Merus

Pioneering Bispecific Antibodies

September 4, 2019

Disclaimer

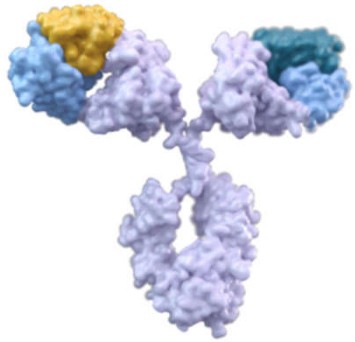
This presentation (including any oral commentary that accompanies this presentation) contains forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995. All statements contained in this presentation that do not relate to matters of historical fact should be 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. 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.

Merus

Merus: Pioneering Bispecific Antibodies Since 2006

4 clinical-stage bispecific antibodies in oncology



Designed by nature. Selected by Merus.

Discovery of novel modes of action based on target combinations

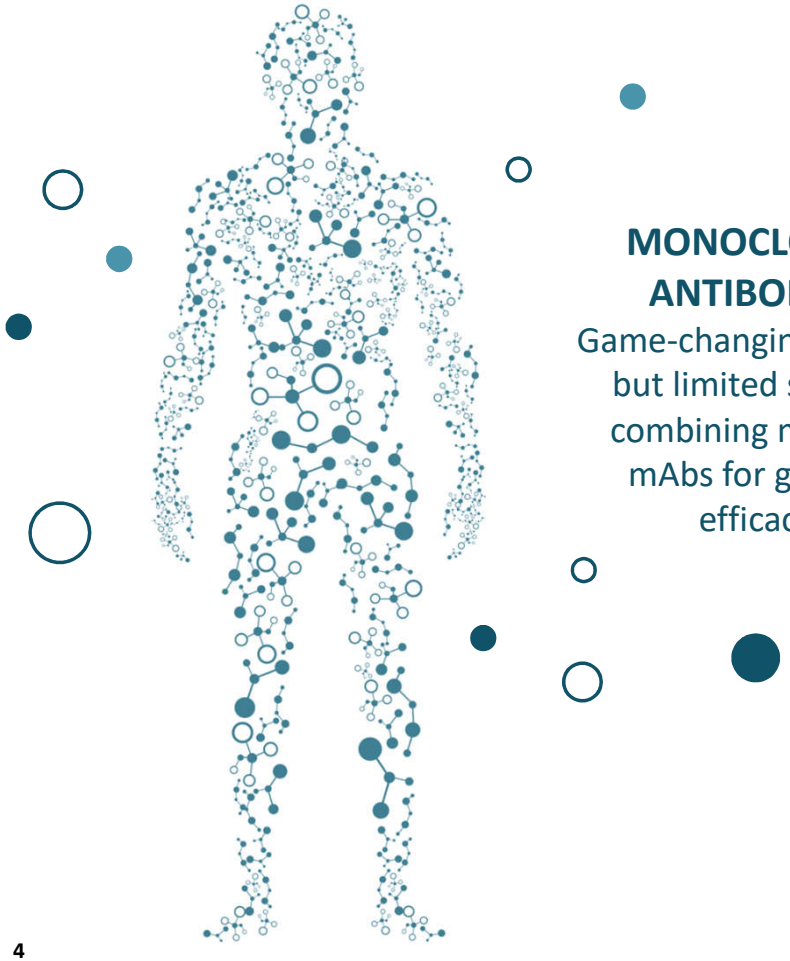
Multiple near term milestones anticipated in next 12 months

Sophisticated proprietary Biclonics® and Triclonics™ technology platforms

Fully integrated discovery-to-manufacturing capabilities

Merus

The Next Wave of Antibodies in Cancer Treatment



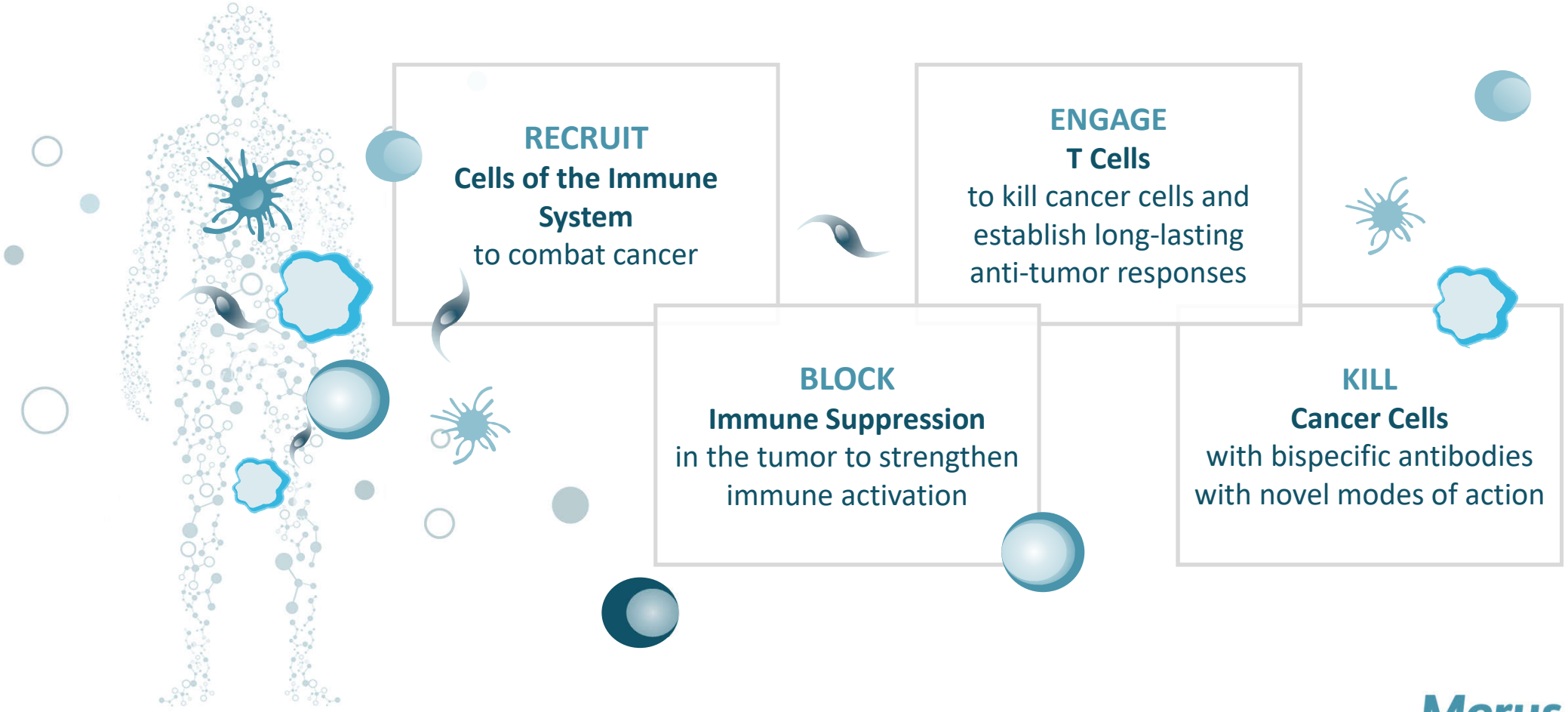
MONOCLONAL ANTIBODIES
Game-changing impact, but limited success combining multiple mAbs for greater efficacy

