Merus

Pioneering Bispecific Antibodies

November 2019

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® platform 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. 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 bispecific antibody candidates; potential delays in regulatory approval, 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® 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 immunotherapies; 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 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; and we may lose our foreign private issuer status and incur significant expenses as a result.

These and other important factors discussed under the caption “Risk Factors” in our Annual Report on Form 20-F filed with the Securities and Exchange Commission, or SEC, on April 3, 2019, 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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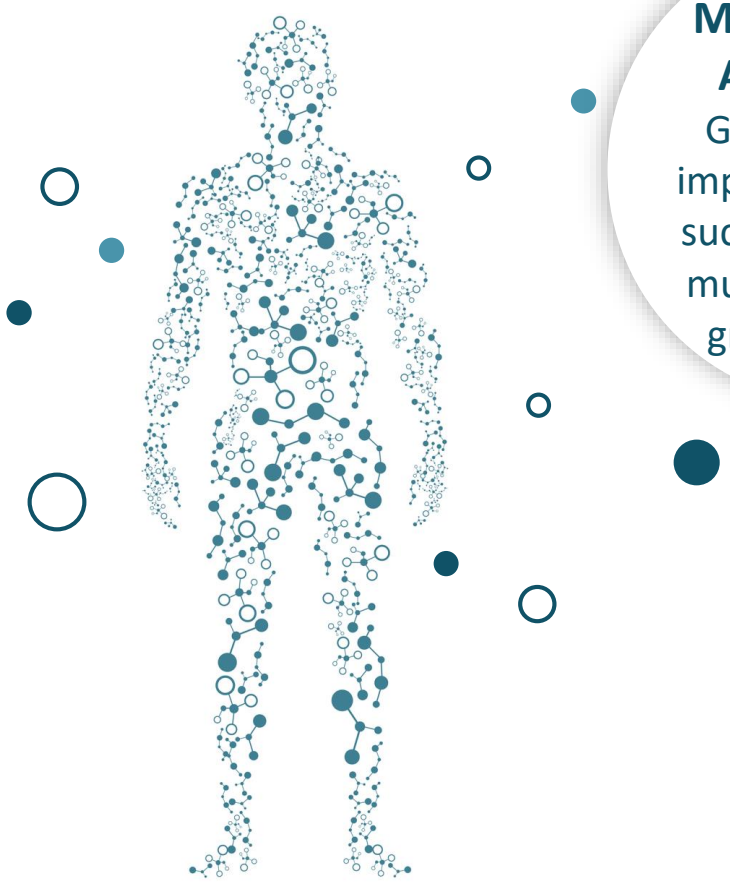
The next wave of antibody-based therapeutics in cancer treatment



MONOCLONAL ANTIBODIES
Game-changing impact, but limited success combining multiple mAbs for greater efficacy

BISPECIFIC ANTIBODIES
Offering novel modes of action and new biology

KILL Cancer Cells
with bispecific antibodies with novel modes of action

BLOCK Immune Suppression
in the tumor to strengthen immune activation

ENGAGE T Cells
to kill cancer cells and establish long-lasting anti-tumor responses

HIGH POTENTIAL
for Immunotherapy and Precision Medicine

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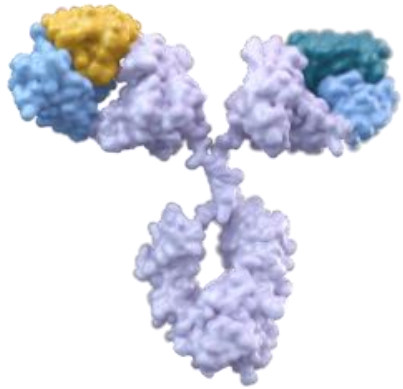
Merus: pioneering bispecific antibodies since 2006

4 clinical-stage bispecific antibodies in oncology



Designed by nature. Selected by Merus.

Sophisticated proprietary bispecific and trispecific technology platforms

Multiple clinical milestones anticipated in the next 12 months

Discovery of novel modes of action based on target combinations and functional screening

Fully integrated discovery-to-manufacturing bi-/tri-specific technology platforms

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Merus clinical pipeline, near term milestones and program status

PROGRAM	TARGETS	INDICATION / DRUG COMBINATION	PRE-IND	PHASE 1	PHASE 2	MILESTONE/STATUS
MCLA-128	HER3 x HER2	NRG1 Solid tumors (monotherapy)				Next update expected around end of 2020
		Metastatic Breast (combination in 2 cohorts)				Phase 2 Results in 2020
MCLA-117	CLEC12A x CD3	Acute Myeloid Leukemia (AML)				Initial Data 1H 2020
MCLA-158	Lgr5 x EGFR	Solid tumors				Trial Update YE 2019
MCLA-145	CD137 x PD-L1	Solid tumors Incyte (ex- U.S.)				Dose Escalation Ongoing
MCLA-129	EGFR x c-MET	Solid tumors BETTA PHARMACEUTICALS (China)				IND Enabling Studies Ongoing
ONO-4685	PD-1 x CD3	Autoimmune disease ONO				Phase 1 Trial Ongoing
....	Undisclosed	Autoimmune disease ONO				

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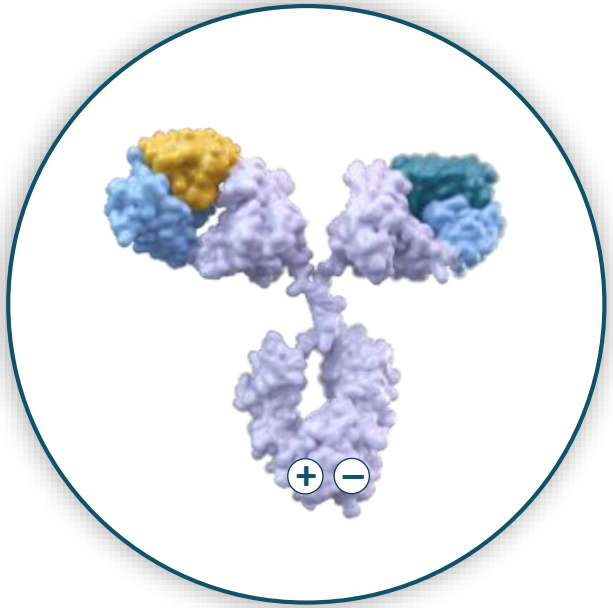
Biclonics® — designed to look and perform like natural human antibodies

BICLONICS® - leveraging the attractive characteristics of natural antibodies

Merus' Bispecific Antibodies are produced by a single cell

Common Light Chain
for 'unforced', natural pairing with 2 different heavy chains



IgG Format
for efficient manufacturing and predictable *in vivo* behavior

Electrostatic attraction
to efficiently drive formation of Biclonics®

Fc Modifications
for Improved functionality (ADCC or silencing)

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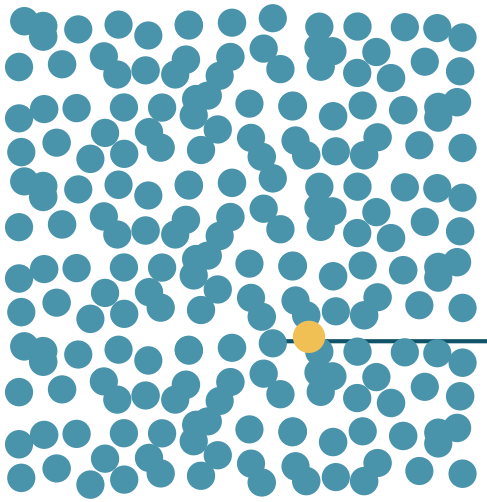
Biclonics® — Selected in functional assays for differentiated activity

