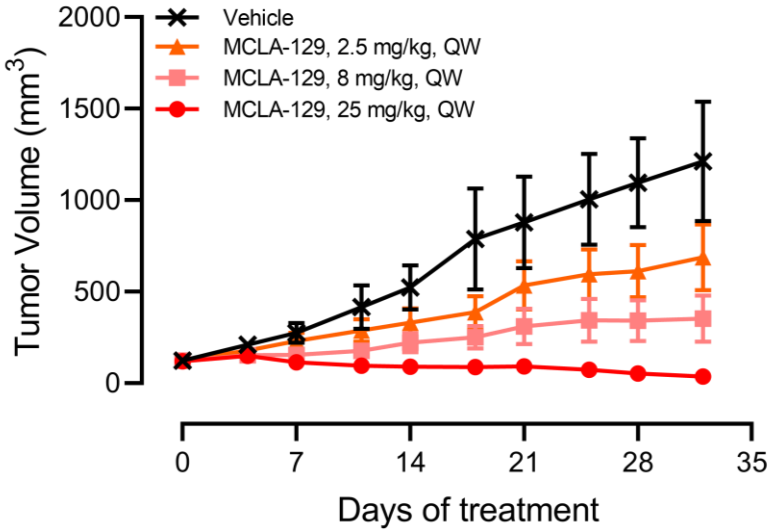
MCLA-129 — Targets both c-MET and EGFR

*Demonstrated Tumor Growth Inhibition and Potent ADCC in Preclinical Studies**

Inhibition of NSCLC EGFR^{del19} tumor growth



Inhibition of NSCLC EGFR^{exon20} tumor growth



Potent Bispecific Antibody

- Blocks EGF and HGF binding to EGFR and c-MET
- Fc enhanced to promote ADCC and ADCP
- Potently inhibit NSCLC tumor growth as monotherapy and in combination with EGFR TKI in preclinical models
- Overcome HGF-mediated EGFR-TKI resistance in preclinical models

* Source: Geuijen (AACR 2019 Poster presentation) https://merus.nl/wp-content/uploads/2019/10/AACR-NCI-EORTC-Poster-LBC07-MCLA-129.pdf
* Source: de Gorter (AACR 2021 Poster presentation) https://merus.nl/wp-content/uploads/2021/04/Merus_poster_MCLA-129_AACR2021.pdf
*Source: de Gorter (AACR 2022 Poster presentation) https://merus.nl/wp-content/uploads/2022/04/Merus_poster_MCLA-129_AACR2022-FINAL.pdf

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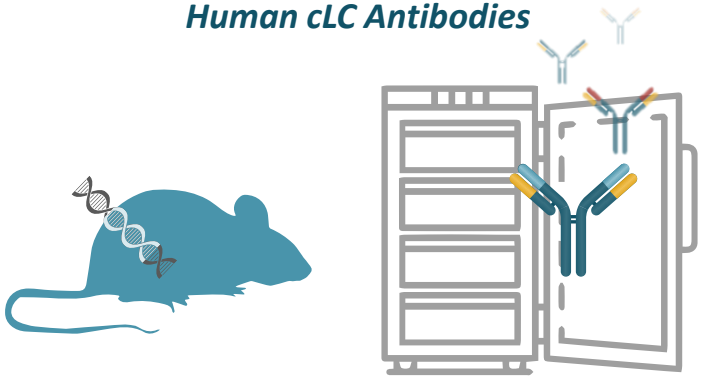
Our Platform – Unique Capabilities in Multispecific Antibodies

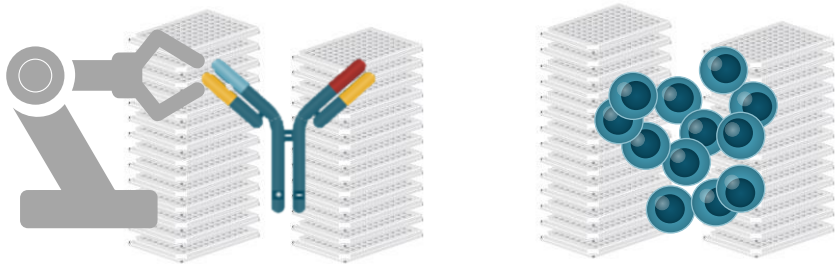
Generate
Human cLC Antibodies



Patented Mouse Technology
"Merus Mouse" (MeMo®) to generate diverse, high quality common light chain (cLC) antibody panels