BISPECIFIC ANTIBODIES
Offering novel modes of action and new biology

High Potential for Cancer Immunotherapy and More

Merus

The Promise of Bispecific Antibodies



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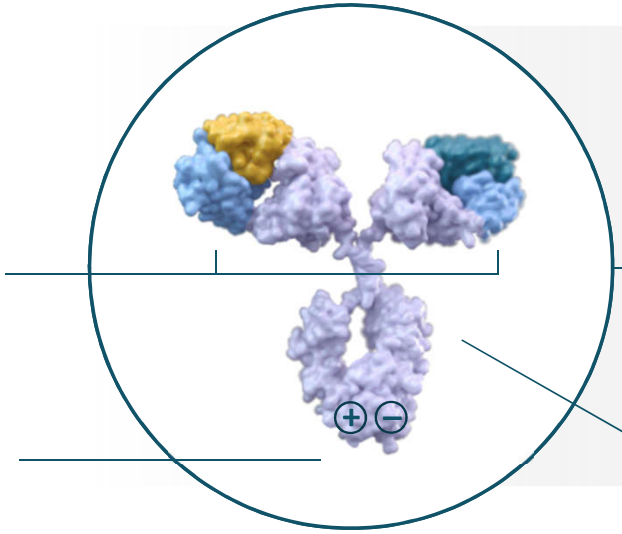
Biclonics® — Designed by Nature. Selected by Merus.

BICLONICS®

Merus' Bispecific Antibody Format 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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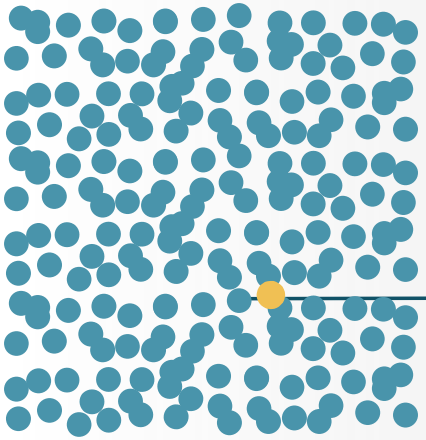
Biclonics® — Designed by Nature. Selected by Merus.

HUMAN ANTIBODY GENERATION

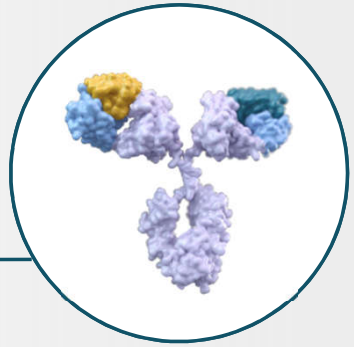
MeMo®
Transgenic Mouse

PANEL GENERATION



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

FUNCTIONAL SCREENING



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

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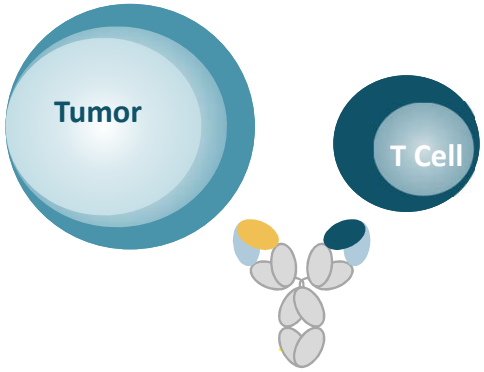
Biclonics® Recruit Innate & Adaptive Immunity To Kill Tumors

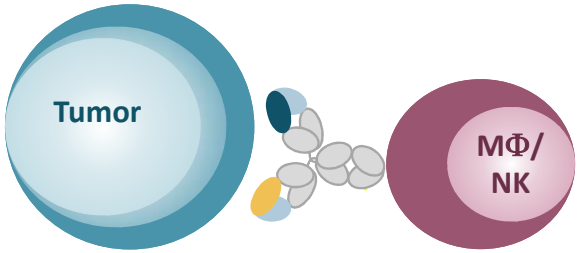
Our Optimal Target Pairs Have First or Best in Class Potential