HUMAN ANTIBODY GENERATION

MeMo®
Transgenic Mouse

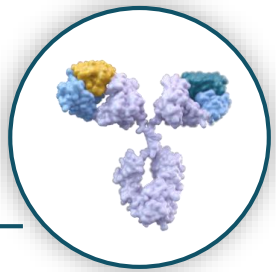
We use a proprietary transgenic mouse to generate panels of high-quality human antibodies

PANEL GENERATION



We can create up to 1,000 Biclonics® against any target pair of choice

FUNCTIONAL SCREENING



We use large-scale functional screening in cell-based assays to identify Biclonics® with novel modes of action

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Clinical Programs
MCLA-128

HER2 x HER3 Biclonics®

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Blocks primary tumor cell growth and escape to HER2/EGFR targeted therapy

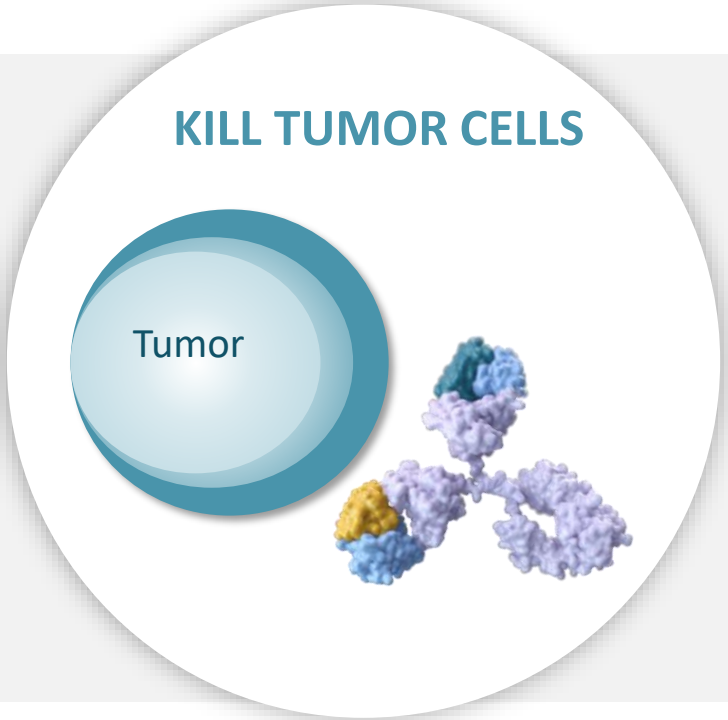
Unique DOCK & BLOCK® mechanism of action potently inhibits neuregulin (NRG)-driven tumor growth

Block HER3

Blocks HER3 signaling even in high neuregulin tumor environments

Enhanced ADCC

Mediates tumor elimination by immune killer cells



Dock HER2

Docks to HER2 expressed on tumor cells to efficiently block HER2:HER3 dimer formation

Combination

Combinations with HER2 targeted therapies possible

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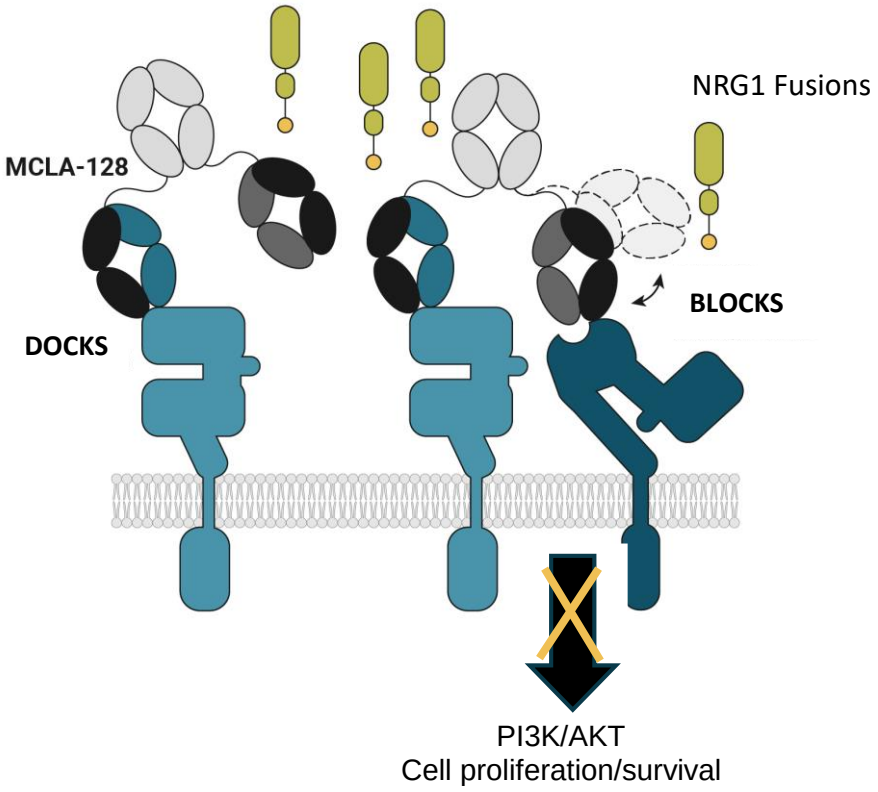
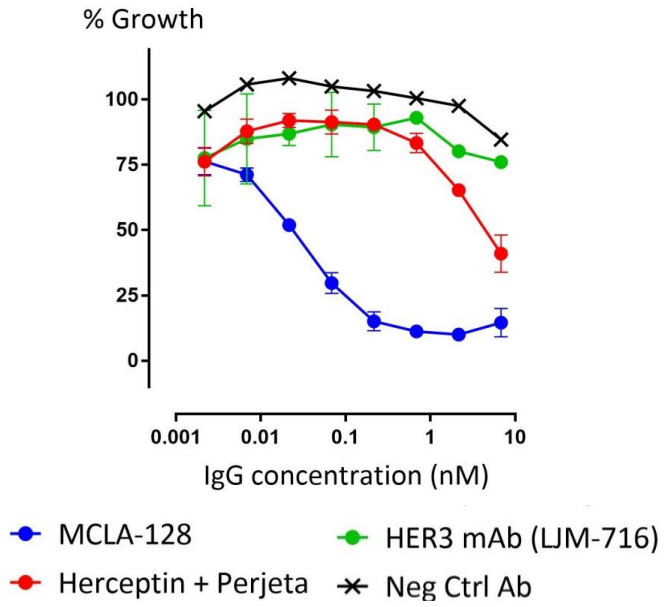
MCLA-128 efficacy demonstrated in preclinical assays, well-tolerated in patients

DOCK ONTO HER2 & BLOCK NRG1 -DRIVEN HER/HER3 DIMER FORMATION



SUPERIOR ACTIVITY SHOWN IN PRECLINICAL MODELS



SAFETY AND TOLERABILITY IN PHASE 1/2 TRIAL

117 PATIENTS EVALUATED*

MCLA-128 Dosing: 750 mg ranging from q1w-q3w

- Single agent well tolerated
- Low risk for immunogenicity

Please Refer to https://merus.nl/publications/ for full data presented. Refer to ASCO poster 2018 and AACR-NCI-EORTC International Conference on Molecular Targets and Cancer Therapeutics 2019

