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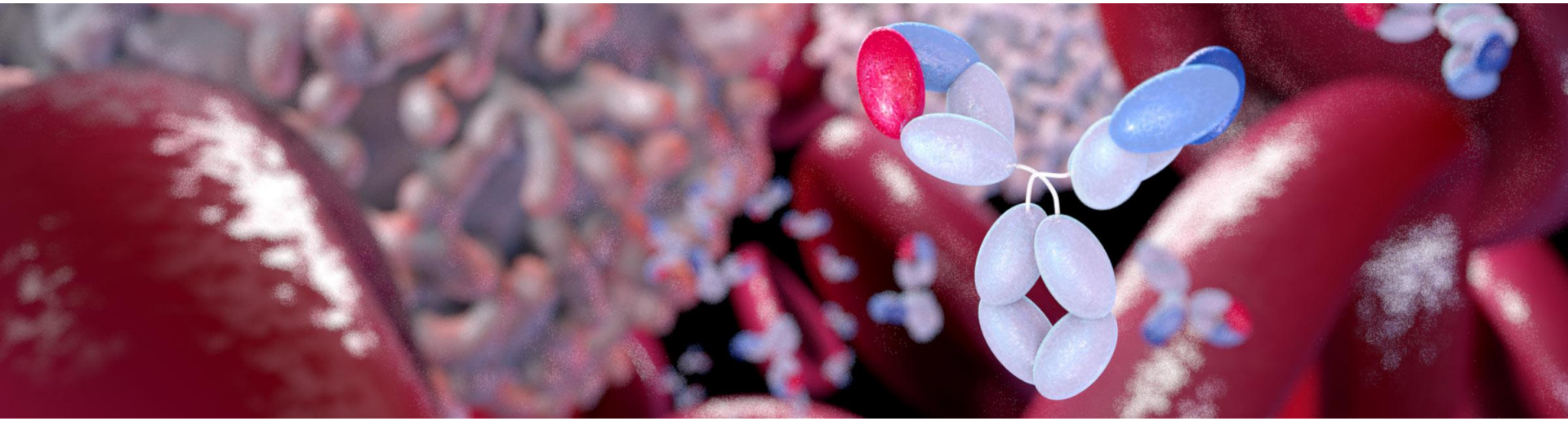

Battling Cancer with Bispecific Antibodies

March 2017

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 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 final prospectus filed with the Securities and Exchange Commission, or SEC, on May 20, 2016 relating to our Registration Statement on Form F-1, 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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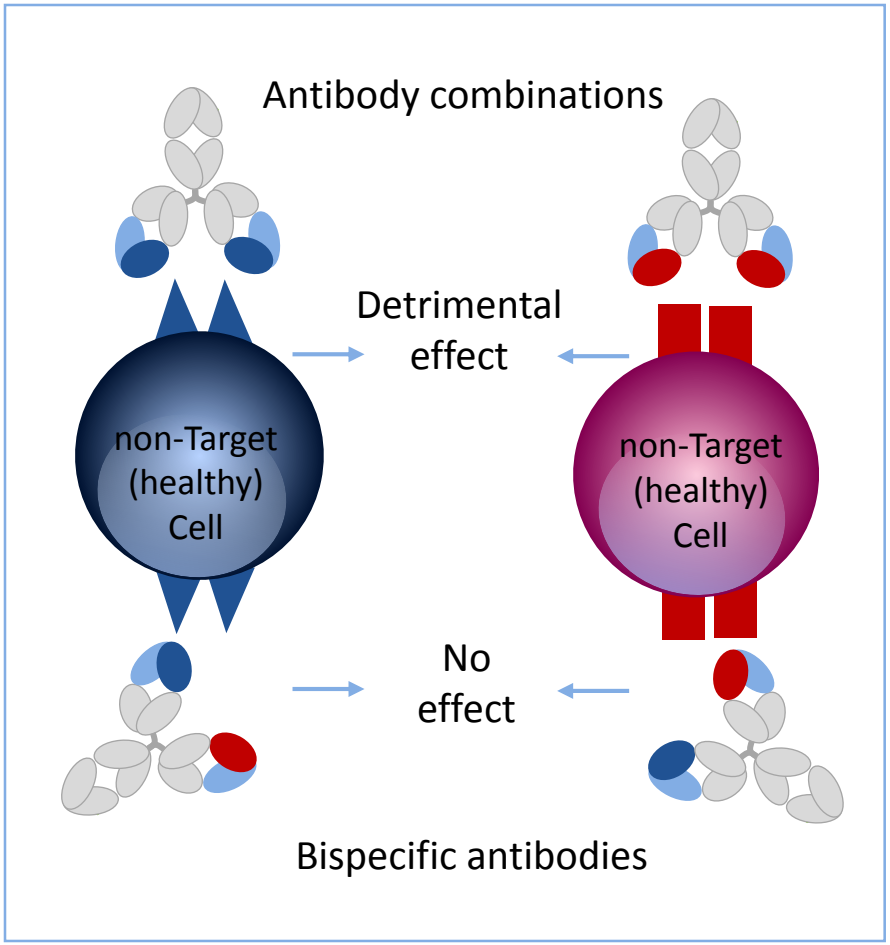
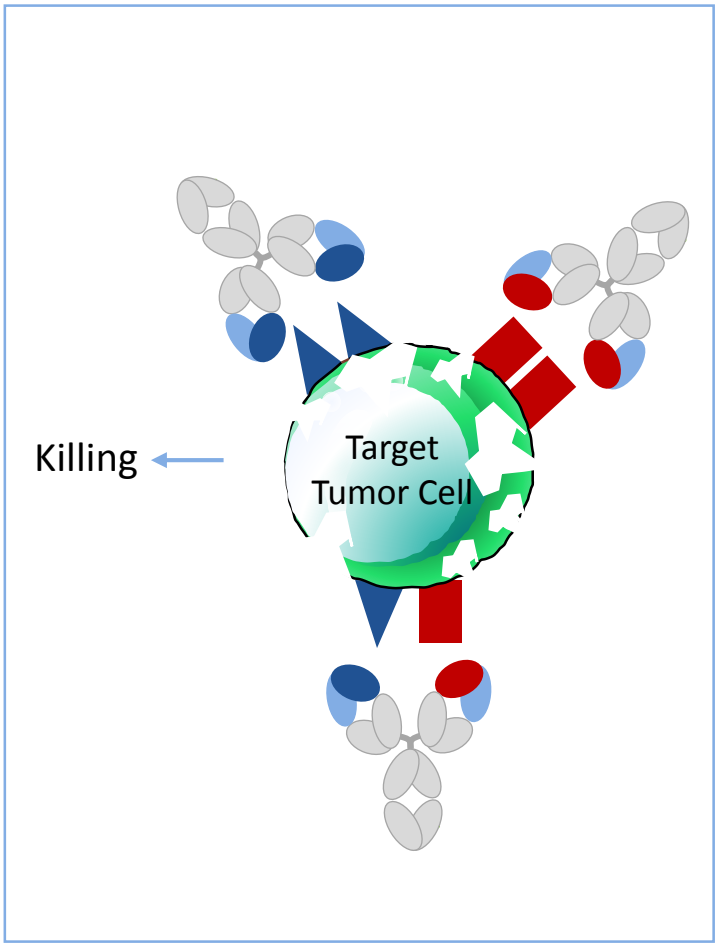
UNIQUE PROPERTIES OF BISPECIFIC ANTIBODIES