T CELL ENGAGE AND KILL



MCLA-117: CLEC12A x CD3

DUAL TUMOR TARGETING TO RECRUIT AND KILL

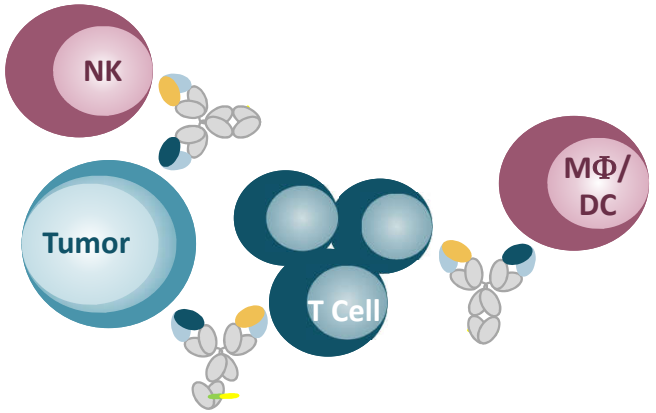


MCLA-158: Lgr5 x EGFR

MCLA-128: HER3 x HER2

MCLA-129: EGFR x c-MET

ACTIVATE ANTI-TUMOR IMMUNITY TO KILL



MCLA-145: CD137 x PD-L1

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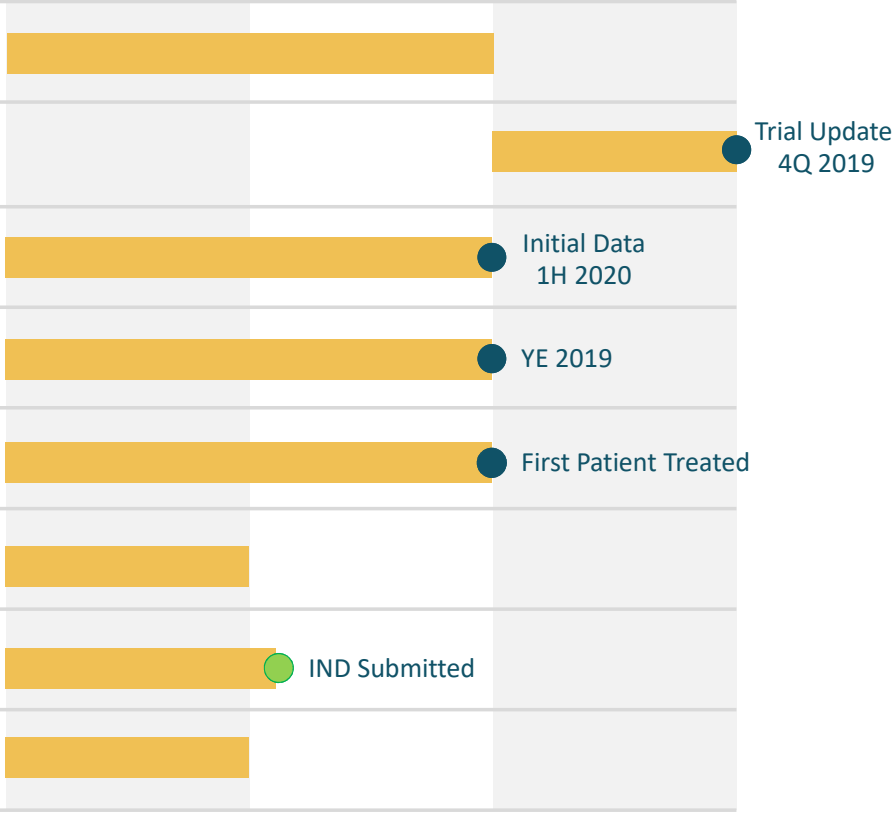
Leading Clinical Pipeline with Multiple 2019 Milestones

PROGRAM	TARGETS	INDICATION / DRUG COMBINATION
MCLA-128	HER3 x HER2	NRG1 Solid tumors (monotherapy)*
		Metastatic Breast (2 cohorts)
MCLA-117	CLEC12A x CD3	Acute Myeloid Leukemia (AML)
MCLA-158	Lgr5 x EGFR	Solid tumors
MCLA-145	CD137 x PD-L1	Solid tumors (Incyte) (ex- U.S.)
MCLA-129	EGFR x c-MET	Solid tumors (BETTA PHARMACEUTICALS) (China)
ONO-4685	PD-1 x CD3	Autoimmune disease (ono)
....	Undisclosed	Autoimmune disease (ono)



*Phase 1/2 Trial

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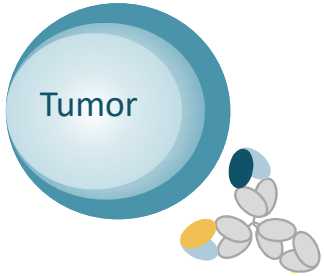
MCLA-128 – HER3 x HER2

Unique DOCK & BLOCK® approach potently inhibits tumor cell growth and survival; In clinic for multiple solid tumor indications

Block HER3

Blocks signaling even in high heregulin stress environments



KILL TUMOR CELLS

Dock HER2

Docks to HER2 abundantly expressed on tumor cells to access HER3

Combinations with HER2 targeted therapies possible

Metastatic Breast Cohort Phase 2 Trial Update Expected 4Q 2019

Note: neuregulin (NRG) is another name for the gene that expresses heregulin

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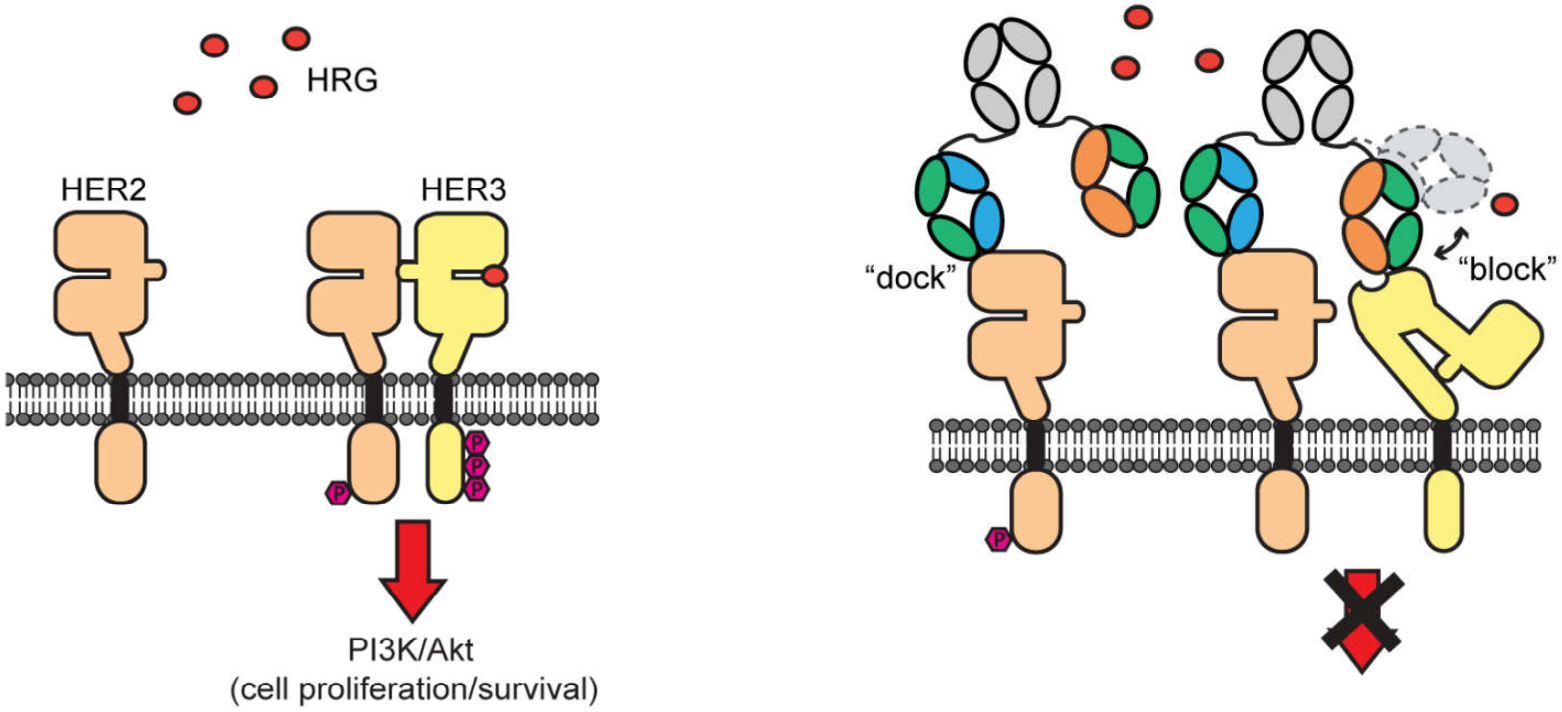
MCLA-128 – Potently Inhibiting the HER3 Signaling Pathway, a Known Driver of Tumor Growth and Survival