* As of Jan. 2019

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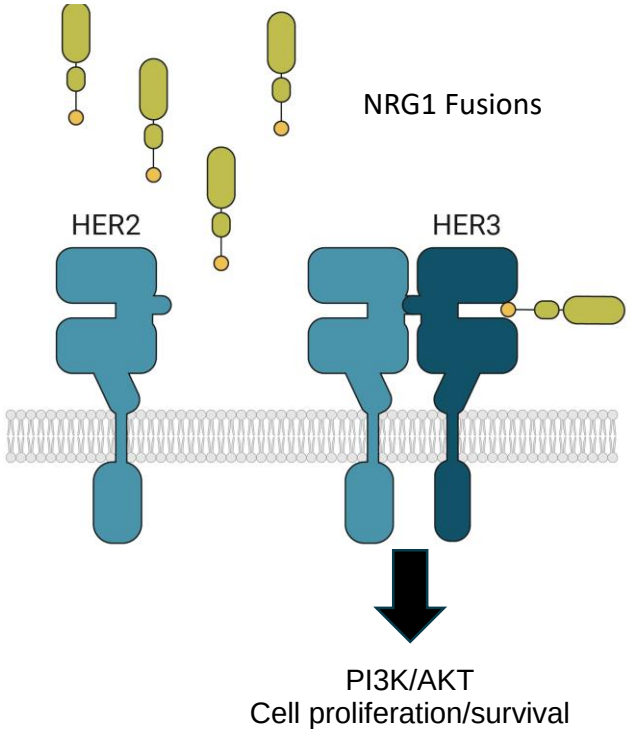
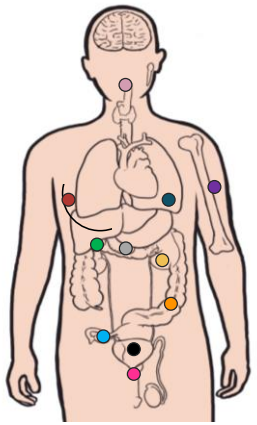
MCLA-128 may offer a novel therapeutic paradigm for *NRG1* Fusion-positive cancers

NRG1 FUSIONS STIMULATE TUMOR CELL GROWTH



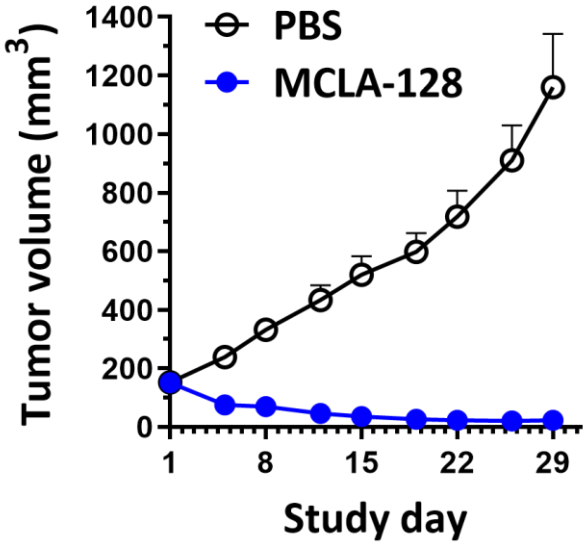
NRG1 FUSIONS ARE FOUND ACROSS MULTIPLE SOLID TUMOR TYPES



- Lung
- Breast
- Kidney
- Gallbladder
- Pancreas
- Prostate
- Ovarian
- Sarcoma
- Bladder
- Colorectal
- Nasopharyngeal

Tumor type	Estimated Incidence (%)
Lung	0.3 – 3.0
Pancreas	0.5 – 1.5
Other	< 1.0

MCLA-128 BLOCKS TUMOR GROWTH IN NRG1 FUSION PDX MODEL



Projections of NRG1 Fusions occurrence (incidence) are based on limited published information

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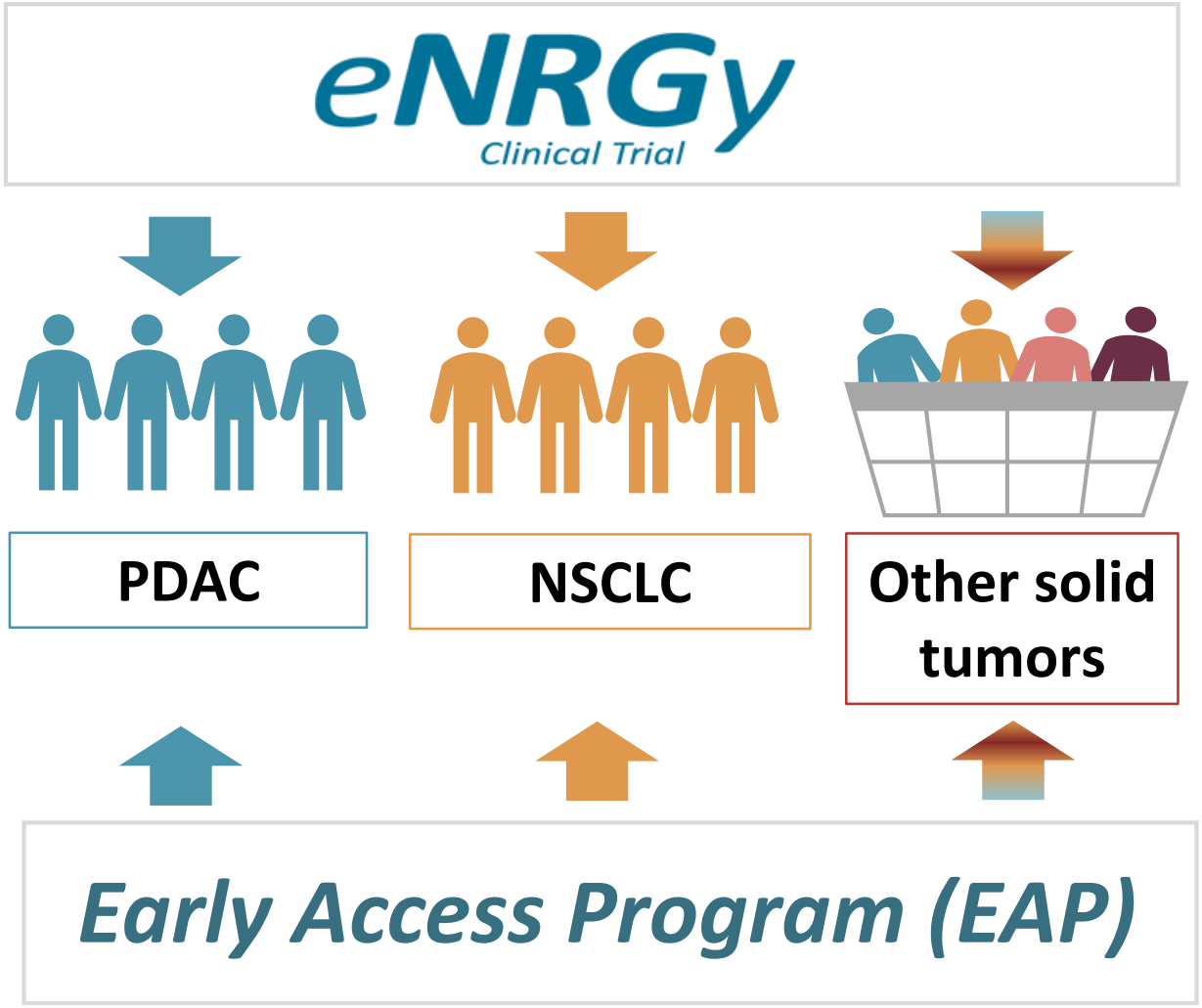
MCLA-128 activity and durability to date with NRG1 fusion^{POS} cancers