Established Inventory
Diverse panels of cLC antibodies against numerous targets

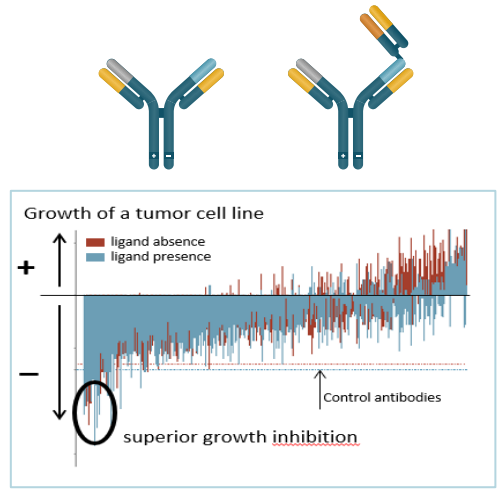
Evaluate
Thousands of Multispecific Abs



Multiclonics® Libraries
Robotics generate thousands of Multiclonics® by combining cLC antibody panels and our patented "DEKK" IgG heterodimerization technology

Unbiased Screening
In-format, unbiased functional screening in relevant molecular and cellular assays

Identify
Best Candidates



Develop unique, best candidates from thousands of different Biclonics® and Triclonics® with potential to achieve meaningful clinical activity in patients

Merus

Merus Collaborations

Strategic relationships expand pipeline potential and clinical reach

Global collaboration of up to 10 Biclonics® programs

$200M at signing and research funding, option to co-fund development of two programs in return for 50/50 US profit split

Collaboration to develop up to 3 T-cell engaging Biclonics® programs

$60M at signing and research funding, milestones and royalties

MCLA-129, EGFR x c-MET collaboration

Betta has rights for China; Merus retains global rights ex-China, phase 1 trials ongoing

Biclonics® Licensing Agreement for a Biclonics® CD3 bispecific antibody.

Phase 1 trial in Japan for ONO-4685, a PD-1 x CD3 bispecific antibody

Patient identification agreements

More than a dozen agreements with top-tier diagnostic companies and industry and academic collaborators

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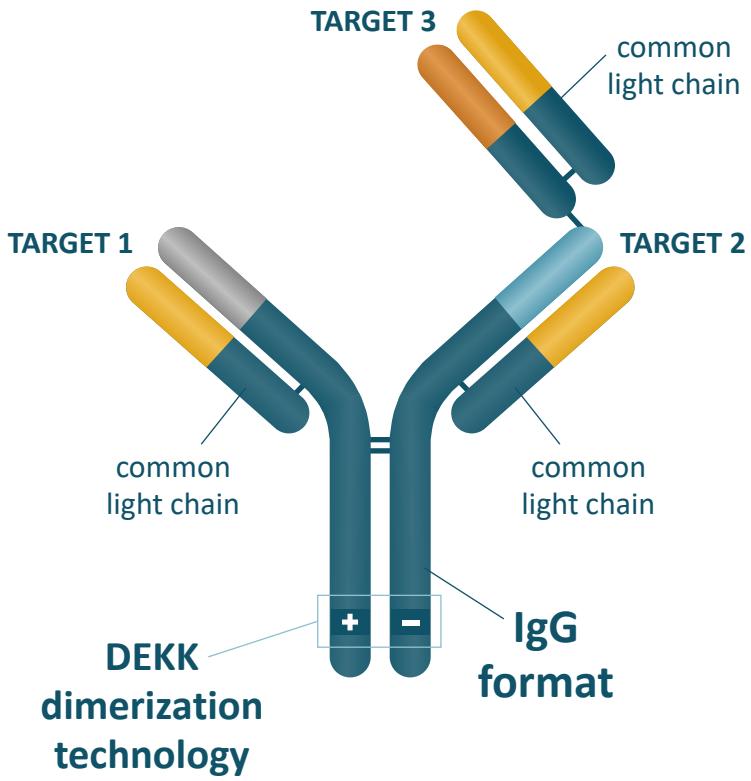
Our Research — Triclonics® and Beyond