- Less toxicity compared to combinations of monoclonal antibodies





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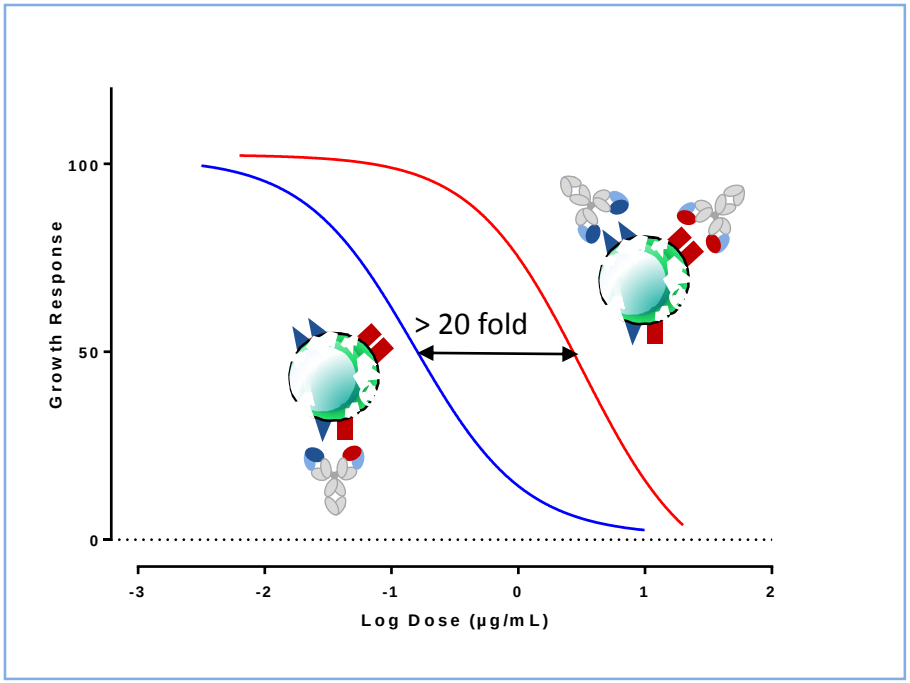
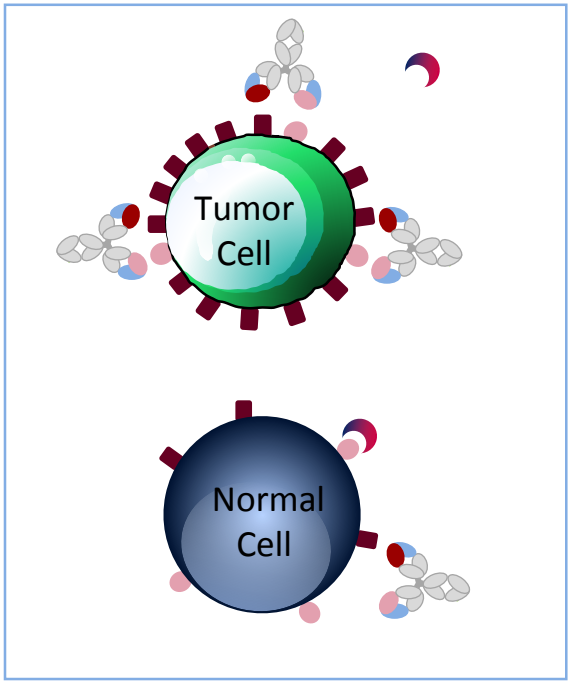
UNIQUE PROPERTIES OF BISPECIFIC ANTIBODIES

- Higher potency compared to combinations of monoclonal antibodies
- Novel mechanisms of action; ‘dock and block’





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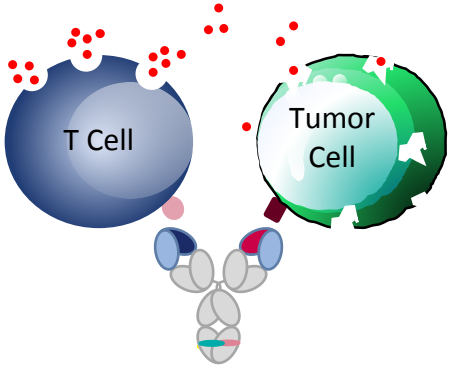
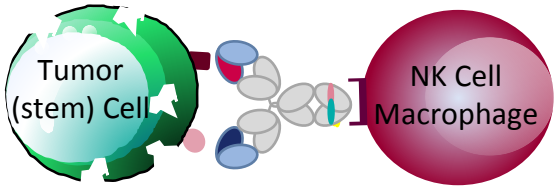
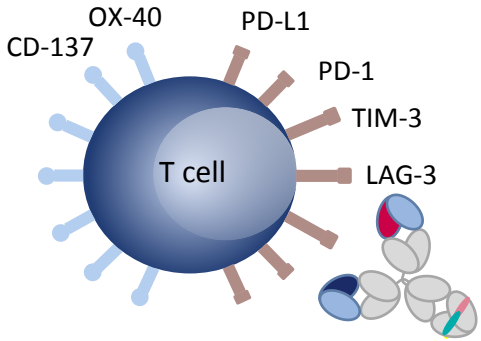
MISSION AND STRATEGY

Develop differentiated cancer therapeutics

based on bispecific antibodies that activate the immune system to kill tumor cells

Engage	Unlock	Activate/Revive
T lymphocytes to kill tumor cells	differentiated target biology	Tumor Killer Cells
		
MCLA-117	MCLA-128 and MCLA-158	Preclinical

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BATTLING CANCER WITH BISPECIFIC ANTIBODIES

Focus	• Bispecific antibodies for unmet medical needs in various cancers • Recruit the immune system to eliminate cancer cells
Technology	• Biclonics® Platform – full length IgG human bispecifics • In format functional screening – differentiated products
Lead Product Candidates	• MCLA-128 for solid tumors in Phase I/2 clinical development • MCLA-117 for AML first in human Phase 1/2 clinical develoment
Balance Sheet	• €66.3mn cash and equivalents at September 30, 2016 (May '16 IPO) • $200mn in upfront payments upon close of Incyte agreement (1Q17)
Experienced Team with Expertise in Immunology, Oncology and Antibody Discovery	• 51 employees (Q4 '16) located in Utrecht (NL) and Cambridge (MA)  Ton Logtenberg, PhD - CEO Professor in Immunology Cofounder and CSO Crucell  Hui Liu, PhD - CBO Molecular Biologist Global Head of BD&L Novartis Oncology  Mark Throsby, PhD - CSO Immunologist Director Antibody Discovery Crucell  Shelley Margetson, CGMA - COO Vice-president Finance PanGenetics  John Crowley, CPA - CFO SVP, Corp. Controller and CAO, Charles River Laboratories  L. Andres Sirulnik, MD, PhD - CMO Senior Global Clinical Program Head, Vice-President Novartis Oncology

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OVERVIEW INCYTE MERUS COLLABORATION

Up-Front Payment	Total $200m in up-front consideration for license and equity • Platform license: $120m • Equity investment: $80m ▪ 3,200,000 shares at $25/share representing 16.6% ownership
Product Rights	One program: Merus retains U.S. development and commercial rights Two programs: Merus has the option to co-fund 35% global development cost in exchange for 50% of U.S. profits with the right to co-detail a product in one of these 2 programs
Milestones & Royalties	Up to 8 programs: Incyte to independently fund all R&D and commercialization activities; Merus to receive: • Development, regulatory and sales milestones of up to $2.8 billion ($350 million/program) • Tiered 6-10% royalties on future sales by Incyte Upon commercialization of the product that Merus retains U.S. rights to, Merus and Incyte will pay reciprocal tiered 6-10 % royalties on sales in their respective territories

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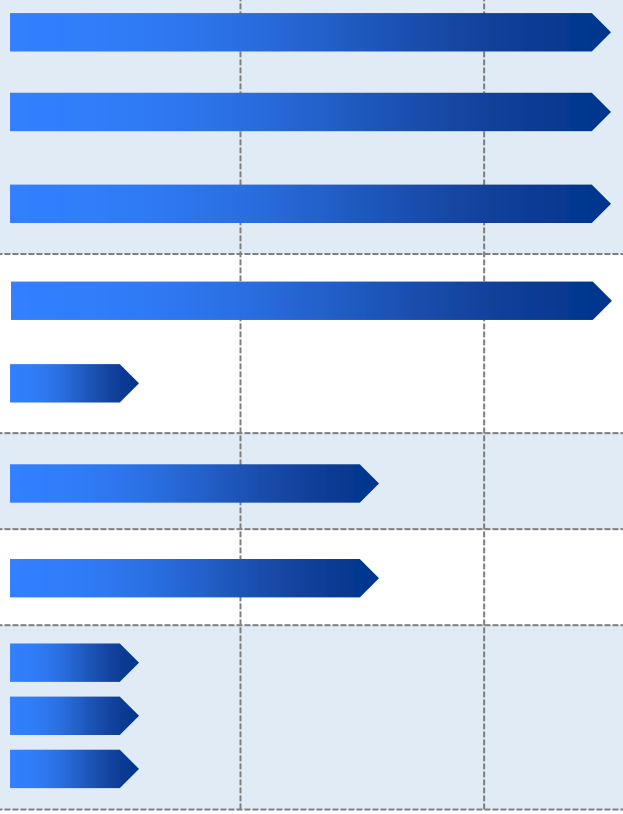
PIPELINE OF BICLONICS® PRODUCT CANDIDATES

Program	Targets	Indication	Pre-Clinical	IND/CTA	Phase 1/2
MCLA-128	HER2, HER3	Breast cancer			
		Gastric cancer			
		Ovarian cancer			
MCLA-117	CD3, CLEC12A	AML			
		MDS			
MCLA-158	Lgr5, EGFR	Colorectal cancer			
MCLA-145	PD-L1, undisclosed	Various solid tumors			
Multiple iMOD Programs[(1)]	Multiple immunomodulatory targets	Various solid tumors			



(1) Includes MCLA-134: PD-1 x TIM-3

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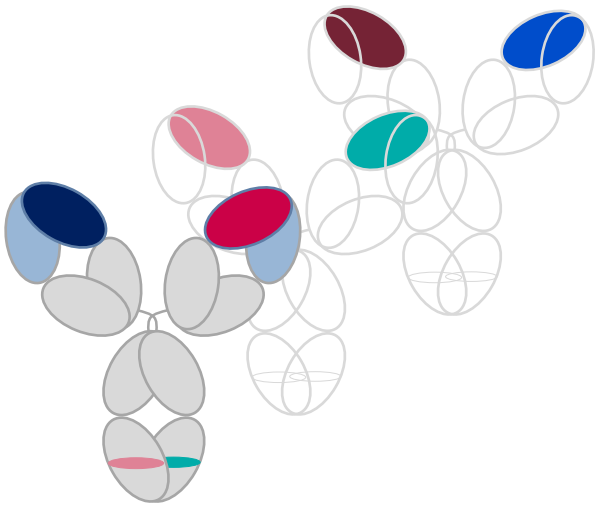
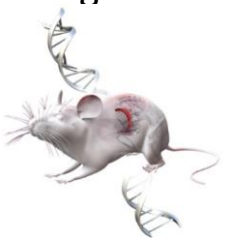