As of October 26 2019		Response by RECIST 1.1	Duration (ongoing)
eNRGy Clinical Trial EAP	NSCLC 6	1 PR (- 41%)	~5 mths
		1 SD*	~7 mths
		2 too early to evaluate	n/a
		2 PD	n/a
	PDAC 3	1 PR (-54%)	7+ mths
		1 SD (-25%)	7+ mths
		1 non-evaluable**	n/a

* Patient discontinued the trial due to poor adherence to treatment (unrelated to any AE or lack of efficacy)

** Patient passed away due to complications related to the underlying disease prior to first tumor evaluation.



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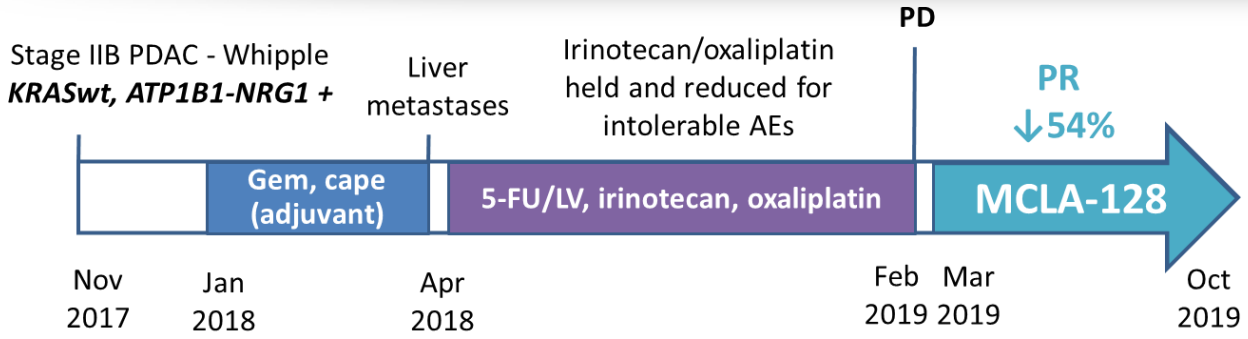
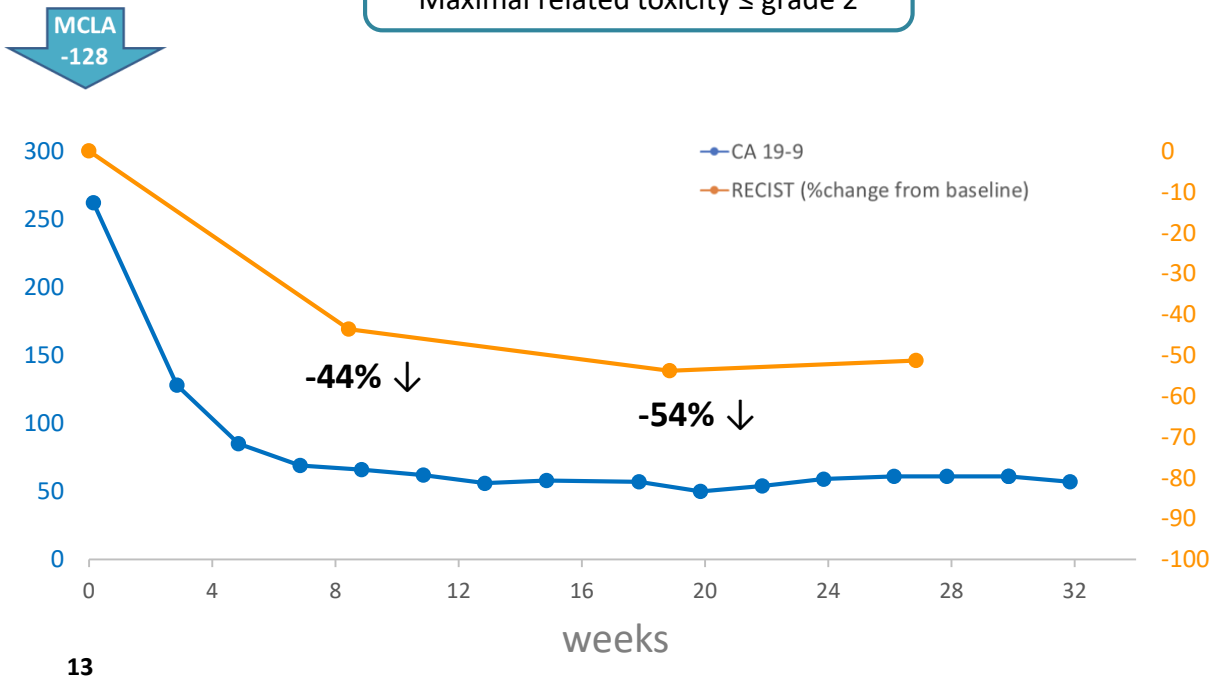
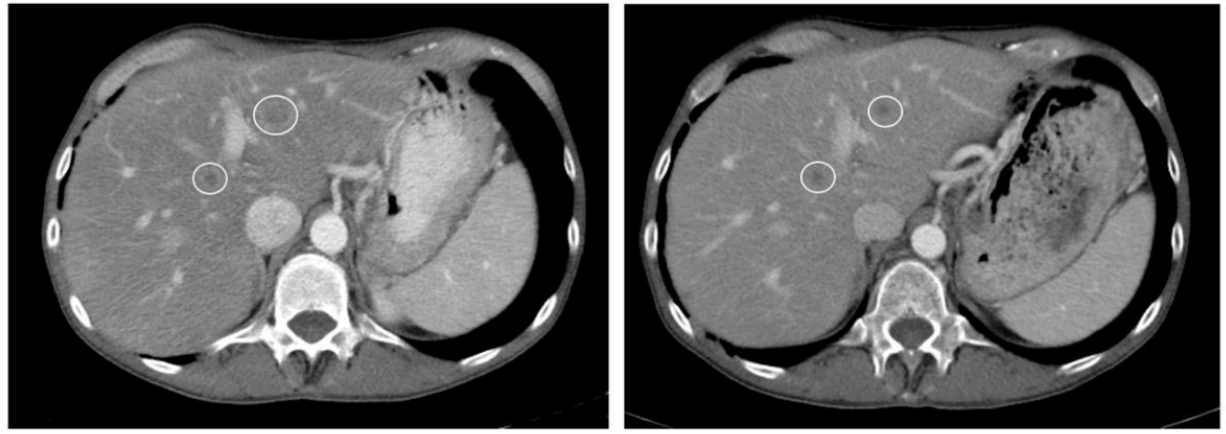
52-year-old male with *ATP1B1-NRG1* pancreatic CA



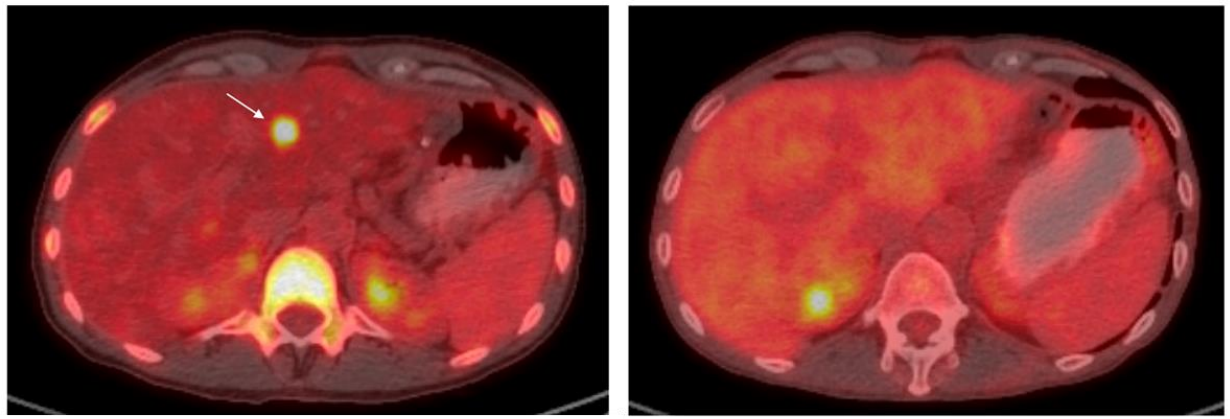
7+ months MCLA-128, 750 mg IV, q2w
Maximal related toxicity ≤ grade 2





