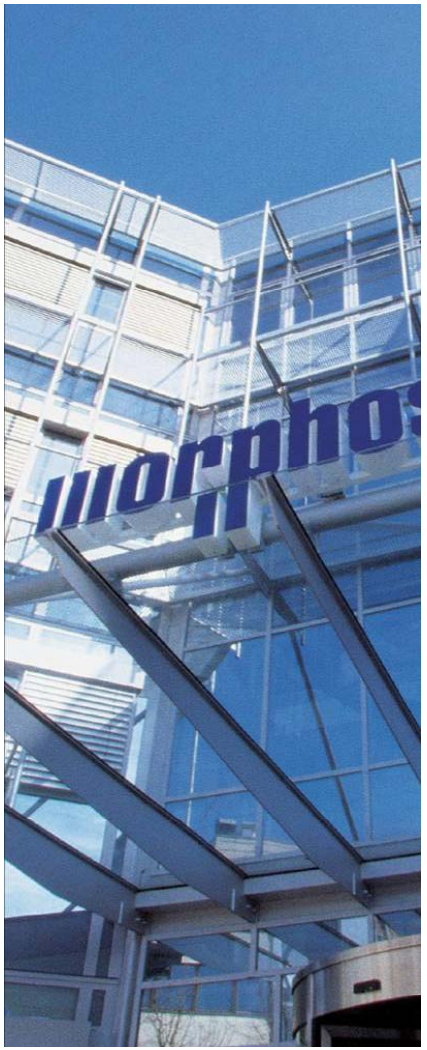
R&D Day 2010

morphosys

London – November 25, 2010

Agenda

09:30	**Simon Moroney**	**Welcome and Introduction**
09:35	Marlies Sproll	Latest Technology Developments
		Update Partnered Pipeline
10:05	Q&A	
10:20	Arndt Schottelius	Introduction Proprietary Portfolio
10:25	John Hamilton	GM-CSF – Central Mediator in Inflammation
10:55	Arndt Schottelius	MOR103 in RA & MS
11:10	Q&A	
11:25	Lisa Rojkjaer	MOR202 in Multiple Myeloma (MM)
		MOR208 in Chronic Lymphocytic Leukemia (CLL)
11:55	Q&A	
12:15	Simon Moroney	Closing Remarks



Today's Presenters

	Simon Moroney Chief Executive Officer MorphoSys AG	▪ Co-founder of MorphoSys ▪ DPhil in chemistry ▪ Scientific positions at ImmunoGen Inc., Harvard Medical School/USA, Universities in Switzerland, UK & Canada
	Marlies Sproll Chief Scientific Officer MorphoSys AG	▪ PhD in biology ▪ Science and management positions in R&D of biopharmaceuticals at Boehringer Ingelheim and Merck KGaA
	Arndt Schottelius Chief Development Officer MorphoSys AG	▪ MD, PhD ▪ Resident physician in gastroenterology ▪ Science and management positions in immunology research at Genentech Inc., Berlex Biosciences and Schering AG
	Lisa Rojkjaer Head of Clinical Development MorphoSys AG	▪ MD, board-certified hematologist ▪ Held academic and hospital appointments in the US & Europe ▪ Clinical development positions at Novo Nordisk, USA and Novartis Pharma AG, USA/Switzerland
	Prof. John Hamilton Director, Arthritis & Inflammation Research Center University of Melbourne, Australia	▪ Pharma/Biotech consultant, author of numerous publications ▪ Current research focus: Control of the development and function of macrophage lineage populations in inflammatory diseases, resulting in several clinical trials for RA ▪ ACR Distinguished Basic Investigator Award in 2002

Safe Harbour

This presentation includes forward-looking statements.

Actual results could differ materially from those included in the forward-looking statements due to various risk factors and uncertainties including changes in business, economic competitive conditions, regulatory reforms, foreign exchange rate fluctuations and the availability of financing.

These and other risks and uncertainties are detailed in the Company's Annual Report.

MorphoSys has Grown into a Unique Antibody Discovery & Development Company

- A technology platform that continues to set new standards for antibody generation
- Deep antibody expertise spanning discovery & development
- Promising proprietary product portfolio with best-in-class potential
- A strong partnership roster creating multiple future product opportunities
- Well financed and independent from capital markets as a source of financing

Strategy and How it Relates to Today's Agenda

PROPRIETARY DEVELOPMENT

- Carefully selected programs in cancer & inflammation
- …taken by MorphoSys to clinical proof-of-concept
- …promise a lucrative upside

PARTNERED DISCOVERY

- A growing number of projects against partners' targets
- …promises to result in multiple marketed drugs
- …delivering a strong flow of milestone and royalty payments

Innovative, Proprietary Antibody Technology & Know-how

- Fulfilling a growing demand for technologies which can deliver superior antibodies
- …more efficacious, against new target types, better producible
- …extending the therapeutic reach and performance of antibodies

Broadest Antibody Pipeline in the Industry: 76 Programs Ongoing

morphosys

Name	Partner/MOR	Target	Indication	Discovery	Preclinic	Phase 1	Phase 2	Phase 3	Market
MOR103	MOR	GM-CSF	RA						
BHQ880	Novartis	DKK-1	Cancer						
CNTO888	Centocor	MCP-1	Immunology/ Cancer						
n.d.	Novartis	n.d.	n.d.						
Gantenerumab	Roche	Amyloid-β	AD						
CNTO1959	Centocor	n.d.	Psoriasis						
BAY79-4620	Bayer Schering	CA IX (MN)	Cancer						
CNTO3157	Centocor	n.d.	Asthma						
n.d.	Novartis	n.d.	Musculoskeletal						
n.d.	Novartis	n.d.	Ophthalmology						
MOR208 (XmAb5574)	MOR	CD19	CLL						
MOR202	MOR	CD38	Multiple Myeloma						
24 Partnered Programs	Partners	Various	Various*						
32 Partnered Programs	Partners	Various	Various*						
2 Programs	MOR	n.d.	Inflammation						
3 Programs	MOR	n.d.	Cancer						
2 Programs	MOR/NOV	n.d.	Inflammation						

* Includes cancer, inflammatory, autoimmune, infectious, musculoskeletal & central nervous system diseases

n.d. – not disclosed

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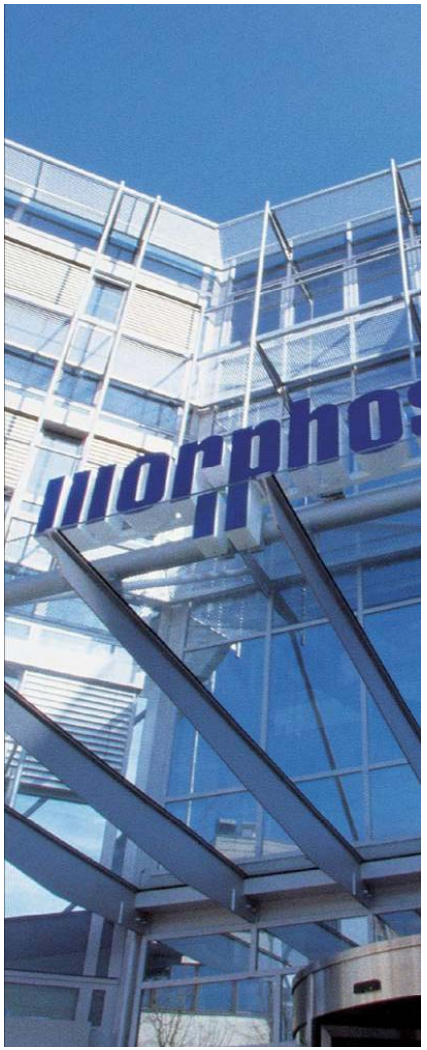
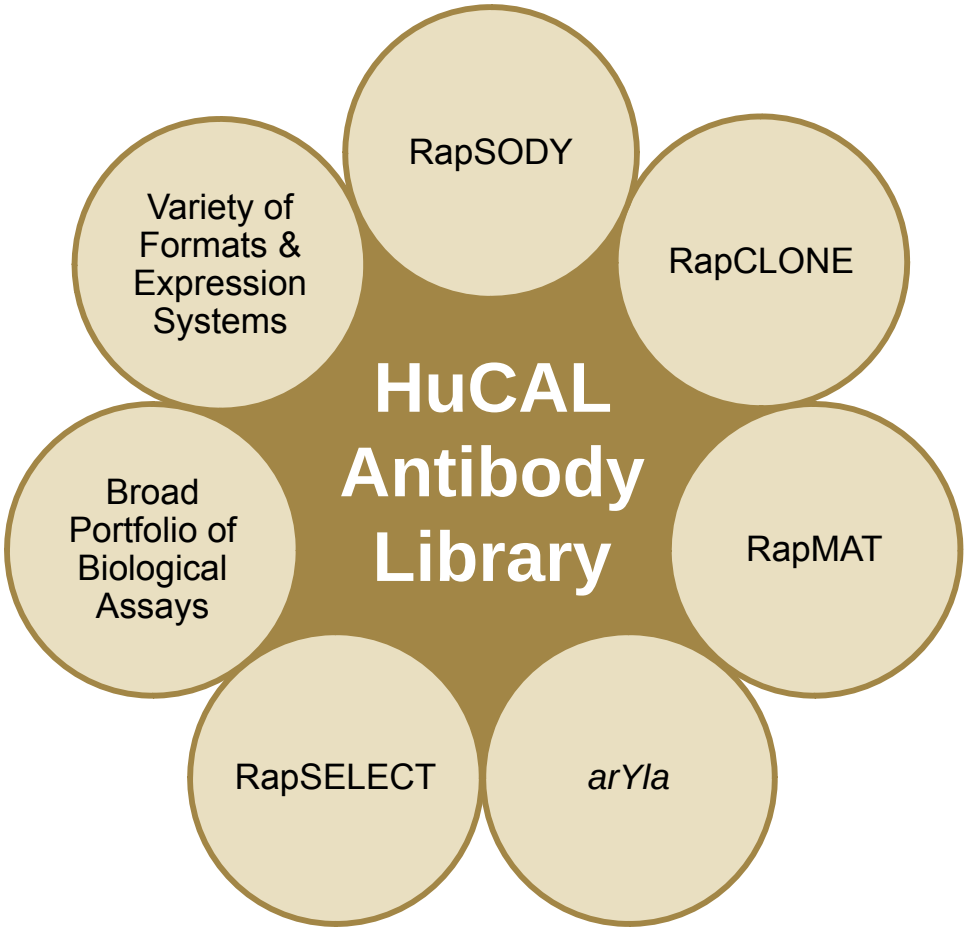