BICLONICS® PLATFORM

TECHNOLOGY SUITE FOR DIFFERENTIATING PRODUCTS

Human antibodies

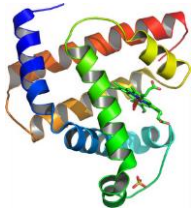
- MeMo® transgenic mouse for large panels of high quality common light chain human mAbs



cLC

Fc region engineering®

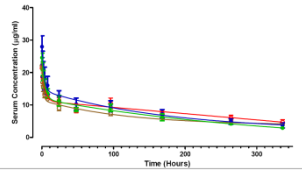
- CH3: > 99% pure Biclonics®
- CH2: Fc-silencing
- Enhanced ADCC



cLC

Predictable in vivo behavior

- normal pK in mice/cyno
- behaviour like IgG in antibody stress tests



DE

KK

Manufacturability

- stability: > 60 passages
- yield: 1 – 2.5 g/L
- scalability: 2000 L
- formulation: standard for IgG

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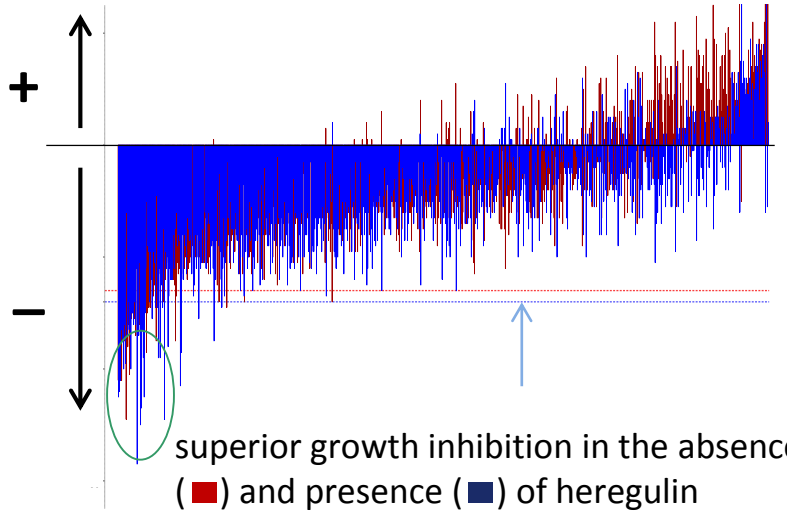
FUNCTIONAL SCREENING UNVEILS DIFFERENTIATED LEADS

- Cell-based screening of large collections of different Biclonics® unveils product leads with differentiated modes of action

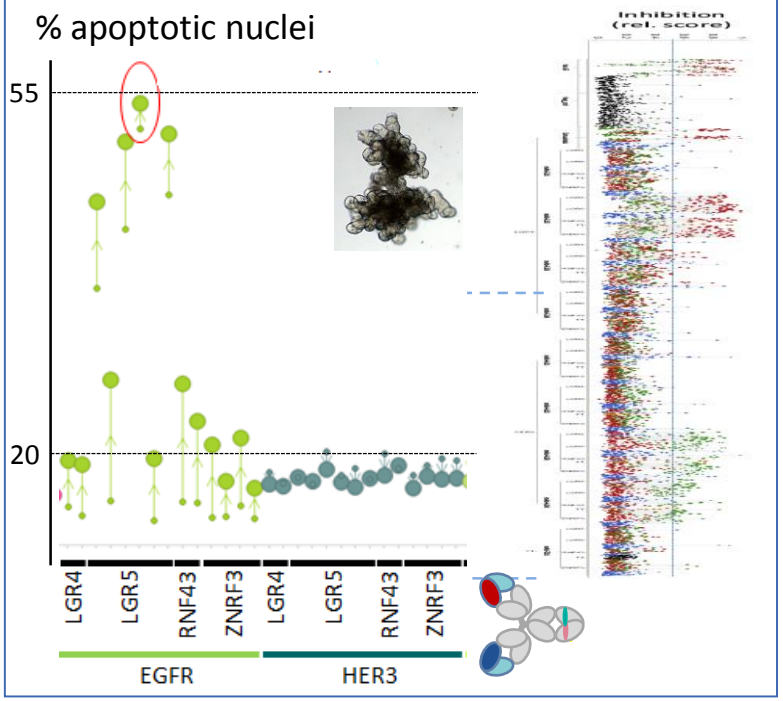
Functional Screening of 750 different EGFRxHER3 Biclonics®

Growth of a tumor cell line



superior growth inhibition in the absence (■) and presence (■) of heregulin

Functional screening of WNT x HER Biclonics®



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MCLA-128: HER2 X HER3 BICLONICS®

MCLA-128: HER2xHER3 BICLONICS®

Medical need/ positioning	**Tumor cells become resistant to HER2-targeted therapies by HER3 activation** **A HER3-targeting bispecific antibody that can be used as single agent or in combinations with HER-targeting biologics (e.g. Herceptin, Kadcyla) or small molecule drugs, or chemotherapy**
Mode of action	HER3 is difficult to target by conventional mAbs 'Dock and Block' mode of action efficiently inhibits signaling via the HER2:HER3 heterodimer Enhanced ADCC for immune effector cell recruitment
Indications	Metastatic cancers of breast, ovary/endometrium and stomach Other solid tumors including non small cell lung cancer and metastatic colon cancer
Target Population	HER2+: breast, gastric, and lung cancer Unselected: ovarian and endometrial cancer • (Un)published, data suggests that the presence of HER2:HER3 heterodimers confers resistance to chemotherapy, independent of HER2 overexpression

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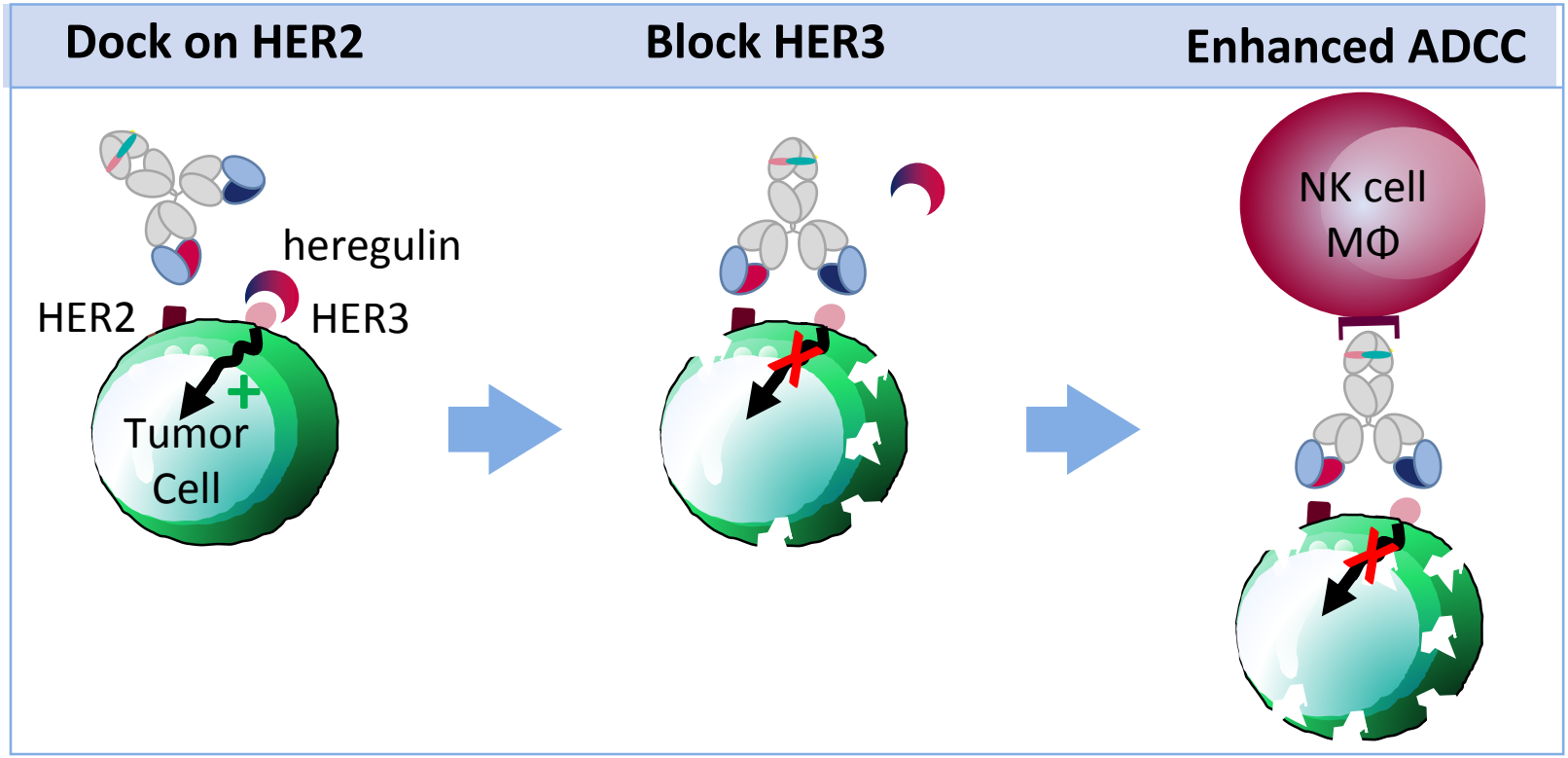
MCLA-128: 'DOCK AND BLOCK' MODE OF ACTION