Note: neuregulin (NRG) is another name for the gene that expresses heregulin

Geuijen et al. (2018) Cancer Cell

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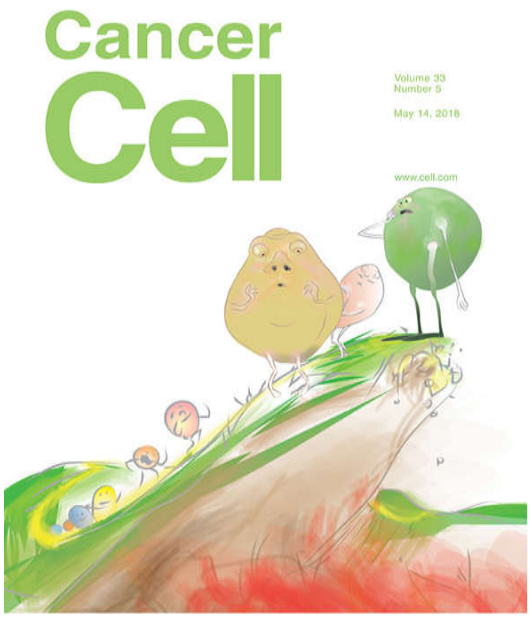
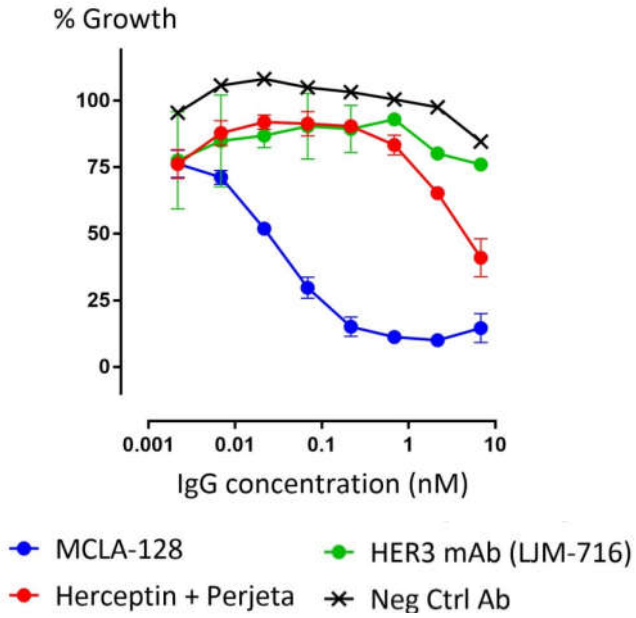
MCLA-128 – Potently Inhibits Heregulin-Driven Growth

PUBLISHED MAY 2018



SUPERIOR ACTIVITY SHOWN PRECLINICAL DATA



SAFETY AND TOLERABILITY DEMONSTRATED IN PHASE 1/2 TRIAL

>100 PATIENTS EVALUATED

MCLA-128 Dosing: 750 mg q3w

- Single agent well tolerated
- Low risk for immunogenicity

Refer to Merus Publications for more information on MCLA-128 published and presented data: https://merus.nl/publications/

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MCLA-128 – Phase 1/2 in Solid Tumors, Phase 2 in Combo MBC

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)			4Q 2019

	DESIGN	ENDPOINTS	STATUS
NRG1 Solid Tumors (Monotherapy)	**Phase 1/2 Study** Phase 1 : dose escalation Phase 2 : exploration in solid tumor cohorts	• Safety, preliminary anti-tumor activity	• Well tolerated • Clinical POC established in MBC • Clinical POC established in Gastric
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) Size: up to 120 patients in U.S. and Europe Dose: 750mg every 3 weeks	• Clinical benefit at 24 weeks	• Trial update 4Q 2019

*Phase 1/2 Trial

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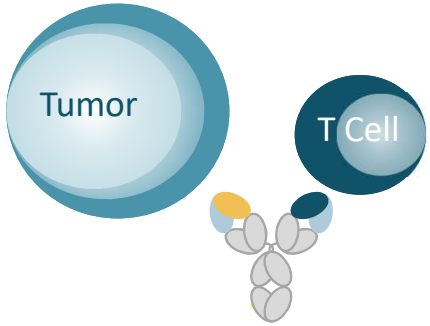
MCLA-117 – Harnessing the Killing Power of T Lymphocytes

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

CLEC12A

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

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



ENGAGE AND KILL

CD3

Low affinity CD3 arm and silenced Fc for controlled T cell activation to avoid toxicity and off-target effects