Baseline CT

8 week CT



Baseline PET

8 week PET

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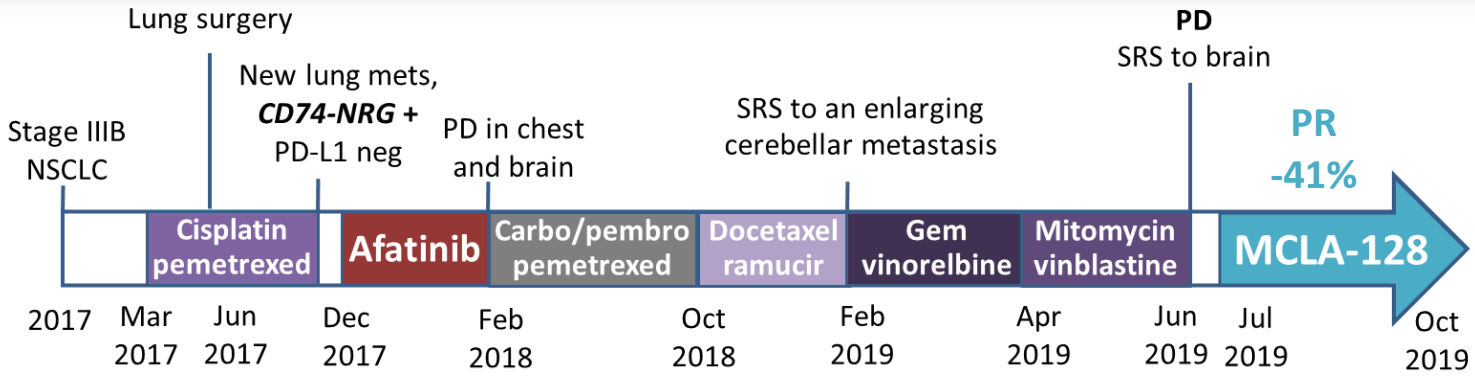
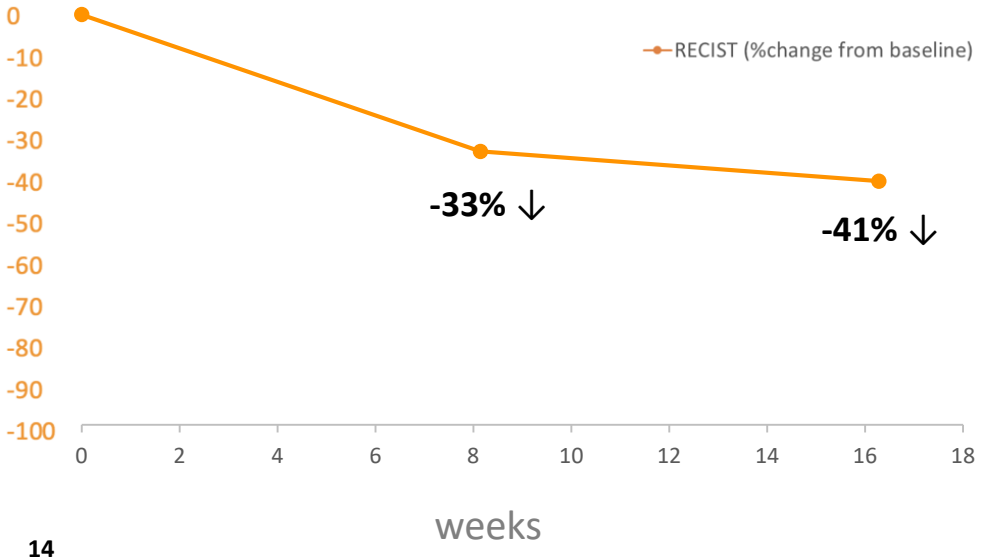
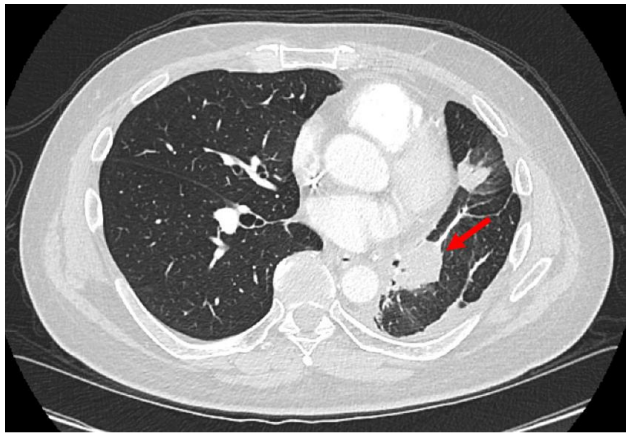
54-year-old male with *CD74-NRG1* NSCLC

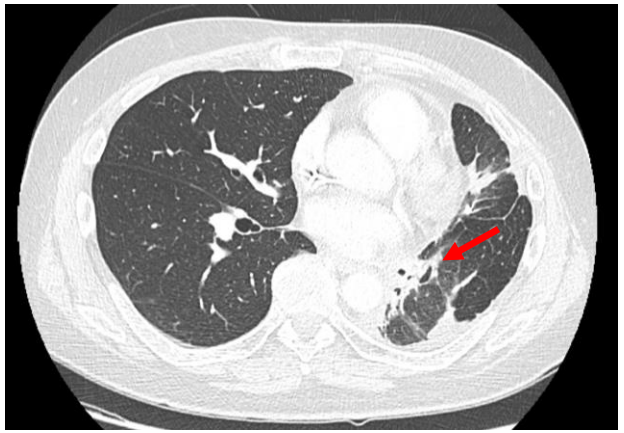


4.5+ months MCLA-128, 750 mg IV, q2w
Maximal related toxicity ≤ grade 1





Baseline



Week 16

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MCLA-128 Metastatic breast cancer Phase 2 combination trial update

KEY TAKEAWAYS

- After completing Phase 2, Merus will only advance development in metastatic breast cancer or gastric cancer with a collaborator
- ER+ cohort is now closed to accrual, >40 patients enrolled
- Merus plans to continue to enroll another approximately 10 patients to reach target accrual in the HER2+ cohort
- Expect to present mature Phase 2 results at medical conference in 2020
  - Primary endpoint of clinical benefit rate at 24 weeks for both cohorts

MCLA-128 MBC INTERIM EFFICACY ANALYSIS

	HER2+ cohort	ER+/HER2^{low} cohort
# patients treated	24	40
Median # of prior lines in metastatic setting	3	2
Visceral involvement, %	71	85
Estimated DCR, %	75	40
Estimated ORR, %	4 (confirmed) 17 (unconfirmed)	0

- Enrollment as of August 31, 2019. Data cut off October 23, 2019
- 100% of the patients in HER2+ cohort received prior trastuzumab, pertuzumab, HER2-directed antibody drug conjugate
- 100% of the patients in ER+/HER2^{low} cohort received prior CDK4/6 inhibitor
- DCR (Disease Control Rate) is defined as the proportion of patients at first assessment who have achieved complete response, partial response and/or stable disease as the best overall response to therapy.
- ORR (Overall Response Rate) is defined as the proportion of patients who have a partial or complete response as the best overall response to therapy.