- Docking of the HER2 arm on HER2 expressed by tumor cells optimally positions the HER3 arm to block heregulin-driven tumor cell growth
- Low fucosylation of the Fc region for enhanced ADCC



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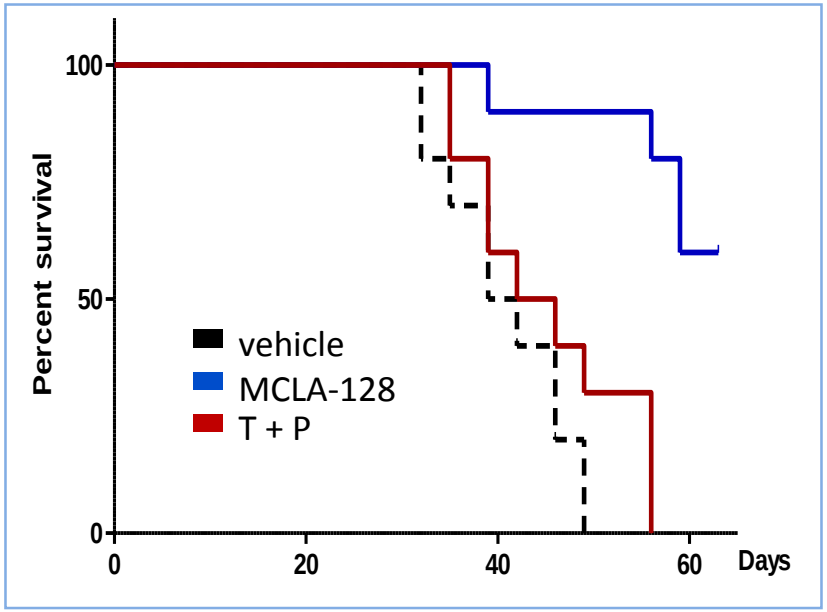
MCLA-128 INHIBITS HER3-DRIVEN GROWTH

Key preclinical findings ***(in vitro)***	More effectively inhibits heregulin-driven cancer cell growth than: • HER3 mAbs, trastuzumab and trastuzumab + pertuzumab • Combinations of Herceptin and HER3 mAbs
(in vivo)	Compared to trastuzumab (T) + pertuzumab (P), MCLA-128 is more effective at inhibiting the growth of cell lines resistant to HER2-targeted therapies



Mice xenografted with cell line JIMT-1(resistant to T + P) show prolonged survival upon treatment with MCLA-128

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SINGLE AGENT MCLA-128 PHASE I/II STUDY

Part 1
 	
Dose Escalation
	• 28 patients with solid tumors; all comers
	• Very good safety profile
	  ◦ No dose-limiting toxicities
	  ◦ Most drug-related AEs were mild to moderate (G1/G2)
	• Recommended dose: 750 mg flat, IV infusion in 2h, q3ws
	• Evidence of activity in heavily pre-treated patients including progressors to chemotherapy/HER2-directed therapies

Part 2
 	
Expansion Cohorts
	• Actively enrolling in indications of interest:
	  ◦ Metastatic breast cancer (20 patients)
	  ▪ Progressed on two HER2-directed therapies and chemotherapy
	  ◦ Ovarian cancer (20 patients) and endometrial (20 patients)
	  ▪ Progressed to platinum-based therapies
	  ◦ Metastatic gastric cancer (20 patients)
	  ▪ Progressed to trastuzumab/chemotherapy

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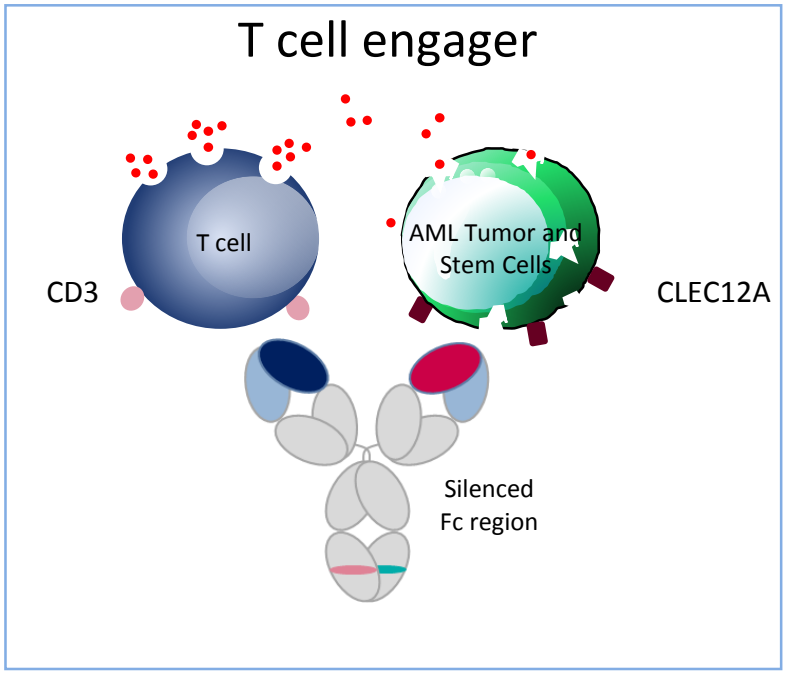
MCLA-117



MCLA-117: CD3xCLEC12A T CELL ENGAGER FOR AML

Medical Need and Positioning	• Increasing patient population with little progress in disease outcomes for last 4 decades; most patients succumb to AML • Induction or maintenance therapy • Minimal residual disease • Rescue therapy for Refractory/Relapsed patients
Mode of Action	• Retargeting T cells via a first-in-class AML target with restricted expression in normal hematopoietic cells • Designed to selectively kill tumor cells/tumor stem cells
Indications	• AML patients with relapse or refractory disease • Newly diagnosed, untreated AML patients > 65 years who are not candidates for the standard intensive AML therapy
Status	• A first-in-human clinical trial was initiated in May 2016

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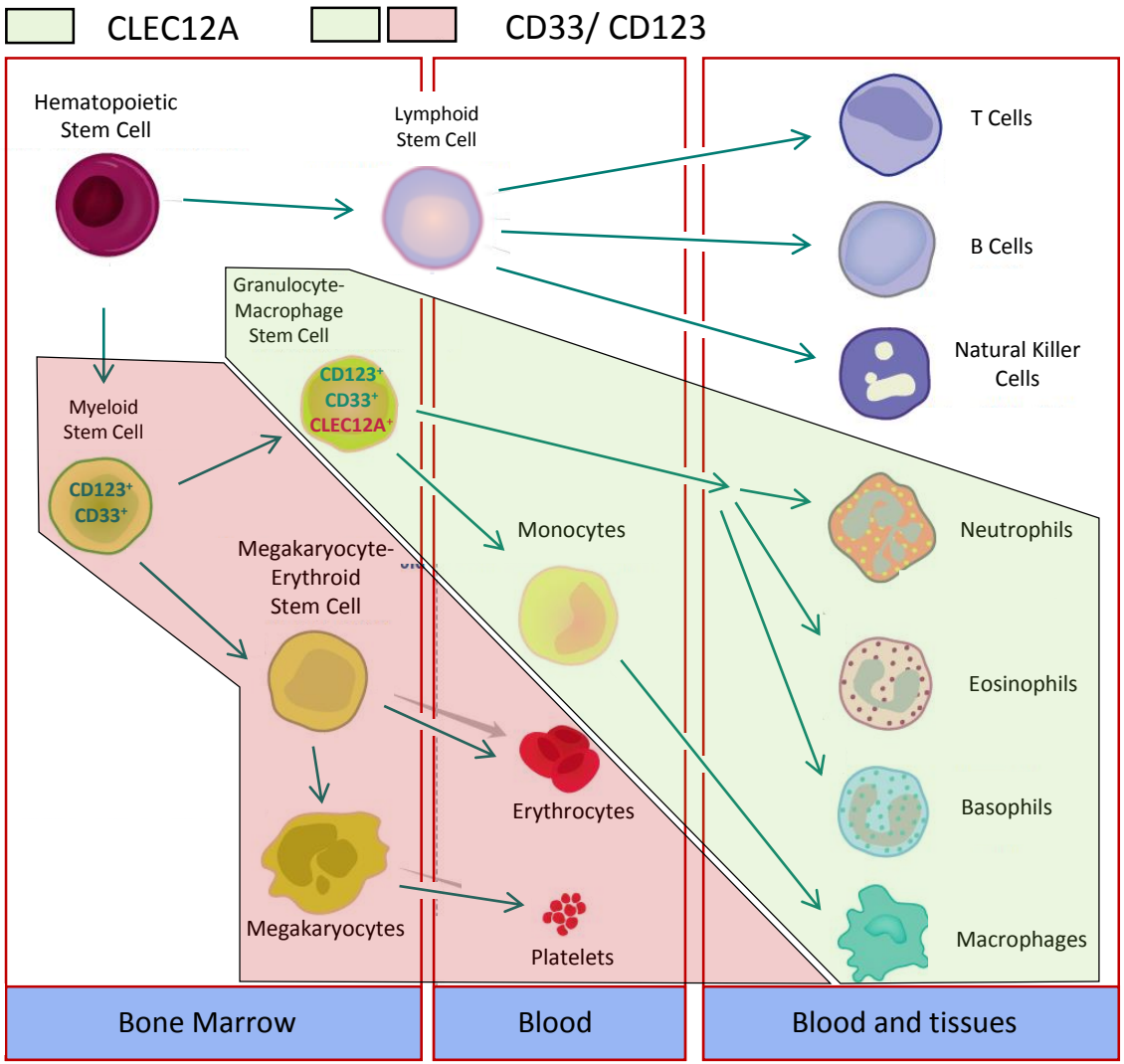
CLEC12A: RESTRICTED TISSUE EXPRESSION