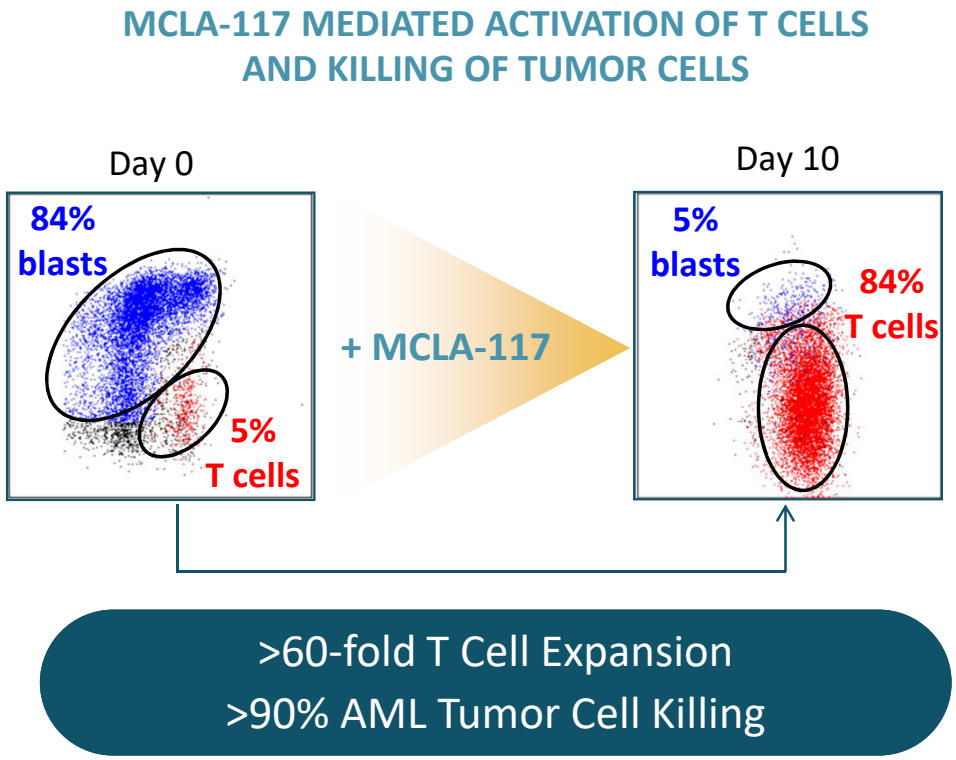
Balanced strategy for activity and safety

Initial data expected at medical conference 1H 2020

Merus

MCLA-117 –Demonstrated Controlled, 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



Experiment Conditions: Primary AML patient samples were cultured in presence of MCLA-117 (200 ng/mL) for 10 days in AML supporting medium. T cells:CD33, AML blasts:CD45.

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MCLA-117 – Phase 1 Trial

	TARGETS	INDICATION / DRUG COMBINATION	PRE-IND	PHASE 1	PHASE 2
MCLA-117	CLEC12A x CD3	Acute Myeloid Leukemia (AML)			1H 2019

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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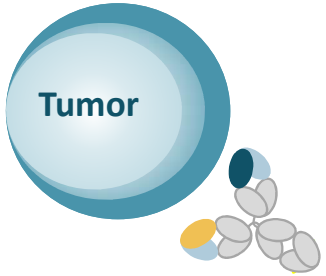
MCLA-158 – Lgr5 x EGFR

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



KILL TUMOR CELLS

EGFR

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

Preclinical data shows higher potency than Cetuximab

Emerging Phase 1 safety data expected end of 2019

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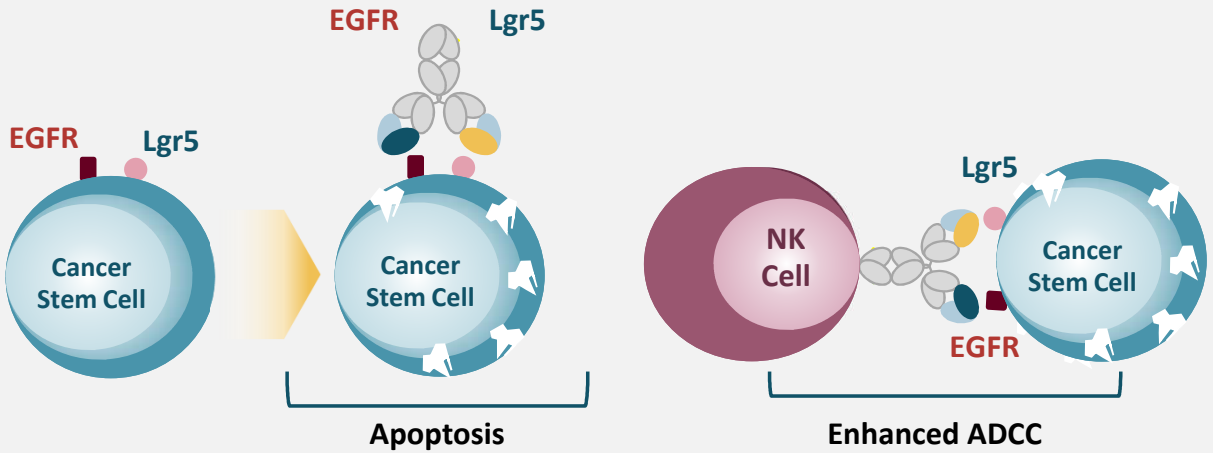
MCLA-158 – Differentiated Target and MOA

MCLA-158 Mechanism of Action



- MCLA-158 designed to eliminate cancer initiating cells that cause growth and metastasis
- Lgr5+ cells are the origin of gastrointestinal cancer
- EGFR x Lgr5 induces apoptosis, potently blocks EGFR signaling in Wnt dysregulated solid tumors

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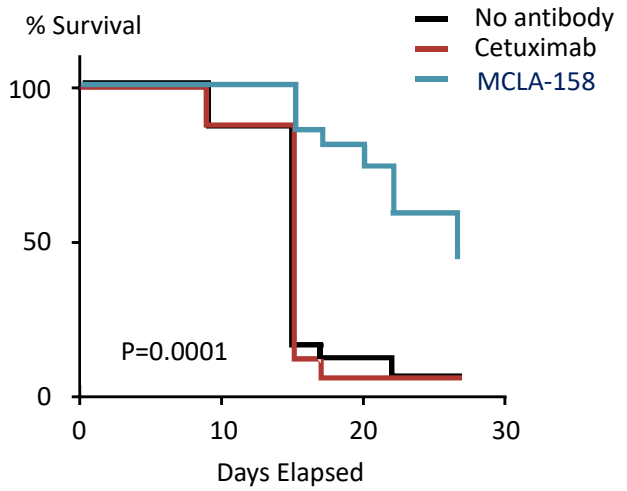
MCLA-158 – Key Preclinical Results in Colorectal Cancer (CRC)

Demonstrated Superior Growth Inhibition, Tolerability and Selectivity of Tumor vs. Healthy Tissue