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Phase 1/2 in NRG1 solid tumors, phase 2 in metastatic breast cancer (combo)

WHOLLY-OWNED	TARGETS	INDICATION / DRUG COMBINATION	PRE-IND	PHASE 1	PHASE 2
MCLA-128	HER3 x HER2	NRG1 Solid tumors (monotherapy)		■	
		Metastatic Breast (2 cohorts)			■

MRUS Fully Owned

	DESIGN	ENDPOINTS	STATUS
NRG1 Solid Tumors (Monotherapy)	**Phase 1/2 Study** Phase 1 : dose escalation Phase 2 : exploration in solid tumor cohorts Dose: 750mg every 2 weeks	• Safety • Preliminary anti-tumor activity	• Well tolerated as Single-Agent in over 100 patients as of Jan. 2019 • Clinical POC achieved
Metastatic Breast Cancer (MBC)	**Phase 2 Study in combination with 2 cohorts in MBC** Cohort 1: HER2+ (MCLA-128 + Herceptin + Chemo) Cohort 2: ER+/HER2^{low} (MCLA-128 + Hormone Therapy) Dose: 750mg every 3 weeks	• Clinical benefit at 24 weeks	• CBR readout at a medical conference in 2020 • Ph. 3 only with a Partner

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Clinical Programs
MCLA-117

CD3 x CLEC12A Biclonics®

Merus

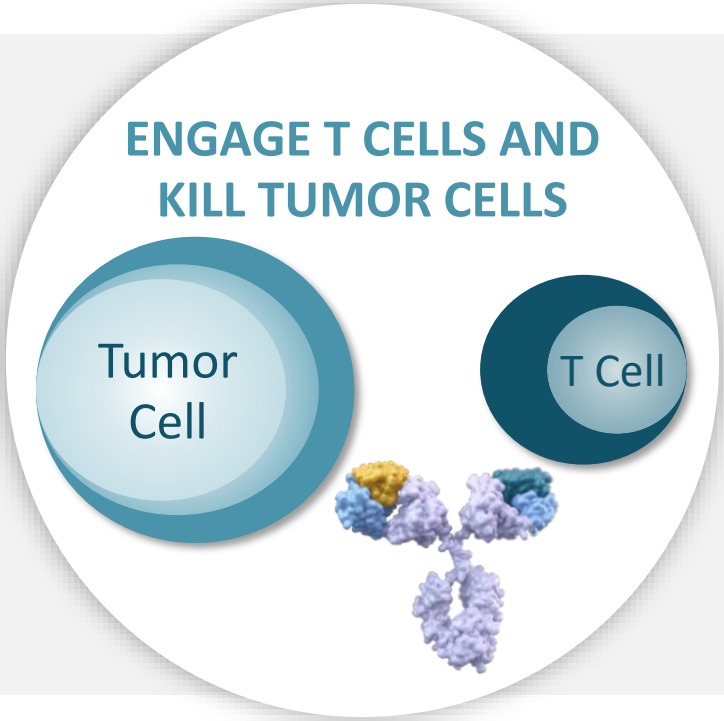
T cell engager for acute AML addressing a potential first-in-class target

MCLA-117 efficiently activates and redirects T cells to kill CLEC12A-expressing acute myeloid leukemia (stem) cells

CLEC12A

Expressed by tumor (stem) cells in ~ 90-95% of AML patients

Expression restricted to hematopoietic system = potential less off-tumor toxicity



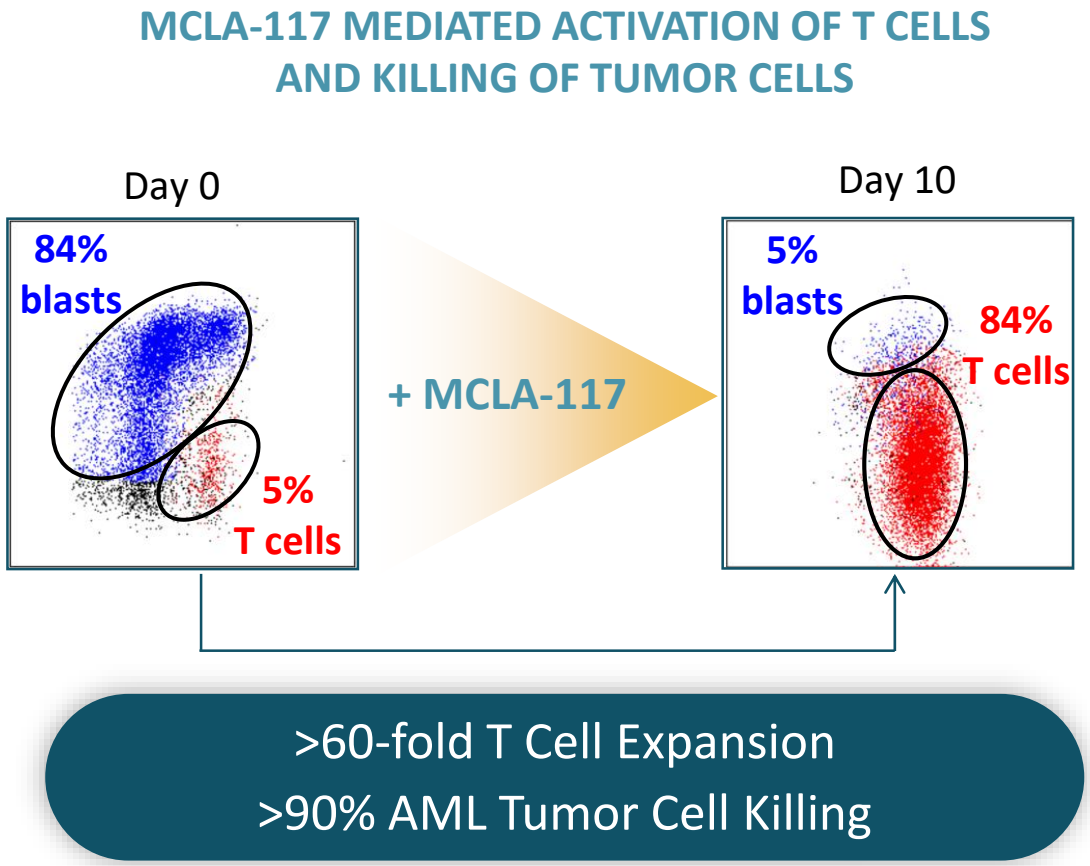
CD3

Low affinity CD3 arm and silenced Fc for controlled T cell activation to reduce toxicity and improve biodistribution

Merus

Potent activation of T cells in preclinical studies

- MCLA-117 efficiently activates and redirects T cells to kill CLEC12A-expressing AML(stem) cells
- Designed to spare the formation of platelets and red blood cells during therapy
- Active across AML patient subpopulations



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Phase 1 Trial

	TARGETS	INDICATION / DRUG COMBINATION	PRE-IND	PHASE 1	PHASE 2
MCLA-117	CLEC12A x CD3	Acute Myeloid Leukemia (AML)			

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DESIGN	ENDPOINTS	STATUS
Single-arm, open-label, dose escalation w/ safety dose expansion • Up to 50 patients with relapsed / refractory AML • Starting dose determined using MABEL dose escalation requirements • Protocol amended July 2019 to allow for the exploration of higher doses	• **Primary Endpoints:** safety, tolerability • **Secondary Endpoints:** PK/PD, anti-tumor response, clinical benefit	• Ongoing in Europe and the U.S. • Preliminary anti-tumor activity has been observed • Initial data expected at medical conference 1H 2020