The MorphoSys Platform Delivers Highly Optimized Therapeutic Antibodies

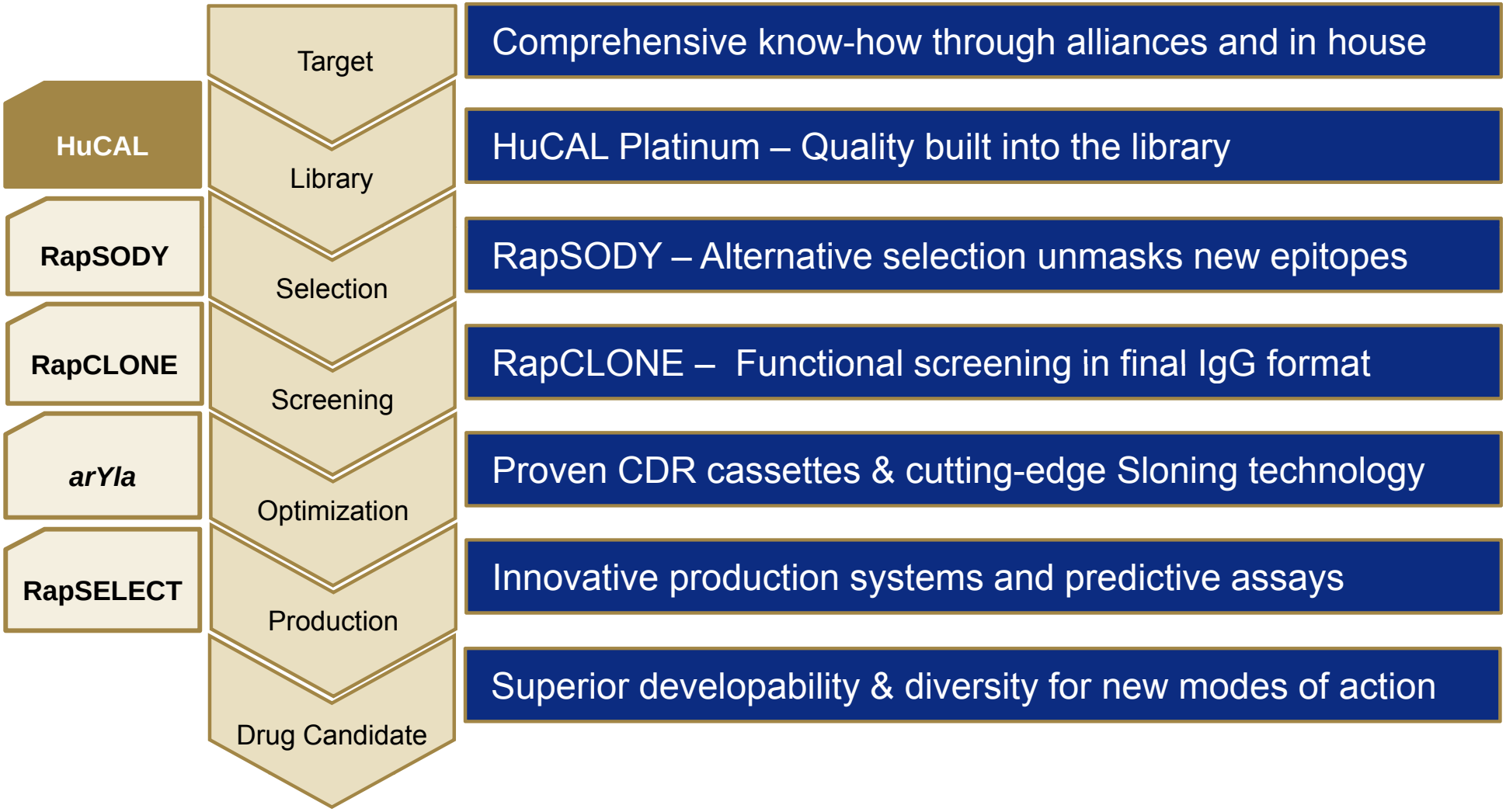


Basis for successful antibody drug development:

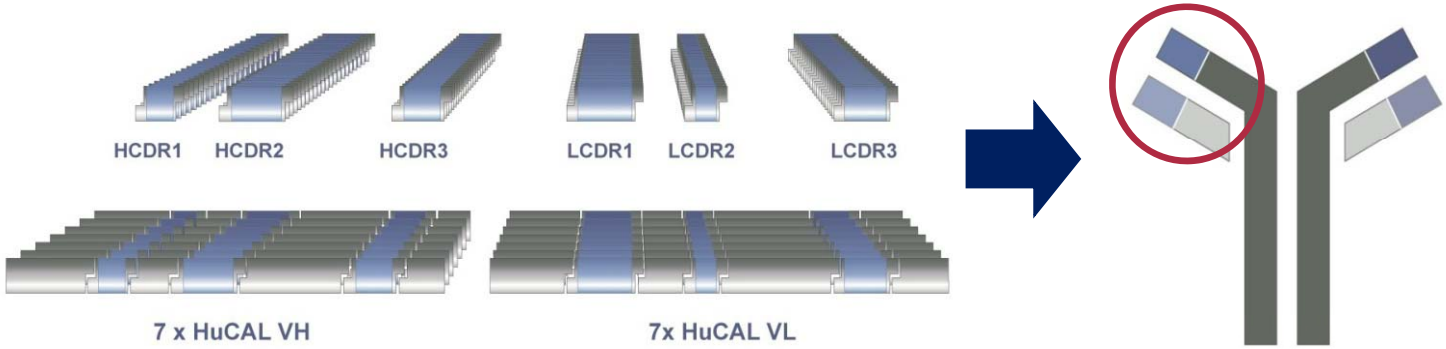
- Reliable human antibody source of high quality & diversity
- Robust high-throughput platform to enable tailored candidate selection, expression & functional profiling
- On-demand optimization technologies to engineer perfect drugs

Develop drugs, not just antibodies

Excellence in Every Step of Antibody Generation



Human Combinatorial Antibody Library HuCAL – An Industry Standard

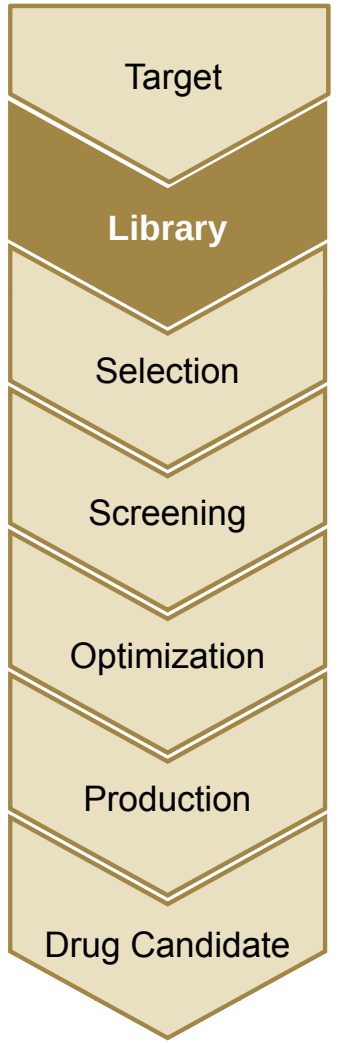


Modular gene design & construction

HuCAL PLATINUM

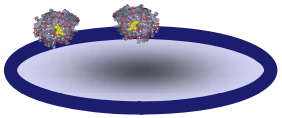
- 45 billion different human antibody Fab fragments
- Very high affinity
- High sequence diversity & structural diversity
- Favorable production characteristics