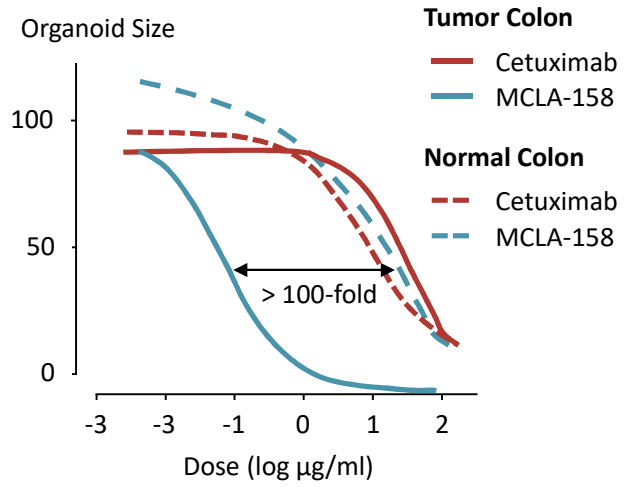
INHIBITION OF ORGANOID GROWTH

Superior **ACTIVITY**



KILLING OF ORGANOIDS FROM TUMOR AND HEALTH TISSUE

Superior **SELECTIVITY**



Superior **TOLERABILITY**

No skin rash in cynomolgus monkeys

When compare to EGFR targeting mAb,Cetuximab analogs

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MCLA-158 – Phase 1 Trial

	TARGETS	INDICATION / DRUG COMBINATION	PRE-IND	PHASE 1	PHASE 2
MCLA-158	Lgr5 x EGFR	Solid tumors			YE 2019

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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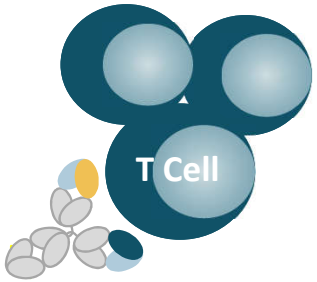
MCLA-145 – CD137 x PD-L1

Potent triple action designed to recruit and activate T cells and prevent their exhaustion for patients with solid tumors

CD137

Activate immune effector cells in context of tumor microenvironment



TUMOR T CELL REVIVAL

PD-L1

Attract T cells into the tumor and block inhibitory signals

Phase 1 First Patient Treated May 9 2019

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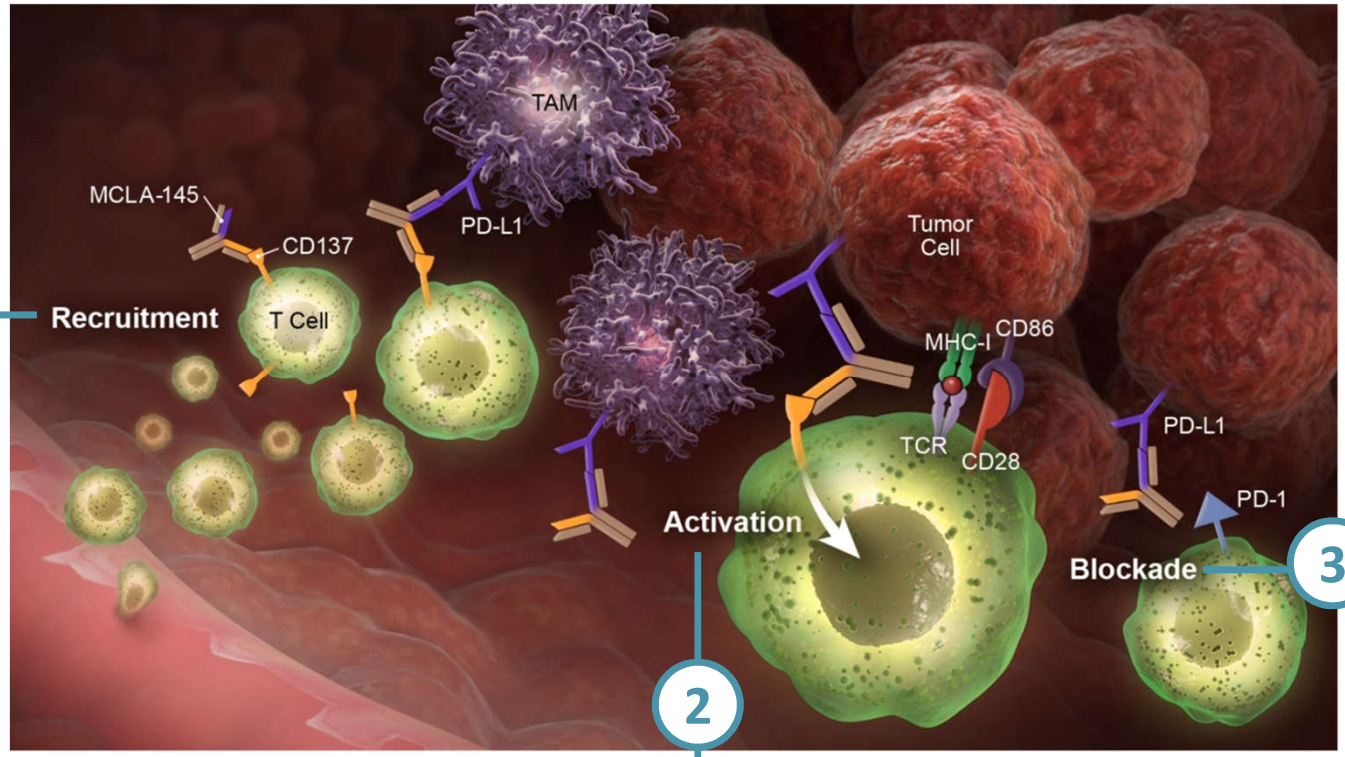
MCLA-145 – Triple Activity by a Single Biclonics®