CLEC12A – normal tissues

- Not expressed on normal tissues outside of the hematopoietic system
- Only on certain myeloid cells of the hematopoietic system
- More restricted expression than CD33 and CD123

CLEC12A - tumor tissues

- Expressed by tumor cells of ~90-95% of patients with AML (> 85% of patients with MDS)
- Expressed by AML tumor stem cells

Expression

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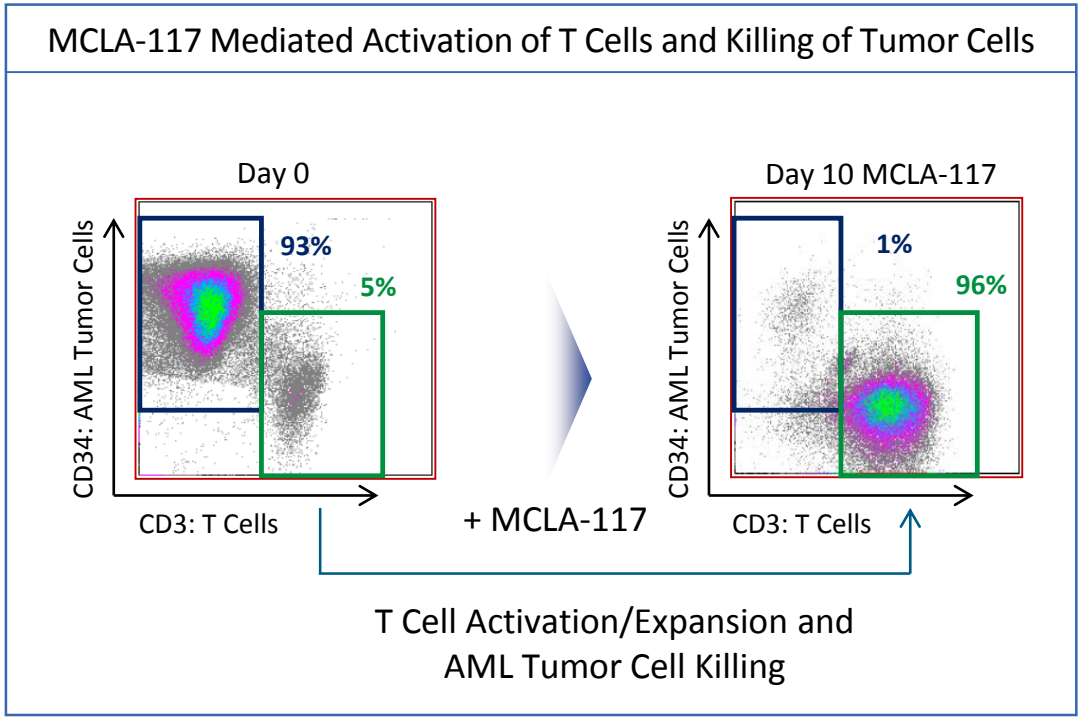
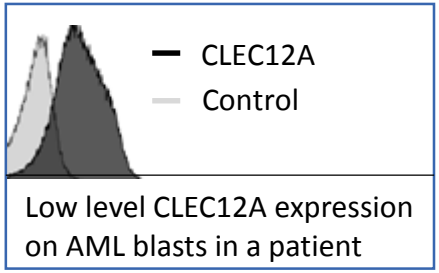
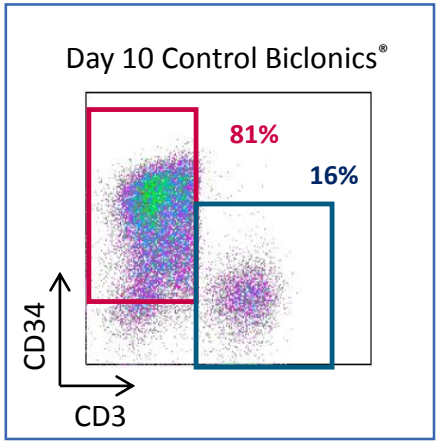
MCLA-117: *EX VIVO* EFFICACY IN AML SAMPLES

- MCLA-117 causes:
  - Specific and efficient lysis of large numbers of tumor cells by low numbers of T cells present in blood samples from AML patients
  - T cell expansion







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MCLA-117: PHASE I/2 TRIAL BUILDUP

Part 1: Dose escalation

	Cohort (# pts)	Dose (mg) flat	Date
AML all subtypes Treatment naïve >65 & Rel/Ref >18	1 (2)	Intra-patient	Q2 ‘16
	2 (2)		
	3 (3)*		
	4 (3)		Q1 ‘17
	5 (3)	Dose escalation	
	6 (3)		
	7 (3,6)		
	8 (3,6)	Inter-cohort	
	9 (3,6)		Q4 ‘17

Safety, recommended dose

* Cohort #4 initiated Jan 2017

RP2D

Part 2: Expansion

#Pts	Cycles	Date
5	2	
10	2	
15	2	Q2 ‘18

- PK
- T cell activation
- Immunophenotype
- PD-L1/TIM3 expression
- Cytogenetics

Safety, preliminary efficacy at recommended dose

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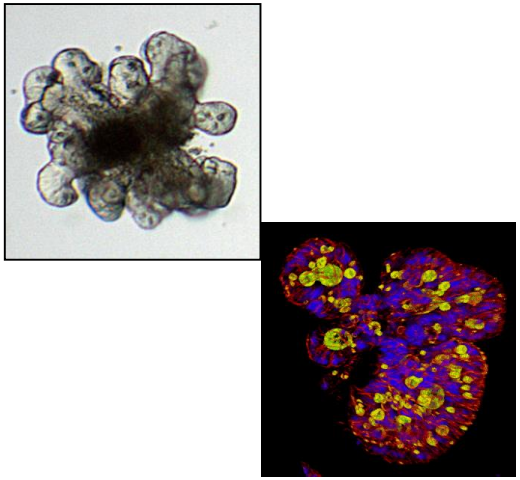
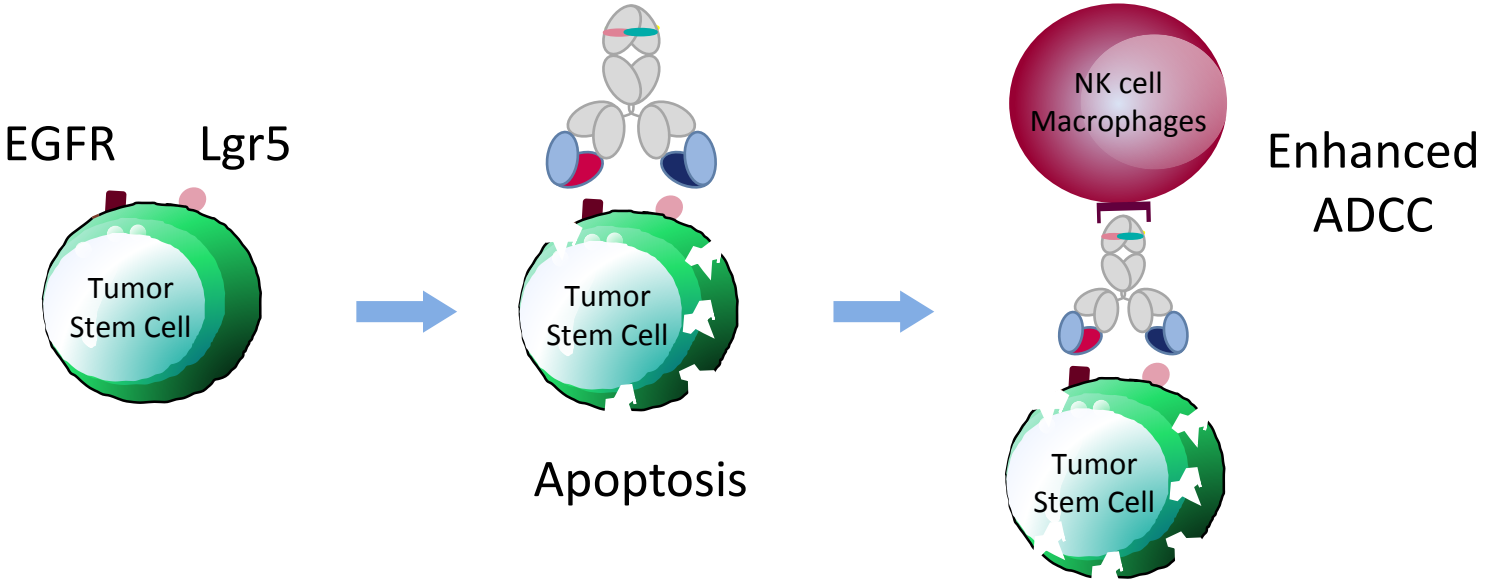