Quality of the antibody is already built into the library

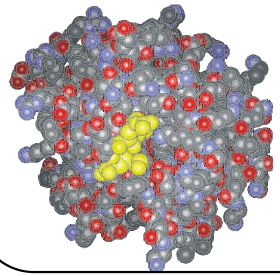


Phage Display Enables Accurate Tuning of Selection Against Various Antigen Classes

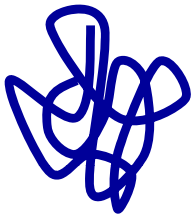
“Native” protein expressed on live cell surface



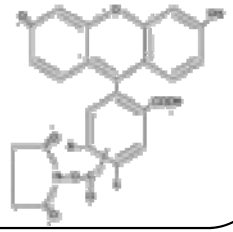
Purified protein



Peptide or protein domain



Hapten/Toxin



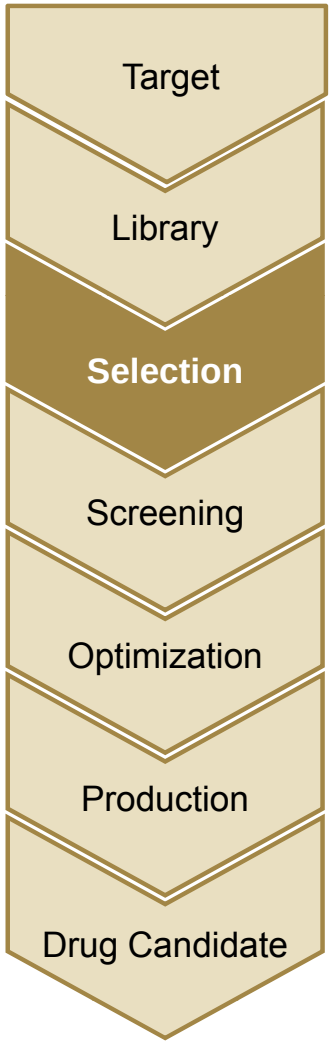
Whole cell panning

Solid phase or solution panning

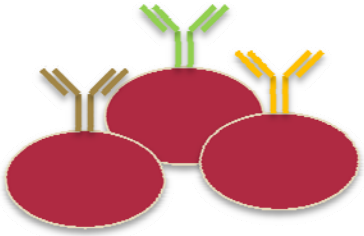
HuCAL is superior to *in vivo* methods through:

- Guided selection on whole live cells or pathogens
- Selection on toxic or non-immunogenic antigens
- Tailored selections on combinations of antigens to address unique functional or species / target cross-reactive epitopes
- Full control over selection process
- First binders with known sequence diversity within 8 weeks

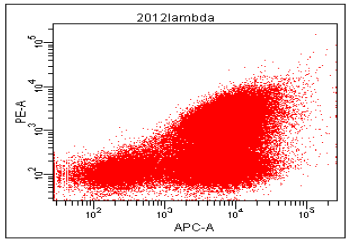
HuCAL delivers a vast diversity of binders, targeting different antigen classes and epitopes.



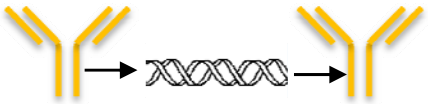
RapSODY: Direct Selection in IgG Format Unmasks Novel Epitopes



Reformating & Surface Display



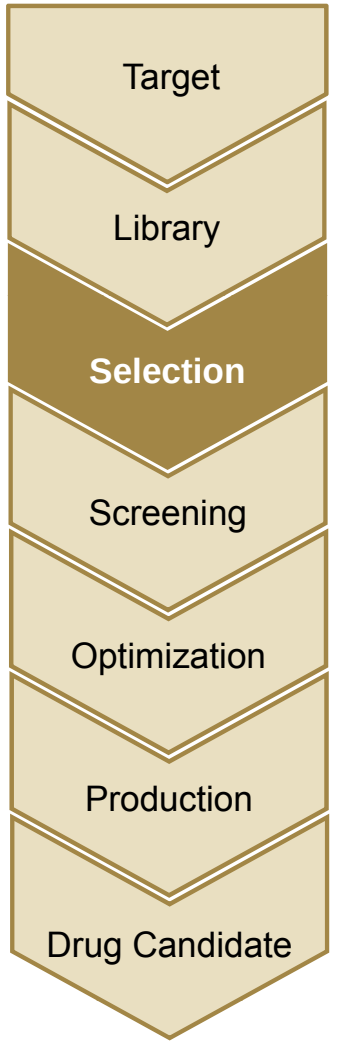
FACS-based Selection



Antibody Sequence Recovery

Rapid Selection of Displayed IgG's (RapSODY) on surface of mammalian cells allows:

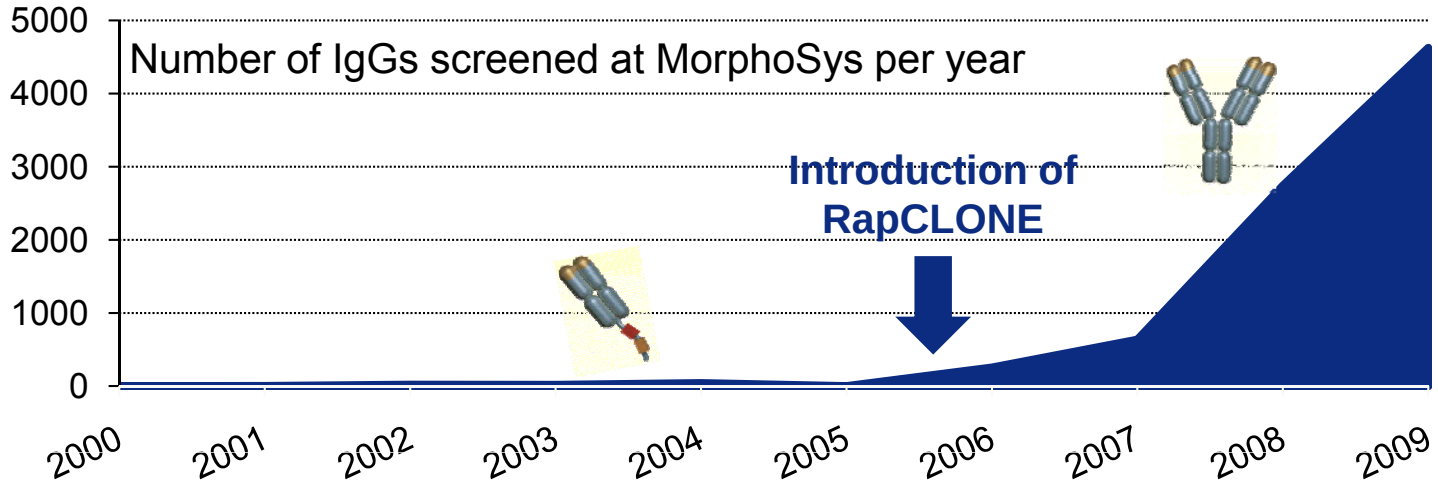
- FACS-based selection of large IgG libraries
- Presentation of antigen in natural cell-surface configuration
- Selection of IgGs showing simultaneously high expression and affinity to target
- Selection for additional and novel epitopes since alternative antibody presentation unmasks epitopes under-represented in phage display process
- Add-on to and fully compatible with modular HuCAL system



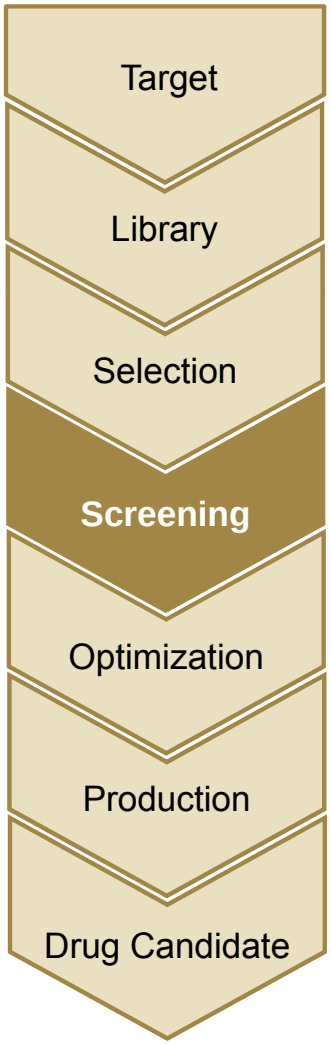
RapSODY facilitates direct selection in final IgG format and opens up novel epitope spaces