Triclonics®

Triclonics® Opportunity

- High throughput production, purification and screening in the trispecific format
- Stable format with predictable behavior that can be produced as if it were a normal monoclonal antibody
- Allows for 3 specificities without the need to engineer each individual Fab
- Leverages Merus' extensive library of established antibody panels that bind tumor antigens and engage and modulate the immune system

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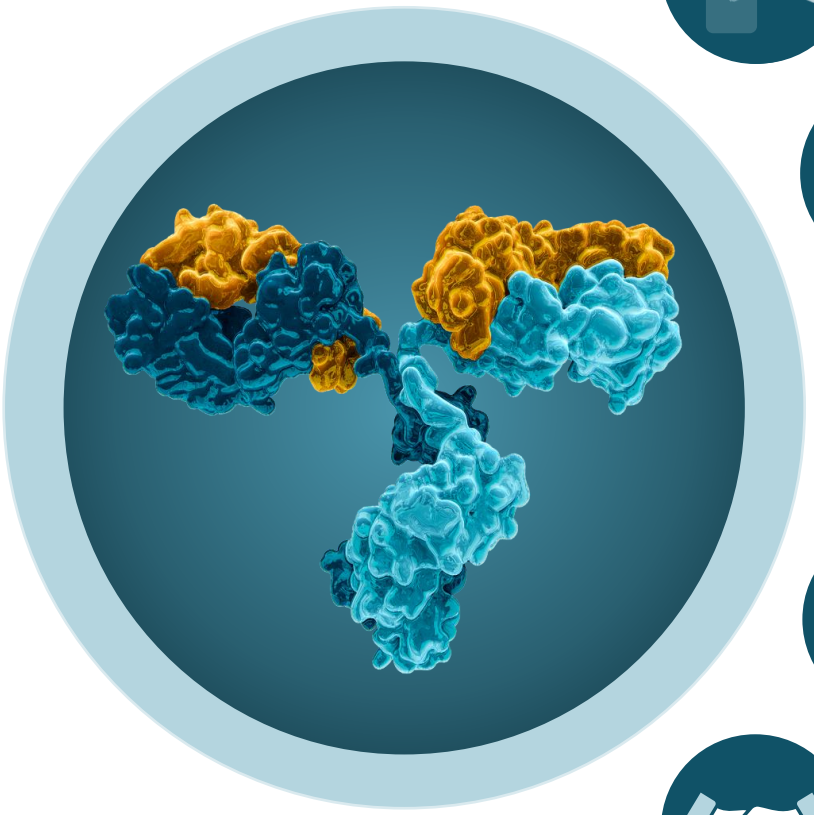
Merus Overview



Oncology-focused Company Developing Multispecific Antibody Therapies

Bispecific and trispecific cancer therapeutic candidates based on the human IgG format

Established Clinical Pipeline

Four clinical stage assets with proof of concept data on Zeno in NRG1+ cancer[1], and early encouraging data on Peto in Head and Neck cancer[2]

Leading Multispecific Antibody (Multiclonics®) Platforms

Common light chain format permits broad high throughput evaluation of Biclonics® and Triclonics®, to develop clinical stage assets with meaningful clinical responses in patients

Near Term Trial Updates and Strong Cash Position Beyond 2024*

Upcoming clinical milestones and program updates planned over the next 12-18 months: Zeno registration-directed program, MCLA-129 initial clinical data, and Peto clinical update

Strategic Collaborations to Unlock Platform Value

Multiple strategic collaborations and license agreements

[1] *Schram et al., ASCO 2022, efficacy data cutoff April 12, 2022 and safety data cutoff January 12, 2022*

[2] *Hollebecque et al., AACR-NCI-EORTC 2021, data cutoff August 9, 2021*

*See August 8, 2022 10-Q noting our belief that our cash, cash equivalents and marketable securities, will fund our operations beyond 2024.

Merus

Merus

closing in on cancer