MCLA-158

MCLA-158: EGFR X Lgr5 BICLONICS®

Unmet Medical Need	• Designed to eliminate cancer stem cells that persist in various solid tumors and cause relapse and metastasis
Differentiated Mode of Action	• Targets the Wnt pathway receptor Lgr5 and EGFR • Induces apoptosis in cancer stem cells • Enhanced ADCC for immune effector cell recruitment



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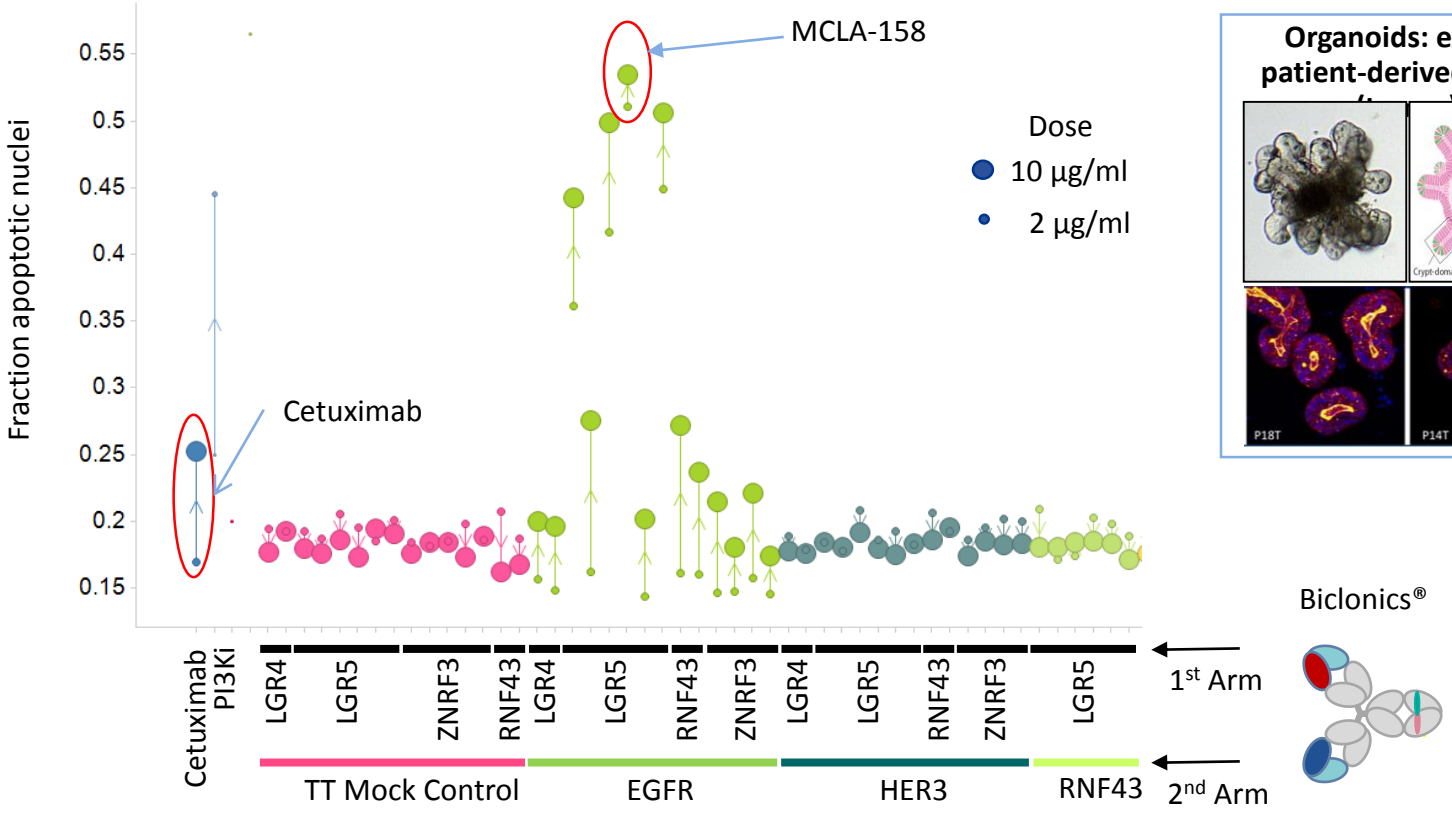
MCLA-158 INDUCES APOPTOSIS IN CRC ORGANOIDS

- In preclinical studies MCLA-158 has been shown to
  - be a potent inducer of apoptosis in organoids derived from patients with colorectal cancer
  - effectively target and kills cancer stem cells



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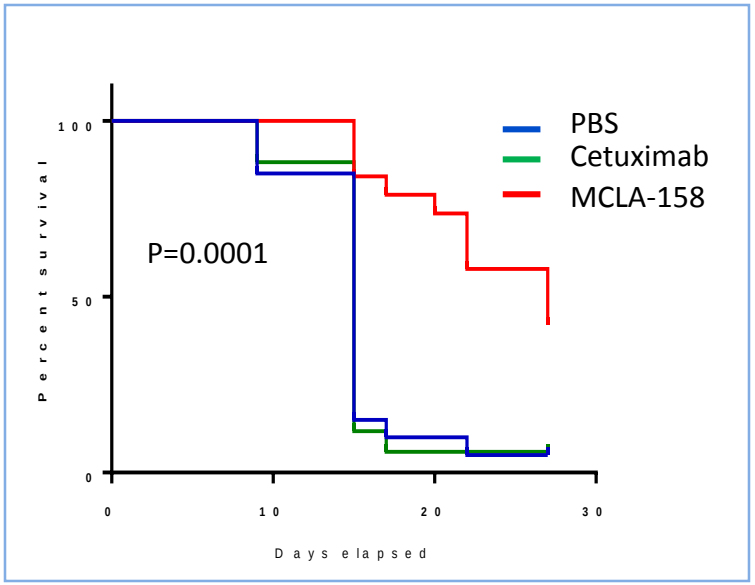
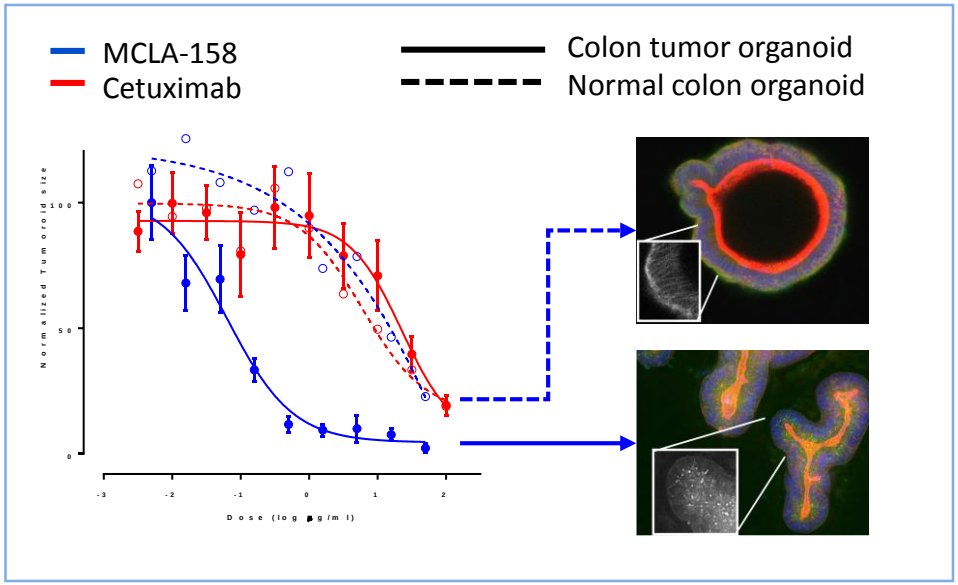
MCLA-158 INHIBITS ORGANOID GROWTH *IN VIVO*