RapCLONE: High Throughput Conversion of Fab to IgG

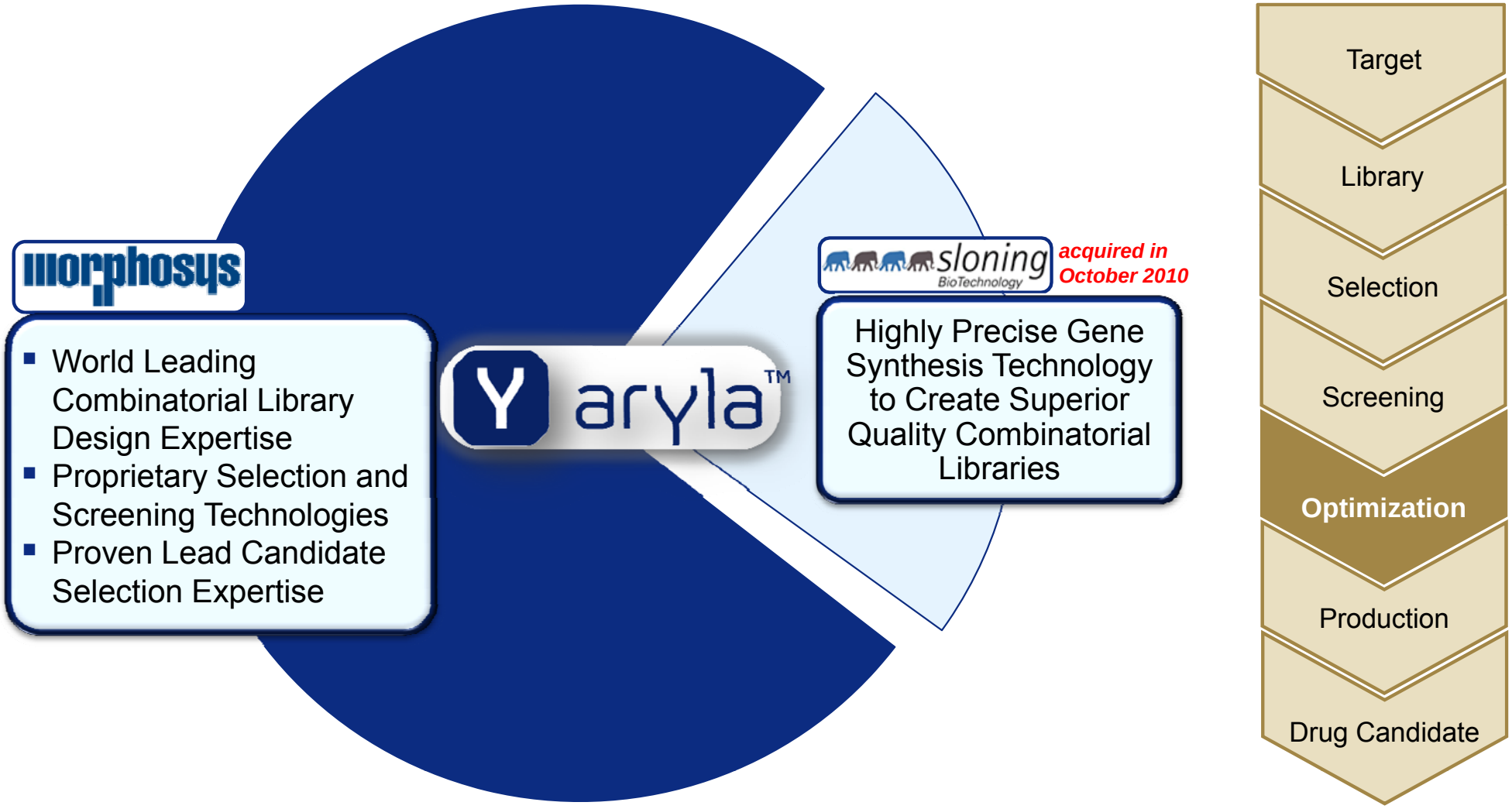
- MorphoSys starts with Fab fragments for efficient antibody characterization
- IgG is most usual drug format
- RapCLONE converts thousands of Fabs to IgG, in a single step, early in discovery



RapCLONE introduced a paradigm shift to high-throughput screening directly in the drug-relevant IgG format



Introducing aryla™ – A Combination of Unique Technologies & Capabilities



HuCAL – "One-Size-Fits-All" Optimization

HuCAL Antibody Candidate

Maturation Library

Pre-built Standard Optimization Library "one-fits-all"