PD-L1 Arm
Recruitment of T cells into tumor micro-environment



CD137 Arm
Potent activation of T cells in tumor

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

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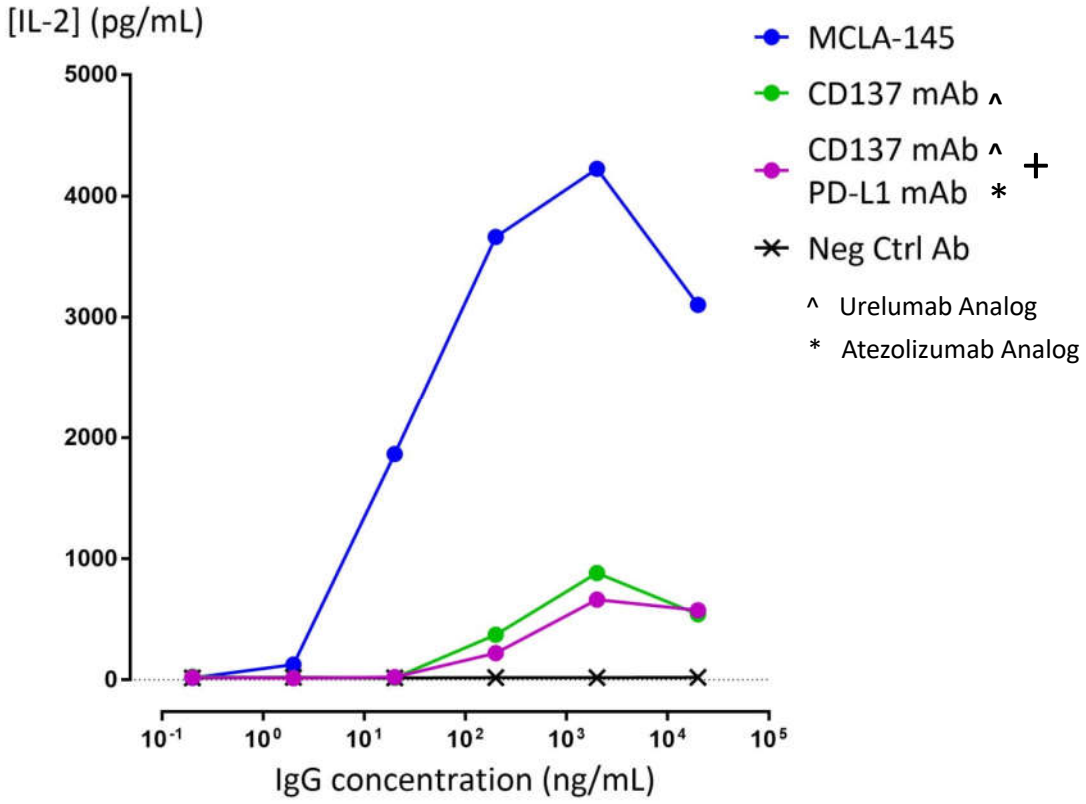
MCLA-145 – Demonstrated Potent T Cell Activation

- 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



Experiment Conditions: T cells PBMC donor stimulated with antibody in the presence of PD-L1 on CHO cells. Antibody added to pre-coated anti-CD3 clone OKT3 plates. Purified T cells, CHO-PD-L1 cells added and incubated for 72 hrs at 37°C. Readout IL-2.

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MCLA-145 – Phase 1 Trial Initiated May 2019

	TARGETS	INDICATION / DRUG COMBINATION	PRE-IND	PHASE 1	PHASE 2
MCLA-145	CD137 x PD-L1	Solid tumors			May 9 2019

DESIGN	ENDPOINTS	STATUS
Global open-label, multicenter dose escalation w/ 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

Merus

Leading Collaborators Increasing Biclonics® Reach

Collaborator Focus On

Ex U.S. Development

Incyte

Merus retains MCLA-145 U.S. rights

Collaborator Focus On

China Development

BETTA PHARMACEUTICALS

先声药业 Simcere

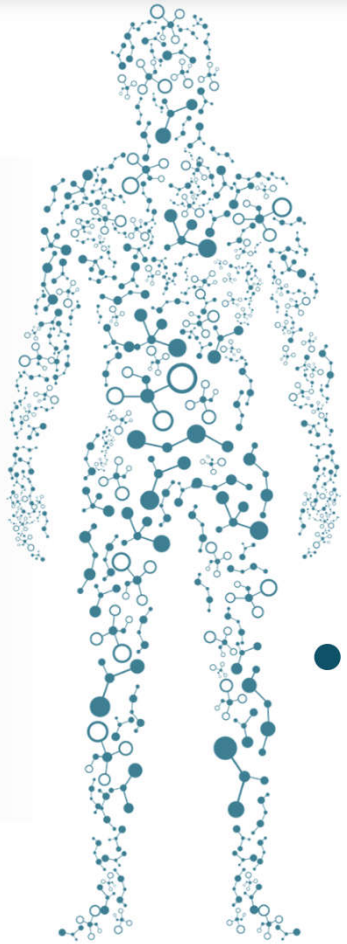
Merus retains Rest-of-World rights

Merus

Expanding Biclonics® Platform

Cancer

Next-Gen Tech



Autoimmune Disease

ONO PHARMACEUTICAL CO.,LTD.

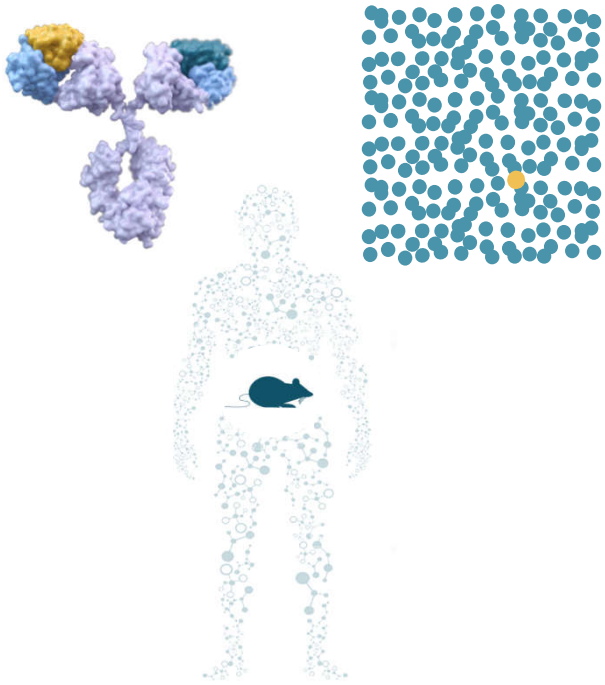
Exploring new formats, designs and targets

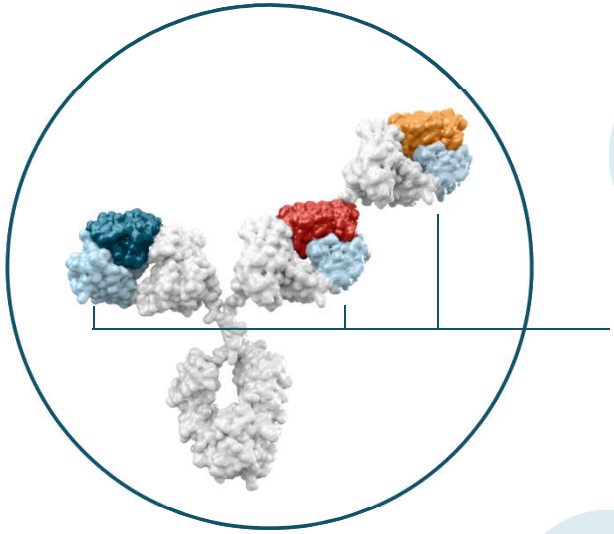
Merus

Proprietary Triclonics™ Platform…Expanding Merus Capabilities

The BICLONICS® Base

Our existing foundation…





TRICLONICS™ Platform

for 2 and 3 different target combinations

1:1:1 or 2:1 format

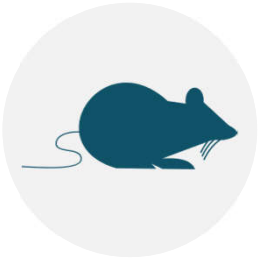
Common light chain for unforced, natural pairing with three (different) V_H regions