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Clinical Programs
MCLA-158

Lgr5 x EGFR Biclonics®

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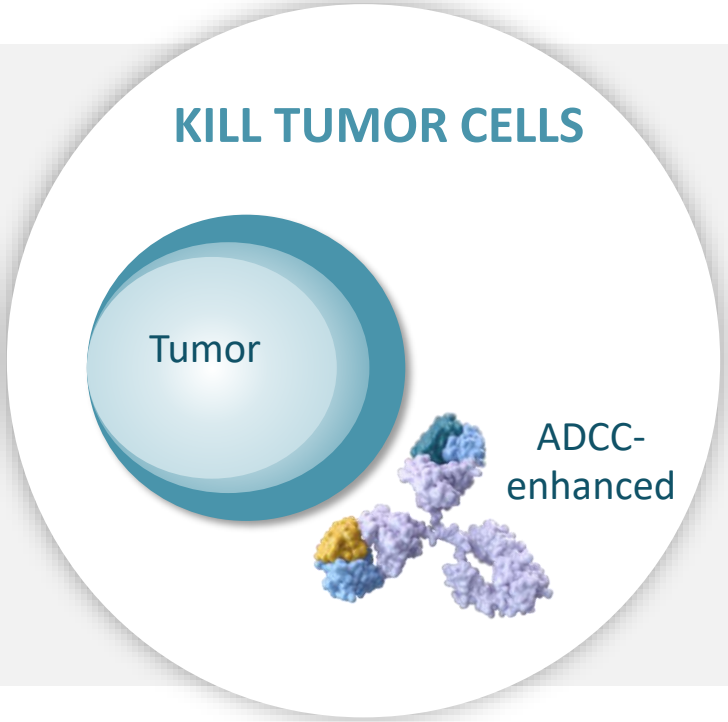
Potently blocks EGFR signaling in Wnt dysregulated solid tumors

Potential to be first colorectal cancer treatment to block growth of tumors with RAS mutations (~50% of patients), a high unmet need

Lgr5

Expressed by intestinal cancer initiating cells

Identified through Merus functional screening and organoid discovery methods



EGFR

Blocks growth in Wnt dysregulated tumors including RAS^{mut}

Preclinical data shows higher potency than Cetuximab

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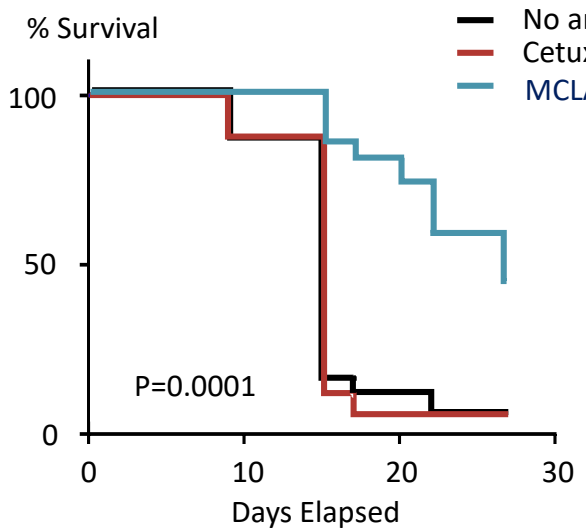
Superior growth inhibition and selectivity of tumor versus healthy tissue

- MCLA-158 is active in xenograft models that are resistant to treatment with Cetuximab
- Unlike Cetuximab, MCLA-158 discriminates between organoids derived from tumor and healthy tissue

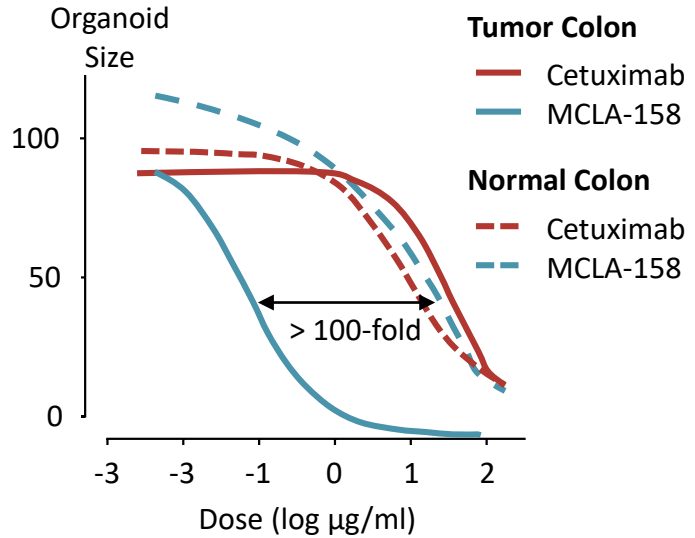
INHIBITION OF ORGANOID GROWTH

Superior **ACTIVITY** compared to Cetuximab



KILLING OF ORGANOIDS FROM TUMOR TISSUE

Superior **SELECTIVITY** for tumor-derived organoids



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Phase 1 Trial

	TARGETS	INDICATION / DRUG COMBINATION	PRE-IND	PHASE 1	PHASE 2
MCLA-158	Lgr5 x EGFR	Solid tumors			

MRUS Fully Owned

DESIGN	ENDPOINTS	STATUS
Global open-label, multicenter dose escalation w/ safety dose expansion phase • Patients with solid tumors • Initial focus on metastatic colorectal cancer	• **Primary endpoint:** safety and tolerability of defined dose • **Secondary endpoint:** single-agent preliminary anti-tumor activity	• On track • Emerging Phase 1 safety data expected YE 2019

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Clinical Programs
MCLA-145

CD137 x PD-L1 Biclonics®

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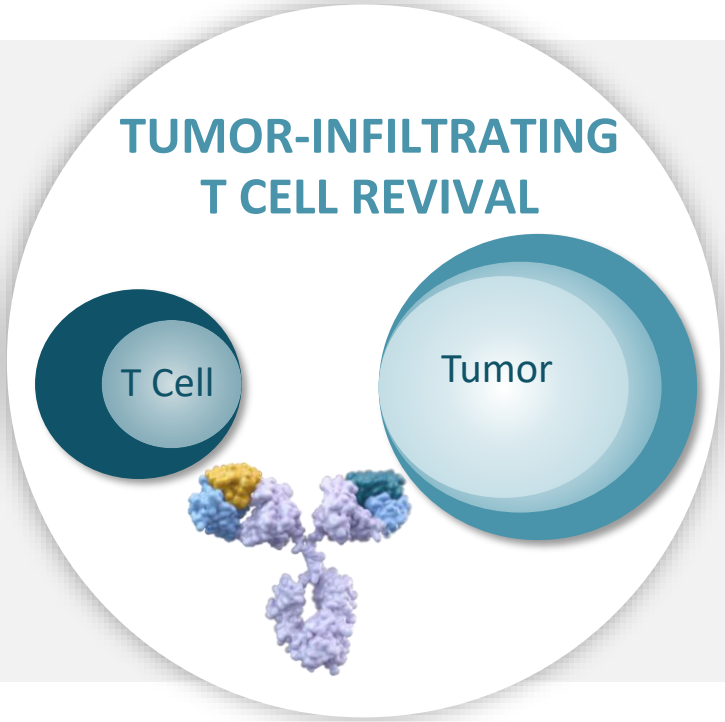
Recruit, activate and prevent exhaustion of tumor-infiltrating T cells

Triple action for potent and durable T cell activation in the tumor micro-environment

CD137

Potent activation of tumor infiltrating T cells dependent on PD-L1 expressing tumor cells and macrophages