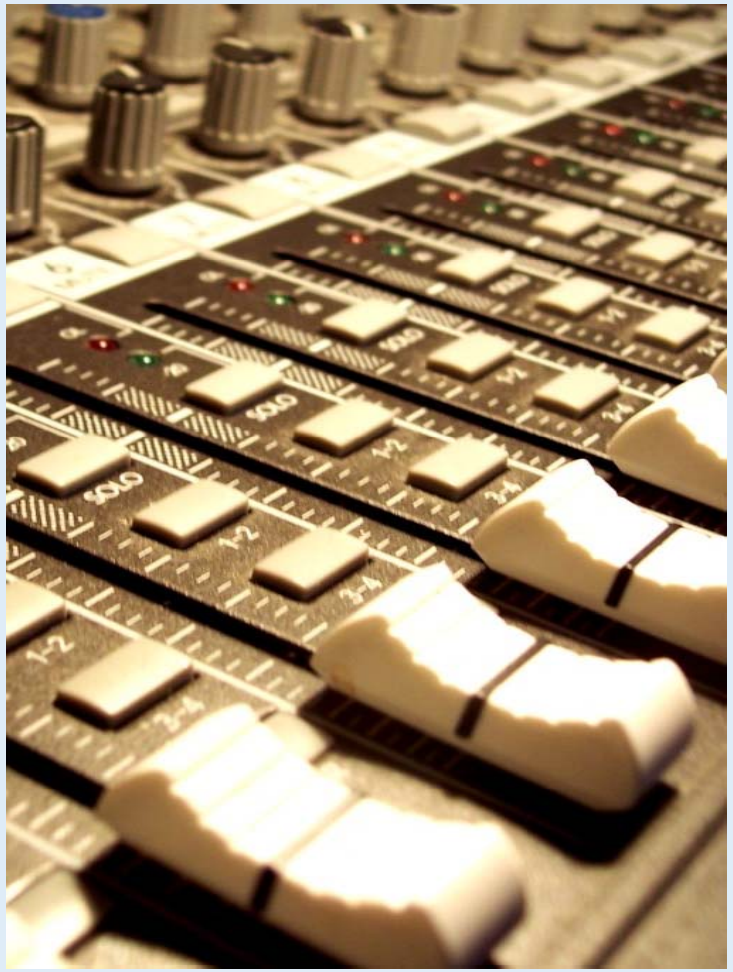
Select and Screen for **Affinity** only

Engineering stability

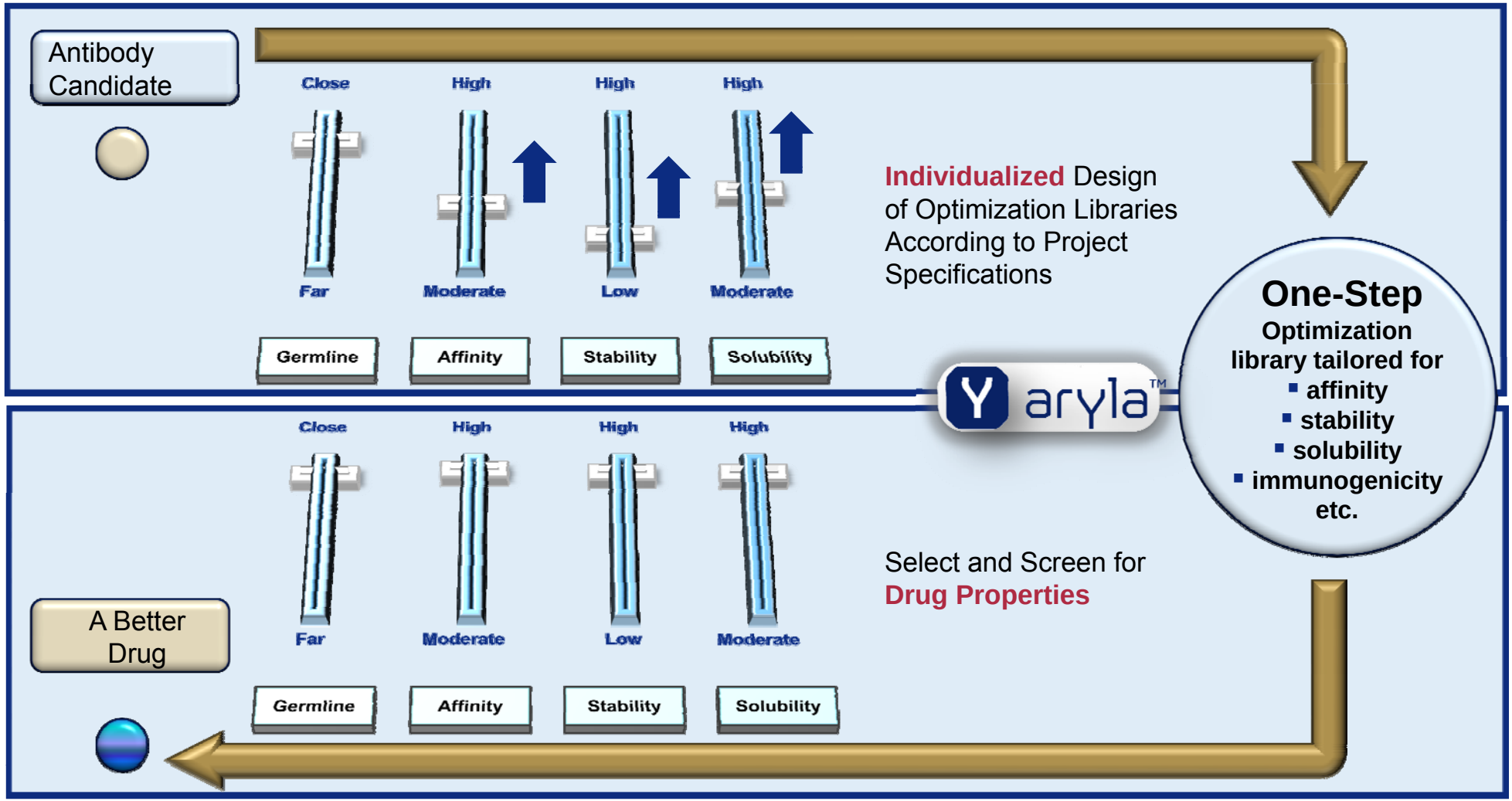
Germlining

HuCAL Drug Candidate

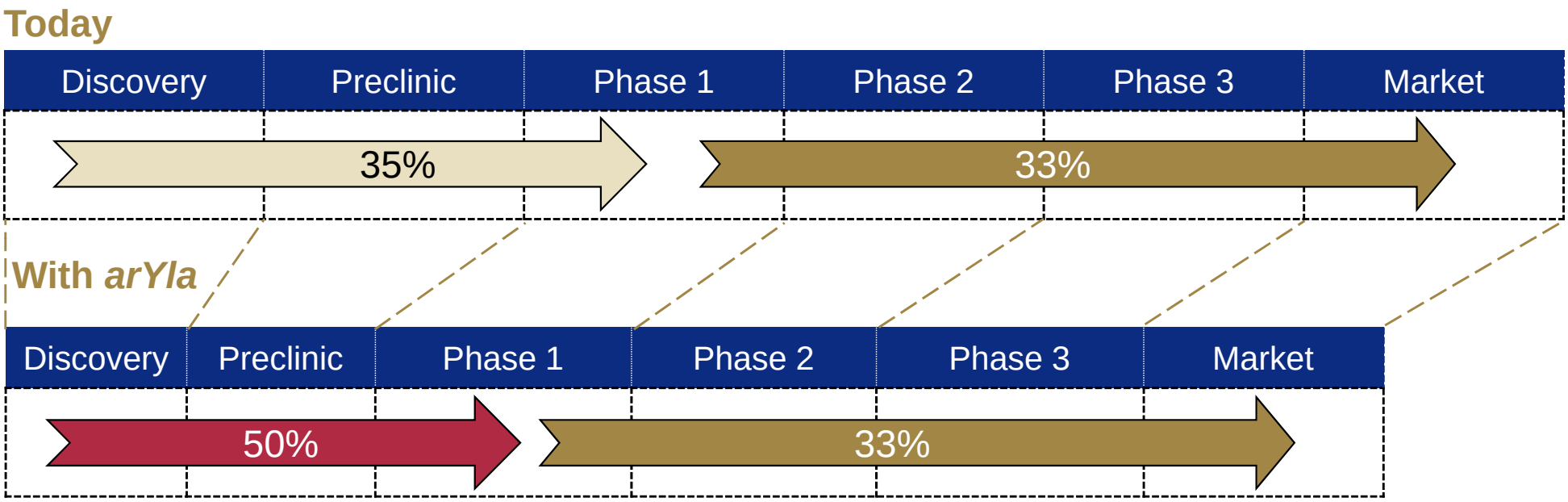
Turn Into



Beyond HuCAL -
The Individually Optimized Antibody Drug



aryla™ – Higher Probabilities of Success & Faster Development



Expectations:

- Shorten time to generate antibody drug candidate by 30%
- Increase proportion of programs reaching clinical development to 50%

Source: MorphoSys experience

Source: MorphoSys projections

Source: Tufts Centre for the Study of Drug Development*

*Metrics for antibody therapeutics development, Janice M. Reichert

Technological Progress
Exploiting New Opportunities

From Mouse to 100% Human Antibody

HuCAL GOLD & PLATINUM & Add-on Modules

Acquisition of Sloning BioTechnology

arYla Technolgy

MORe to come!

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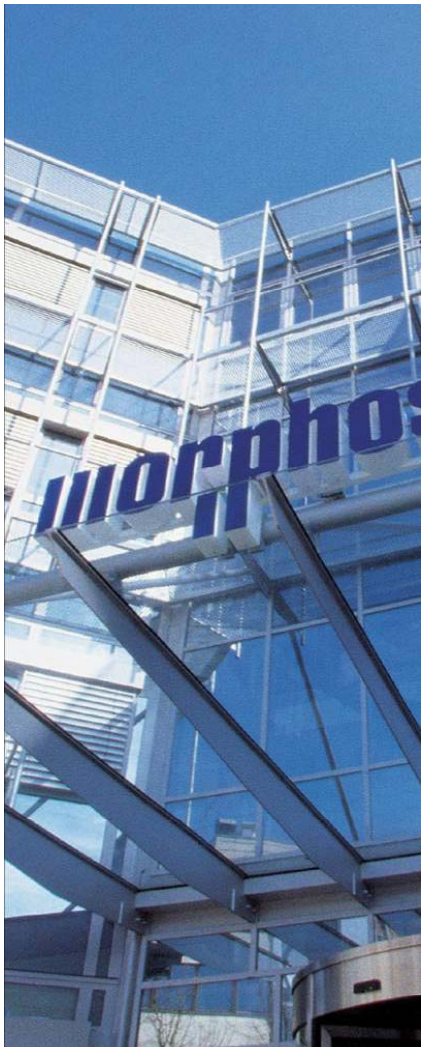

Novartis Alliance
One of the Largest Technology Alliances

NOVARTIS

Discovery	Pre-clinic	Phase 1	Phase 2
■ 20 discovery programs steady-state ■ ~75 employees (FTEs) at MorphoSys ■ Currently two joint pre-development programs	Currently ten active programs	Currently two active programs	Currently two active programs

- Novartis's biologics pipeline of antibodies is growing
- Number of published joint patent applications so far: 13
- Antibody development programs across various indications

Novartis Alliance: Landmark Deal

Timeline	■ May 2004: Initial deal, including equity stake ■ November 2007: Major expansion ■ November 2017: End, subject to 2-year extension option
Novartis pays…	■ Approx. €20m p.a. technology license including HuCAL internalization fees ■ Approx. €20m p.a. in research funding ■ Over €250m milestones (probability adjusted) ■ Royalties on all resulting drugs
Novartis gets…	■ Preferred access to HuCAL for use in over 100 discovery programs
Co-development option	■ Shared costs & profits (20% – 50%) on selected co-developed programs
Excluded	■ Most infectious disease targets