New biology and modes of action

Merus

Strong Intellectual Property Positioning

PLATFORM

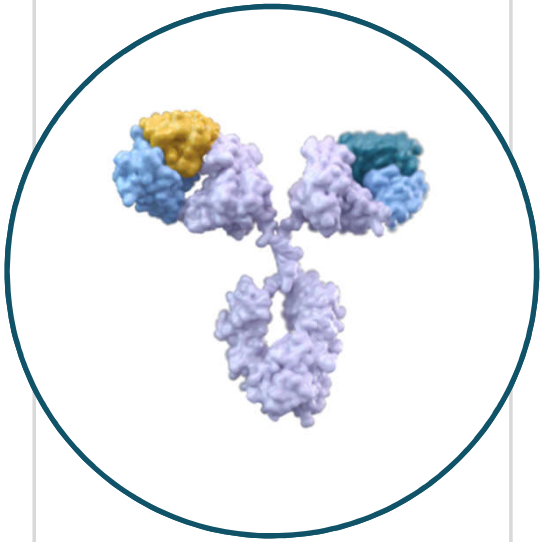


MeMo® Transgenic Mouse

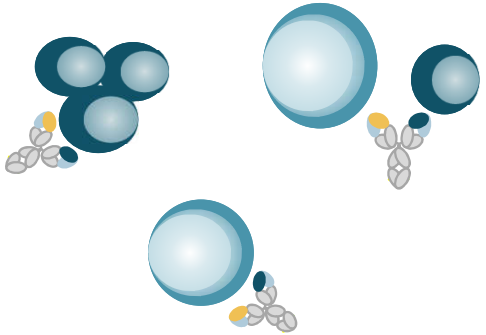
Spleen to Screen®

Proprietary host cells

DEKK heterodimerization



PROGRAMS

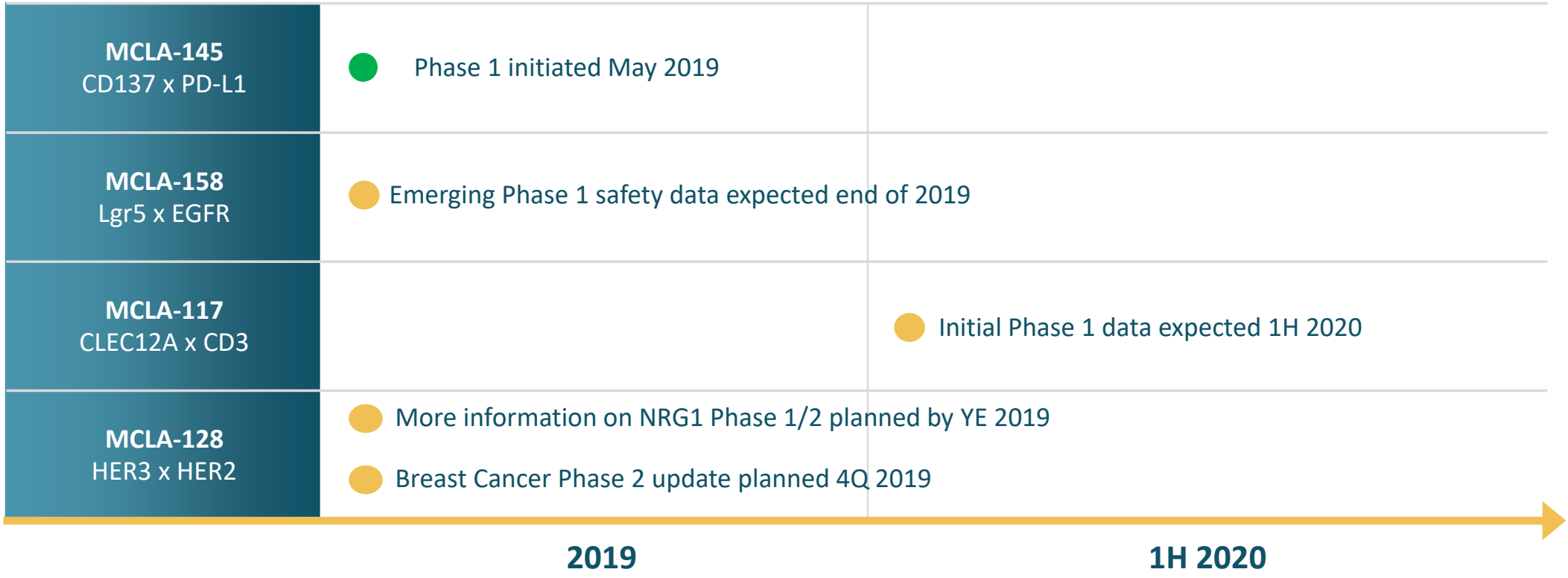


Multiple Biclonics® Triclonics™ with novel modes of action

Uniquely positioned to develop innovative bispecific antibody therapeutics

Merus

Multiple Near Term Milestones Anticipated



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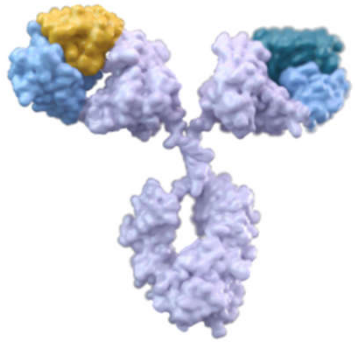
Merus: Pioneering Bispecific Antibodies Since 2006

4 clinical-stage bispecific antibodies in oncology



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Multiple near term milestones anticipated in next 12 months

Sophisticated proprietary Biclonics® and Triclonics™ technology platforms

Fully integrated discovery-to-manufacturing capabilities

Merus

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Pioneering Bispecific Antibodies