- MCLA-158 is a novel stem cell targeting bispecific antibody.
  - More potent than EGFR targeting mAbs (e.g. Cetuximab)
  - Effectively eliminates tumor stem cells *in vitro* and *in vivo* ($KRAS^{WT}$ sand $KRAS^{mut}$)





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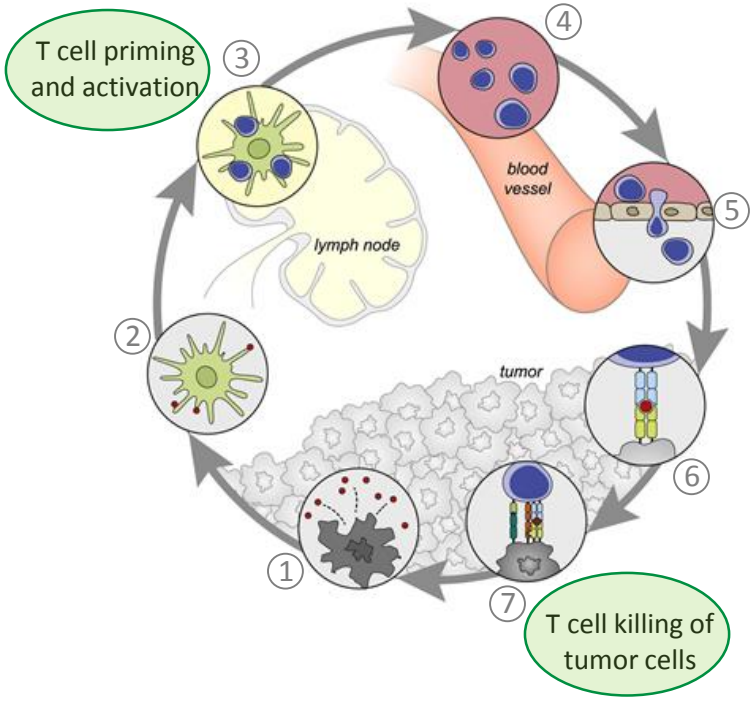
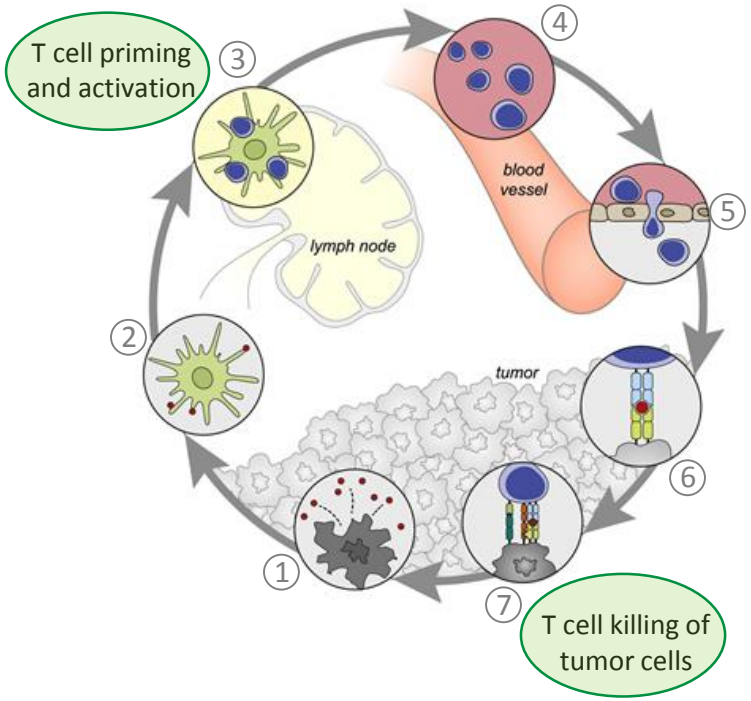
IMMUNO-ONCOLOGY



BICLONICS® FOR IMMUNO-MODULATION - iMODS

- Merus develops Biclonics® that bind to combinations of:
  - Co-inhibitory targets
  - Costimulatory and co-inhibitory targets
  - Tumor-associated antigens and co-stimulatory/co-inhibitory targets



- <u>Potential advantages</u>
  - More potent revival/ activation of tumor-specific T cells
  - Improved targeting to the tumor micro-environment
  - Lower risk of toxicity (autoimmune disease)
  - Attractive for combination therapies with additional compounds/vaccines

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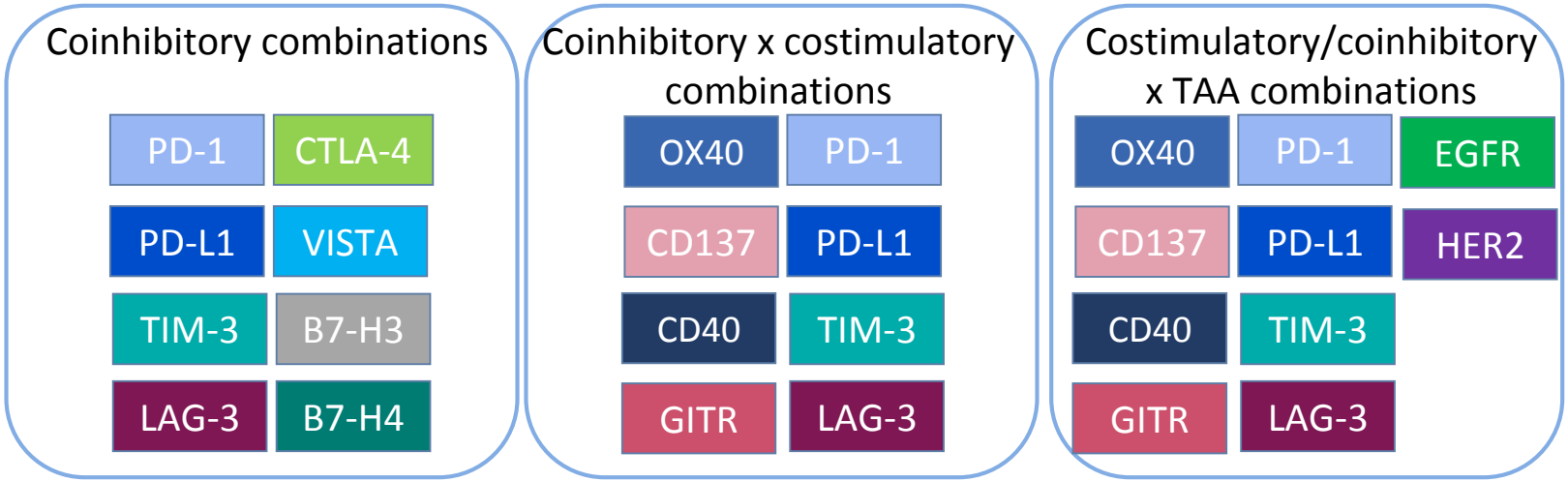
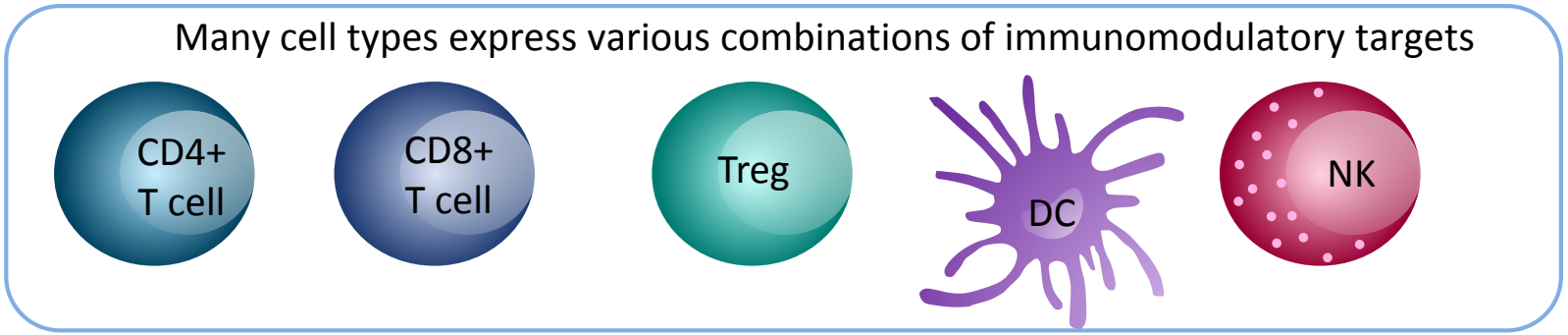
EMPIRICAL iMOD TARGET COMBINATION DISCOVERY

- Combine target panels with high diversity (affinity, epitope, sequence)
- Screen for functional activity *in vitro* using reporter assays, primary T cell and PBMC based assays