Increasing Number of Partnered Programs in Clinical Development

New in 2010

Name	Partner	Target	Indication	Discovery	Preclinic	Phase 1	Phase 2	Phase 3	Market
BHQ880	Novartis	DKK-1	Cancer						
CNTO888	Centocor	MCP-1	Oncology IPF						
n.d.	Novartis	n.d.	n.d.						
Gantenerumab	Roche	Amyloid-β	AD						
BAY79-4620	Bayer Schering	CA IX (MN)	Cancer						
CNTO1959	Centocor	n.d.	Psoriasis						
CNTO3157	Centocor	n.d.	Asthma						
n.d.	Novartis	n.d.	Musculoskeletal						
n.d.	Novartis	n.d.	Ophthalmology						
New									
New									
New									

PoC achieved

Phase 2 started

Up to 3 further INDs expected in 2010

BHQ880: DKK-1 Specific Antibody in Multiple Myeloma Associated Osteolysis

NOVARTIS

Target DKK-1

- Soluble factor, secreted by multiple myeloma cells, inhibits differentiation of osteoblasts (bone formation) and causes increase in bone resorption which weakens the bones
- Inhibition of DKK-1 is expected to release suppression of bone formation and to prevent the development of osteolytic disease

Antibody BHQ880

- HuCAL IgG1 antibody, blocks DKK-1 binding to LRP5/6
- High specificity and affinity for human and mouse DKK-1

Active Clinical Trial

- **Phase 1/2** (USA and Australia): BHQ880 in combination with standard chemotherapy and zoledronic acid in relapsed or refractory myeloma patients
- Assessment of maximum-tolerated dose (MTD) and dose limiting toxicity (DLT) of escalating doses of BHQ880 (up to a maximum dose of 20 mg/kg); 9 months minimum treatment period
- Study closed to enrollment, treatment and bone marker follow up continuing. Early results presented at ASH 2009.
- Initiation of Phase 2 studies in multiple myeloma patients with renal insufficiency in combination with standard chemotherapy (without bisphosphonates) and as single agent in high risk smoldering myeloma is scheduled for Q1 2011

CNTO888: MCP-1 Specific Antibody in Cancer and Idiopathic Pulmonary Fibrosis

Target MCP-1

- Monocyte chemoattractant protein 1 (or CCL2)
- Produced by cancer cells as well as the tumor and bone micro-environment
- Role in fibroblast activation

Antibody CNTO888

- HuCAL IgG1 antibody targeting human MCP-1 with high specificity and affinity (~22pM)
- Inhibition of tumor growth in multiple models

Completed Clinical Trials

- **Phase 1:** Study of the safety and efficacy of single agent CNTO888 in patients with solid tumors

Active Clinical Trials

- **Phase 1b**: Study of the safety and efficacy of CNTO888 in combination with Standard of Care chemotherapy in patients with solid tumors
- **Phase 2**: Study of the safety and efficacy of single-agent CNTO888 in patients with metastatic castrate resistant prostate cancer
- **Phase 2** (USA, Canada, Belgium, Germany, Netherlands)**:** Study to evaluate the safety and effectiveness of CNTO888 administered i.v. in subjects with Idiopathic Pulmonary Fibrosis (IPF)
  - Patients will be treated for up to 48 weeks (and followed by 72 weeks)
  - Estimated study completion date: June 2012 (Estimated enrollment: 120)

Gantenerumab: Amyloid Plaque Specific Antibody for Alzheimer's Disease

Target Amyloid β

- Aβ peptide appears to be the main constituent of amyloid plaques deposited in the brains of Alzheimer's diseases patients
- Inhibition of Aβ production and aggregation or increased Aβ-clearance might result in disease-modifying treatments of AD

Antibody Gantenerumab

- Human IgG, high affinity (sub-nM) and specificity, targeting aggregated Aβ
- Chronic treatment in transgenic mouse model led to a significant reduction in amyloid load

Active Clinical Trial

- **Phase 2:** Multi-center, randomized, double-blind, placebo-controlled parallel-group study
- Evaluate the effect of Gantenerumab on cognition and functioning and the safety and pharmacokinetics in patients with prodromal Alzheimer's Disease
- Subcutaneous doses every 4 weeks for 104 weeks (3 groups: placebo, 105mg or 225mg)
- Study completion date: May 2015 (Estimated Enrollment: 360)

Pre-clinical and Clinical Data

- Two Phase 1 studies in AD patients completed
- Pre-clinical data presented at ICAD meeting in July 2010

BAY 79-4620: Anti-CA IX Immunoconjugate in Solid Tumors

Bayer HealthCare
Bayer Schering Pharma

Target CA IX (carbonic anhydrase IX; MN)

- Overexpressed in a wide range of human solid tumors, such as gastric cancer, non-small cell lung cancer, pancreatic cancer, and colorectal cancer
- Highest expression in areas of tumor hypoxia
- Expression is associated with increased malignancy and is predictive of prognosis in several cancers

Antibody Drug Conjugate BAY 79-4620

- A novel antibody-drug conjugate (ADC) targeting CA IX-positive tumors
- HuCAL IgG antibody coupled to cytotoxic MMAE (monomethyl auristatin E, Seattle Genetics)
- Conjugate potently inhibits proliferation and induces cell death of human CA IX-expressing tumor cells by inhibition of tubulin polymerization (EC50 low nM)

Clinical Trial Design

- Two **phase 1** studies initiated (Europe/USA)
- Determination of the safety and tolerability of the drug, pharmacokinetics profile of BAY79-4620 and its key metabolites, and the maximum tolerated dose (MTD)
- Estimated Study Completion Date: January 2012 (USA)/February 2013 (Europe); Estimated enrollment: 55 (USA)/55 (Europe)

Pre-clinical Data

- Pre-clinical data were presented at AACR in April 2010

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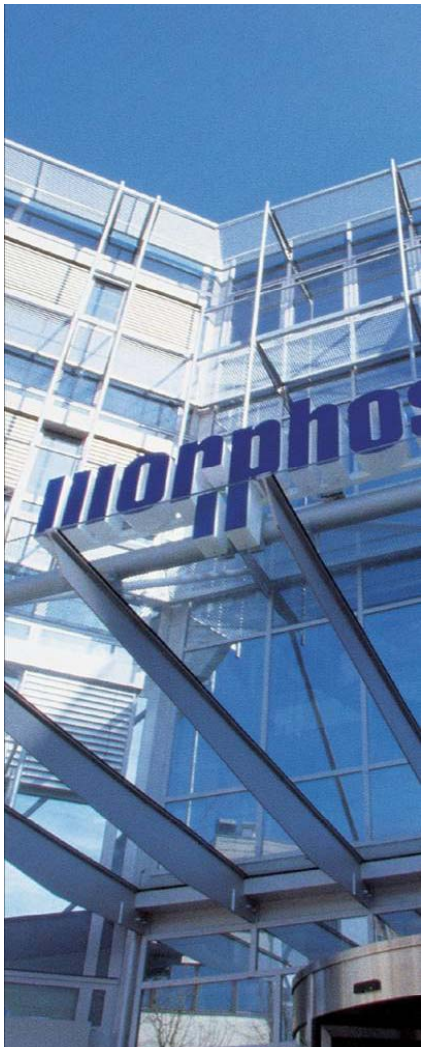
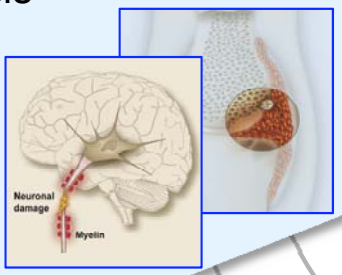
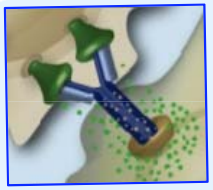


Focus of Proprietary Portfolio in Inflammation and Oncology

Inflammation

- Rheumatoid Arthritis/ Multiple Sclerosis
  - MOR103
- Bone and joint diseases
  - Galapagos collaboration





Oncology

- Hematological diseases
  - Myeloma (MOR202)
  - Lymphoma (MOR208/XmAb5574)
- Solid tumors

- Focus built on core competencies (scientific and medical expertise)
- Leveraging network of leading KOLs

Creating Value by Building a Sustainable Proprietary Pipeline

Internal Target Scouting
e.g. GM-CSF (MOR103), CD38 (MOR202)

Outlicensing

MorphoSys‘s Opportunities to Create Value

Target Validation | Discovery | Pre-Clinic | Phase 1 | Phase 2 PoC | Phase 3 | Market