PD-L1

Targeting to PD-L1 positive cells in the tumor

Blocking of T cell inhibitory PD-1/ PD-L1 interactions in tumor

Attracting T cells into the tumor

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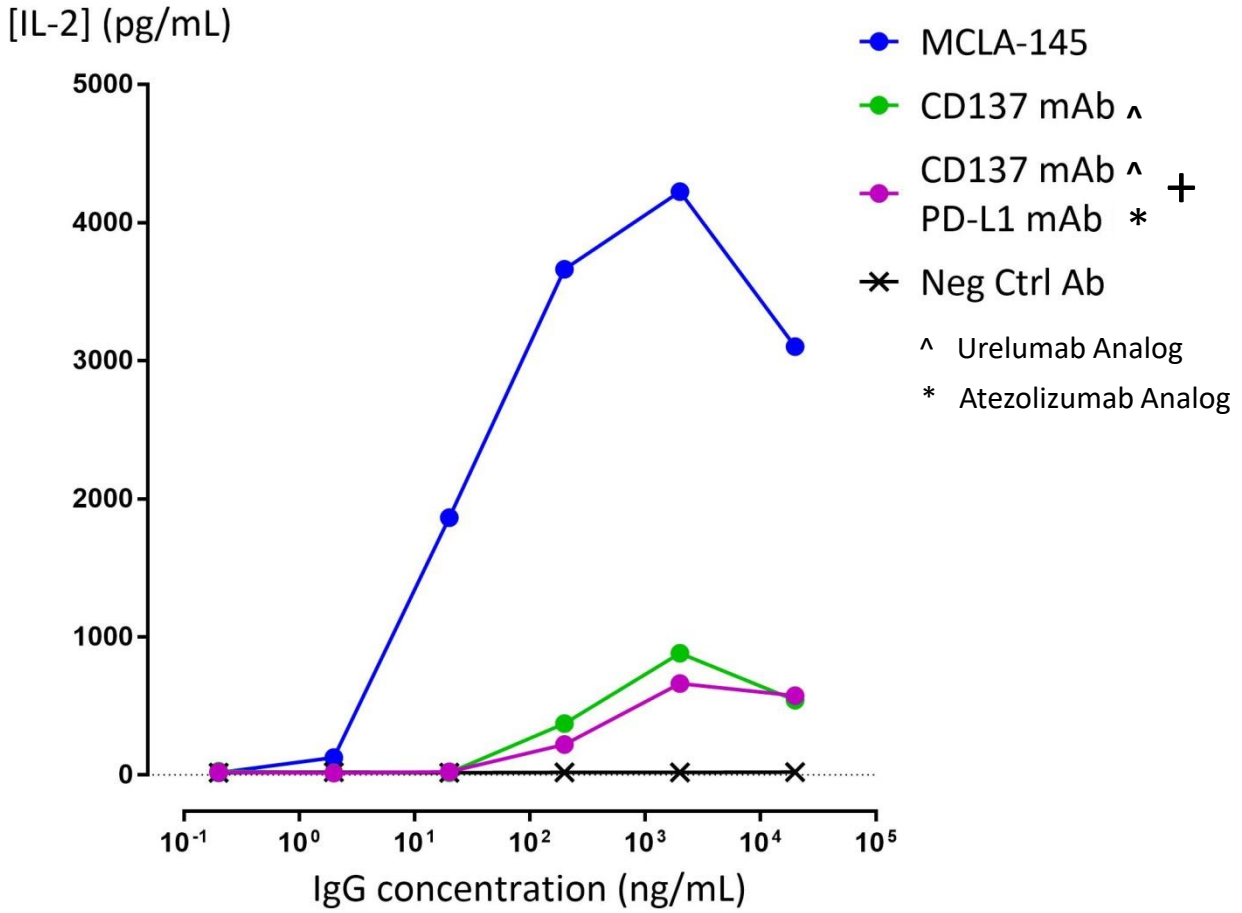
Demonstrated Potent T Cell Activation

- Binds to PD-L1 and CD137
- Preclinical work demonstrates
  - recruitment of T cells into the tumor
  - blocking of inhibitory PD-1/PD-L1 axis
  - potent T cell activation
- Potential to overcome the known side effects of CD137 agonists in development

MCLA-145 preclinical data presented at AACR 2019

PRIMARY T CELL TRANSACTIVATION ASSAY



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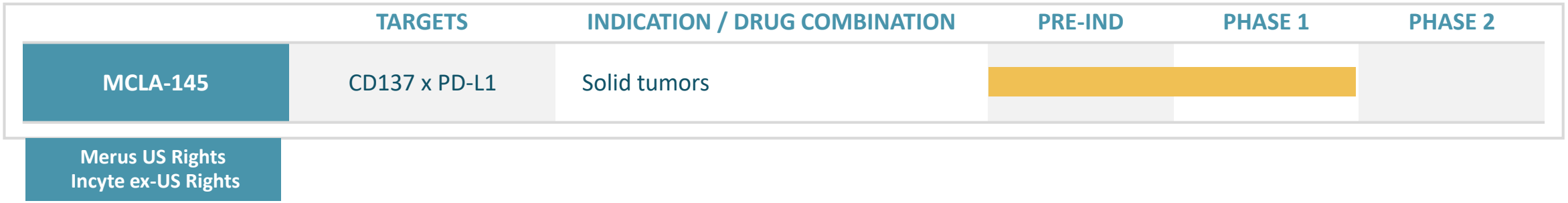
Phase 1 trial initiated in May 2019



DESIGN	ENDPOINTS	STATUS
Global open-label, multicenter dose escalation with dose expansion phase • Patients with advanced solid tumors	• **Primary endpoint:** dose finding, safety and tolerability • **Secondary endpoint:** single-agent preliminary activity	• IND cleared January 2019 • First patient dosed May 9, 2019

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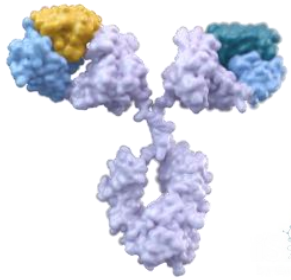
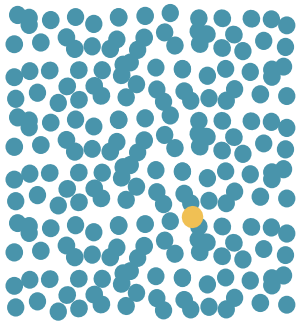
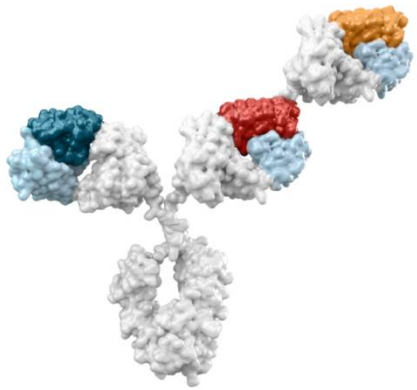
The new Triclonics™ platform for additional differentiated modes of action

The BICLONICS® Beginning

Our existing foundation…



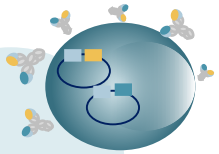




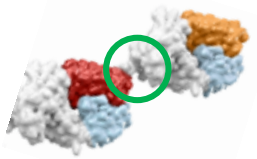
Designed by nature.
Improved by Merus.

Next Generation TRICLONICS™ Platform

for 2 or 3 different targets



Common light chain for unforced pairing with 3 (different) V_H regions



Linker diversity for added functionality

1:1:1 or 2:1 format for new biology/modes of action

Merus

Merus

Pioneering Bispecific Antibodies