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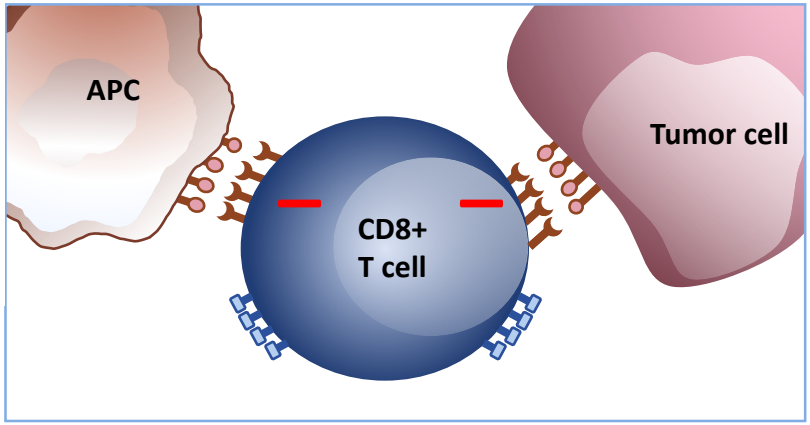
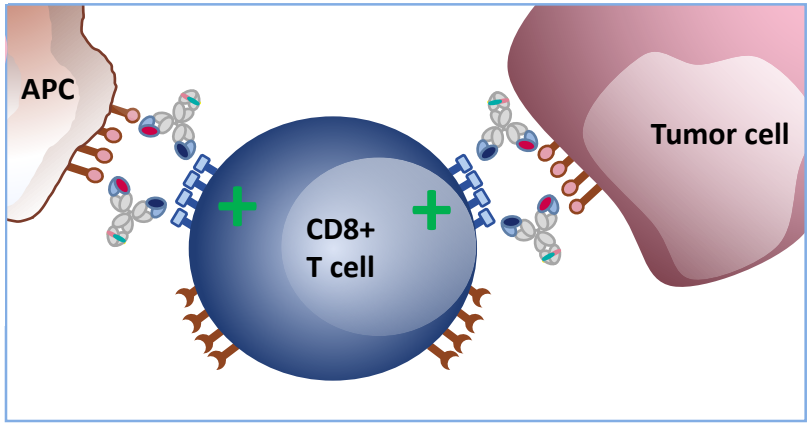
FUNCTIONAL CHARACTERISTICS OF LEAD BICLONICS®

- Functional characteristics of Lead Biclonics addressing coinhibitory x costimulatory Combinations
  - Costimulation of CTLs
  - Blockade of CTL suppression
  - Recruitment and activation of NK cells
  - Suppression of Treg cell generation
- Potential outcomes *in vivo*
  - Enhanced tumor immunity with memory response
  - Reduction in irAE's





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INSTITUTE FOR RESEARCH IN BIOMEDICINE

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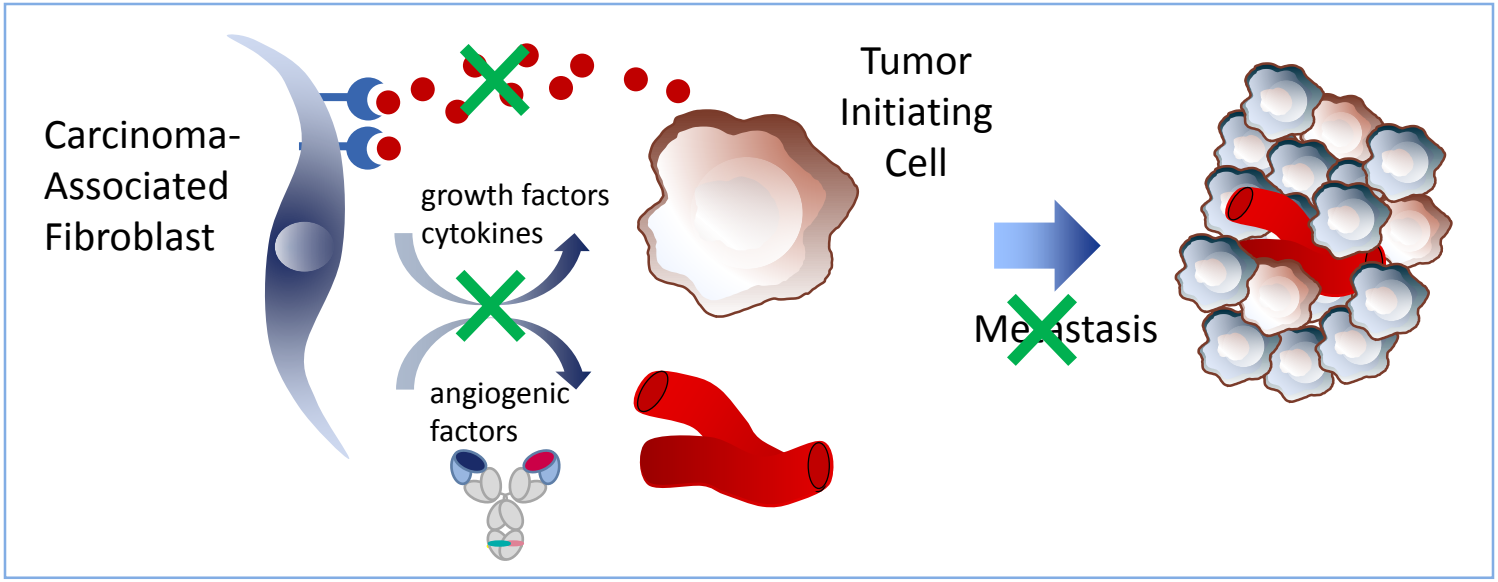
TUMOR MICROENVIRONMENT

TUMOR-STROMAL SIGNALLING CREATES METASTATIC NICHE

- The tumor stroma is an important modulator and driver of tumorigenicity ("architects of cancer pathogenesis").
- The stromal and epithelial components of a tissue act in an integrated, reciprocal fashion.
- Develop Biclonics that disrupt tumor-stromal signalling loops => block formation of metastatic lesions



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NEAR-TERM MILESTONES

2017	Interim results from Part 2 of Phase 1/2 trial of MCLA-128 in mBC	**1Q**
	Lead identification of the first Biclonics® for immuno-modulation	**1Q**
	IND filing planned for Phase 1/2 clinical trial of MCLA-117	**2Q**
	Interim results expected from Part 1 of Phase 1 clinical trial of MCLA-117 in AML	**2H**
	Topline results expected from Part 2 of Phase 1/2 trial of MCLA-128 in multiple indications	**2H**
	IND filing planned for Phase first-in-human clinical trial of MCLA-158	**4Q**
2018	Topline results expected from Part 1 of Phase 1 clinical trial of MCLA-117	**1Q**
	Topline results from Part 2 of Phase 1 clinical trial of MCLA-117 in AML expected	**1H**

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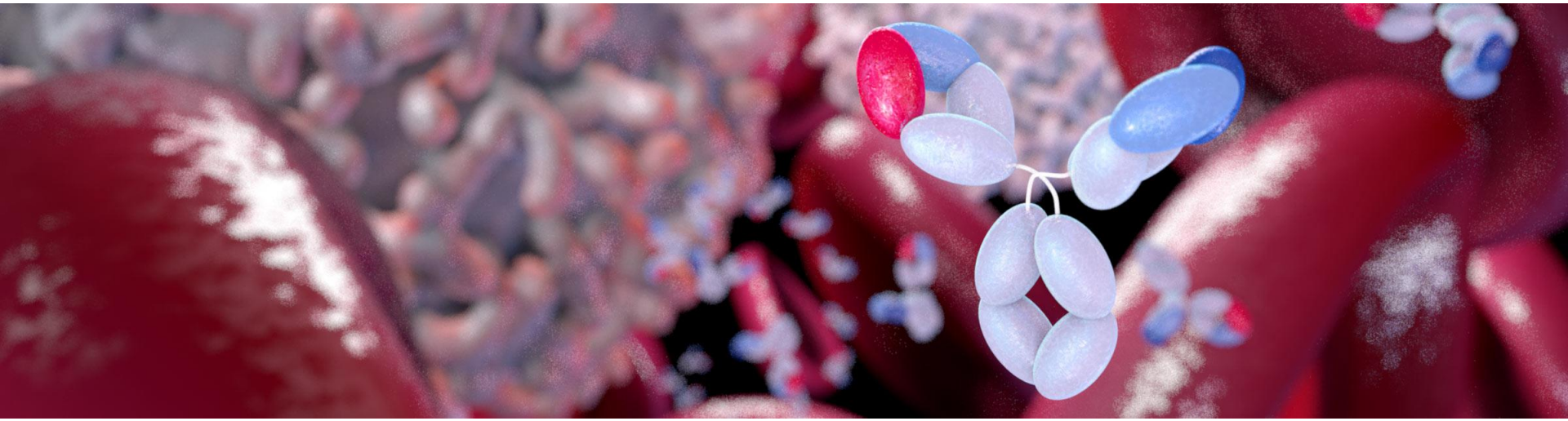

Battling Cancer with Bispecific Antibodies

March 2017