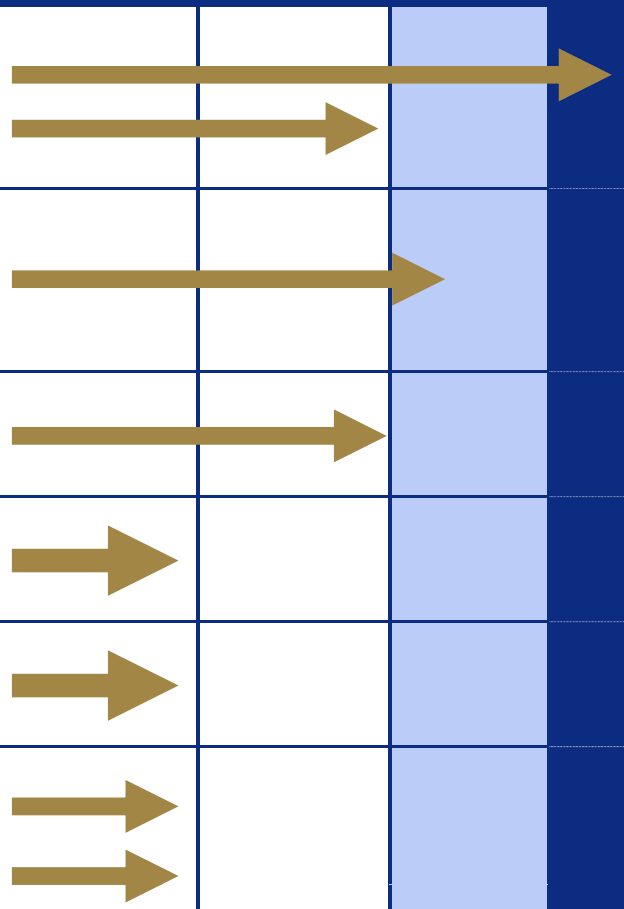
Academic Collaborations
e.g. Absynth, for access to innovative targets in infectious diseases

Xencor‘s CD19 Antibody (MOR208/XmAb5574)

Strategic Alliances
Five Galapagos targets in bone and joint diseases, of which two could lead to discovery programs in 2011

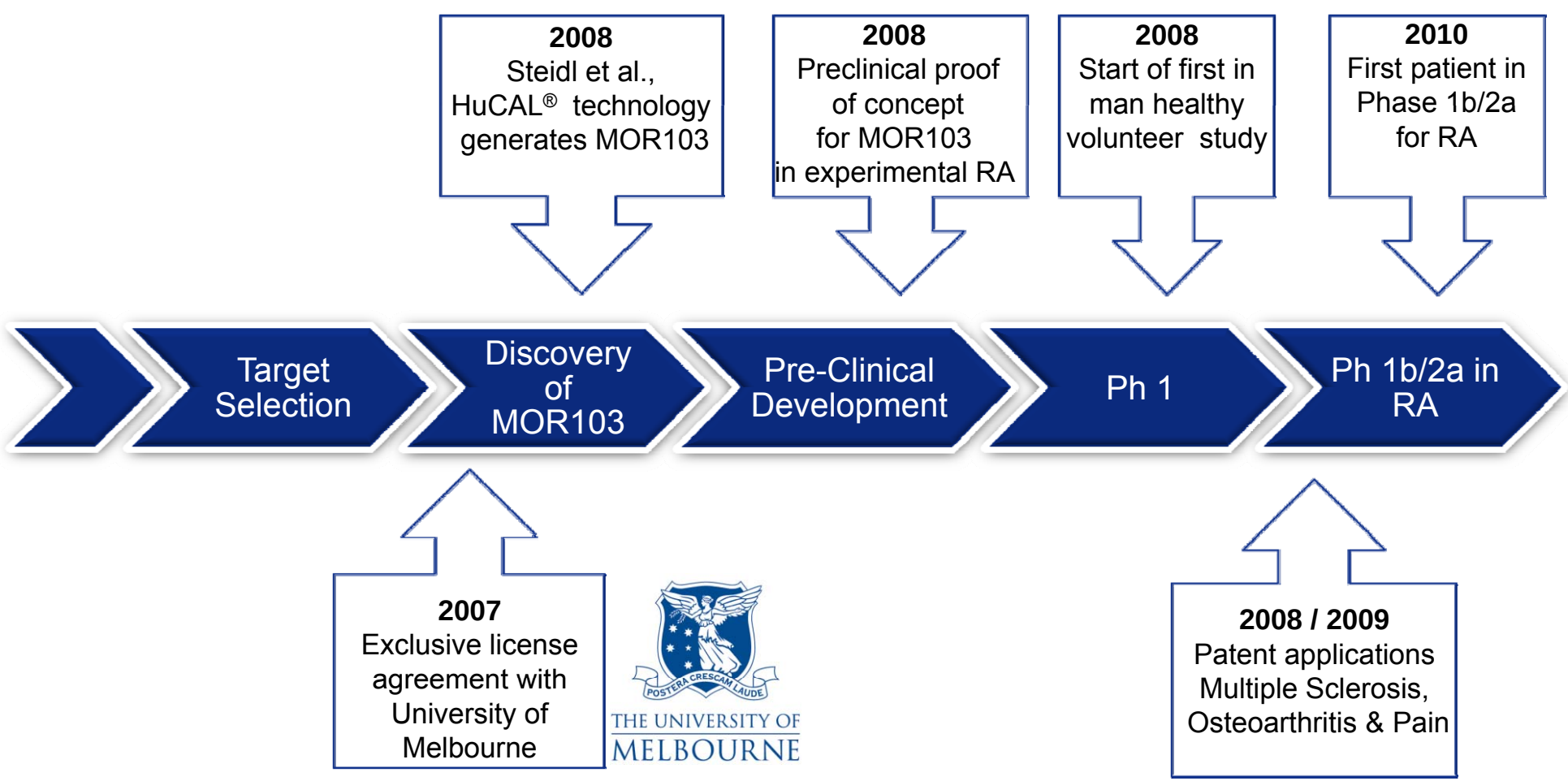
Growing Proprietary Portfolio: Ten Active Programs

Name	Target	Indication	Discovery	Preclinic	Ph 1	Ph 2	Ph 3	Market
MOR103	GM-CSF	Rheumatoid Arthritis (RA) Multiple Sclerosis (MS)						
MOR208/ XmAb5574	CD19	Chronic Lymphocytic Leukemia (CLL)						
MOR202	CD38	Multiple Myeloma (MM)						
2 Programs	n.d.	Inflammation						
3 Programs	n.d.	Oncology						
2 Programs MOR/NOV	n.d.	Inflammation						



n.d. – not disclosed

Genesis of the MOR103 anti-GM-CSF Program



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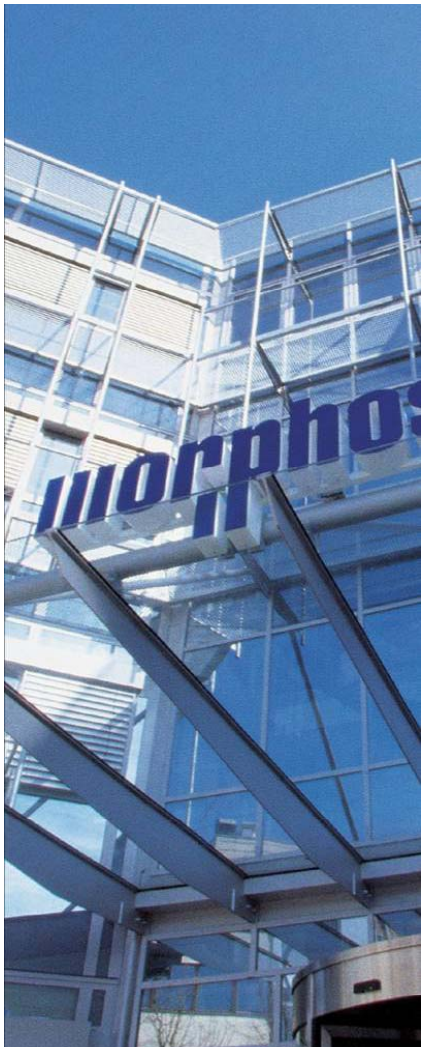
09:30	Simon Moroney	Welcome and Introduction
09:35	Marlies Sproll	Latest Technology Developments
		Update Partnered Pipeline
10:05	Q&A	
10:20	Arndt Schottelius	Introduction Proprietary Portfolio
10:25	**John Hamilton**	**GM-CSF – Central Mediator in Inflammation**
10:55	Arndt Schottelius	MOR103 in RA & MS
11:10	Q&A	
11:25	Lisa Rojkjaer	MOR202 in Multiple Myeloma (MM)
		MOR208 in Chronic Lymphocytic Leukemia (CLL)
11:55	Q&A	
12:15	Simon Moroney	Closing Remarks



GM-CSF as a target in inflammation

John A Hamilton

**Arthritis and Inflammation Research Centre, The University of Melbourne,
Department of Medicine, The Royal Melbourne Hospital,
Parkville, Victoria, Australia**

THE UNIVERSITY OF MELBOURNE

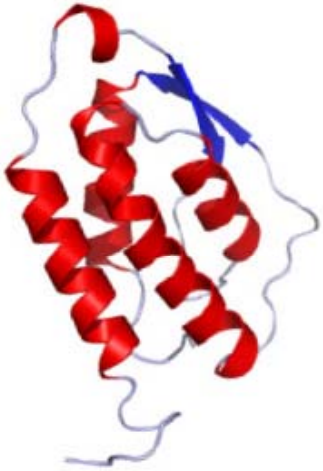
Topics

- Biology of GM-CSF
- GM-CSF as a proinflammatory cytokine
- Indications for anti-GM-CSF therapy
- Advantages vs caveats

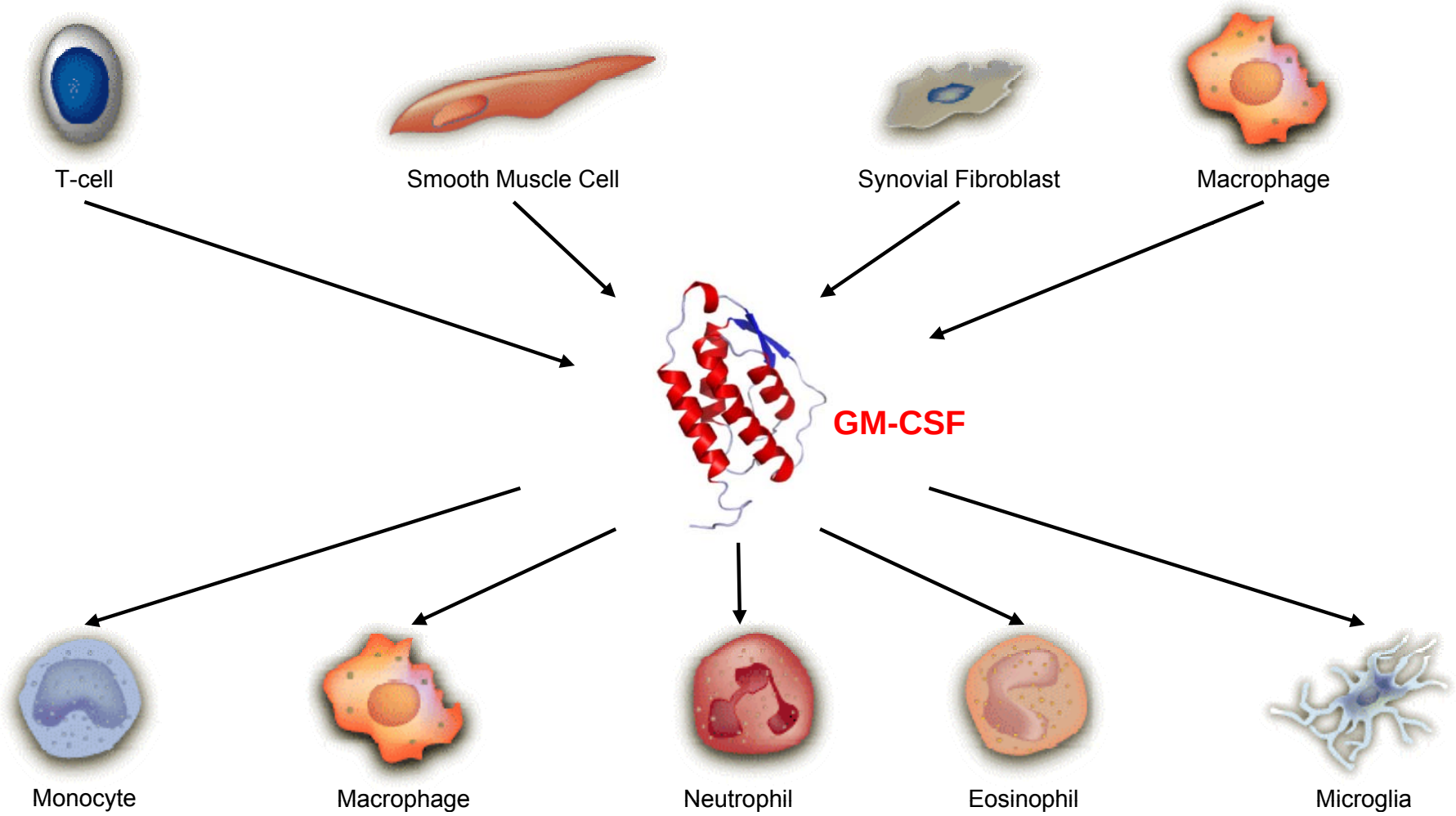
GM-CSF

Structure and Activity

- Glycoprotein: MW≈15kDa
- Binds GM-CSF-receptor (GM-CSF-R)
- GM-CSF-R is a transmembrane glycoprotein (α- and β-subunits)
- Initiates Stat-5 signalling



GM-CSF producing and responding cells



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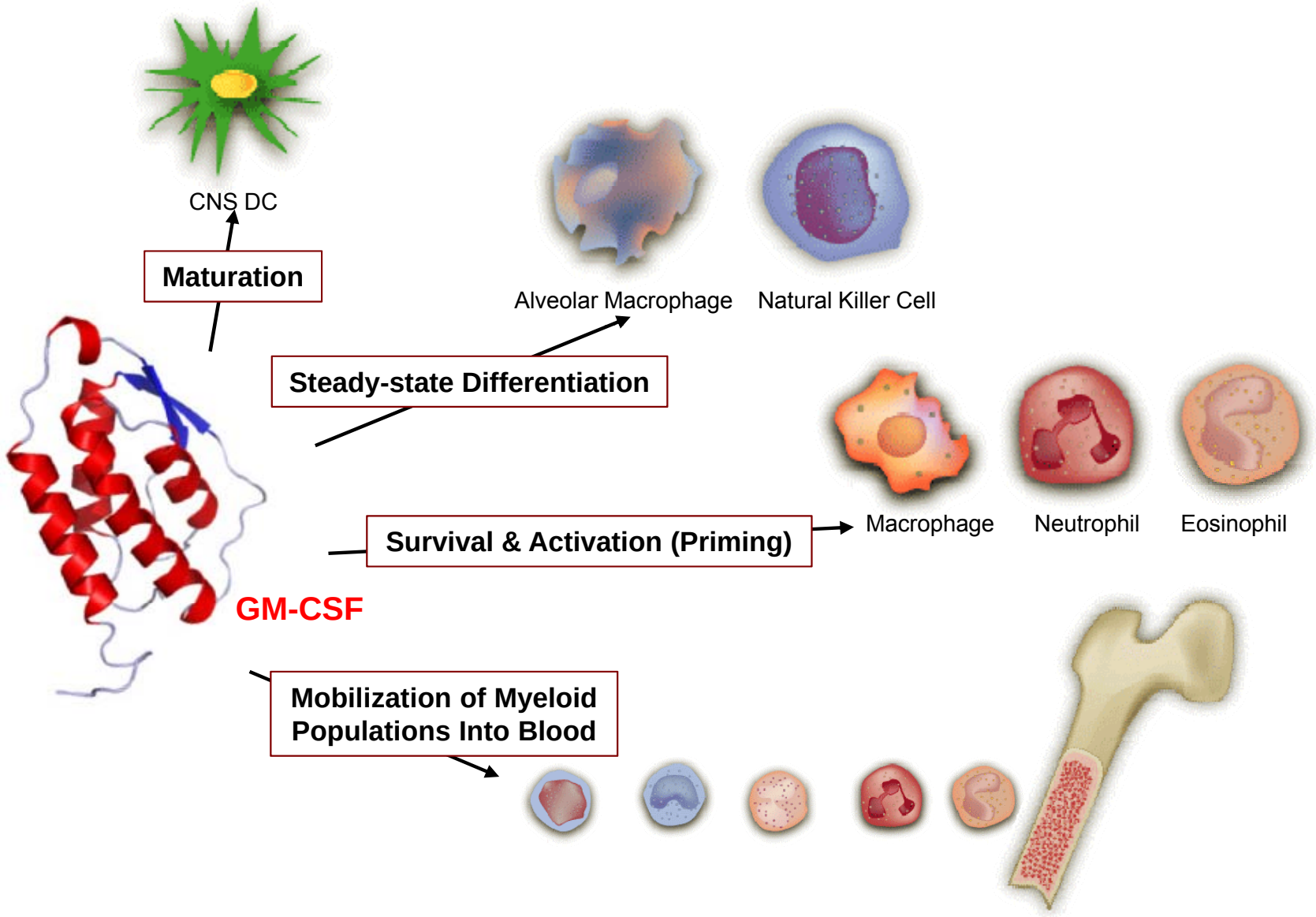
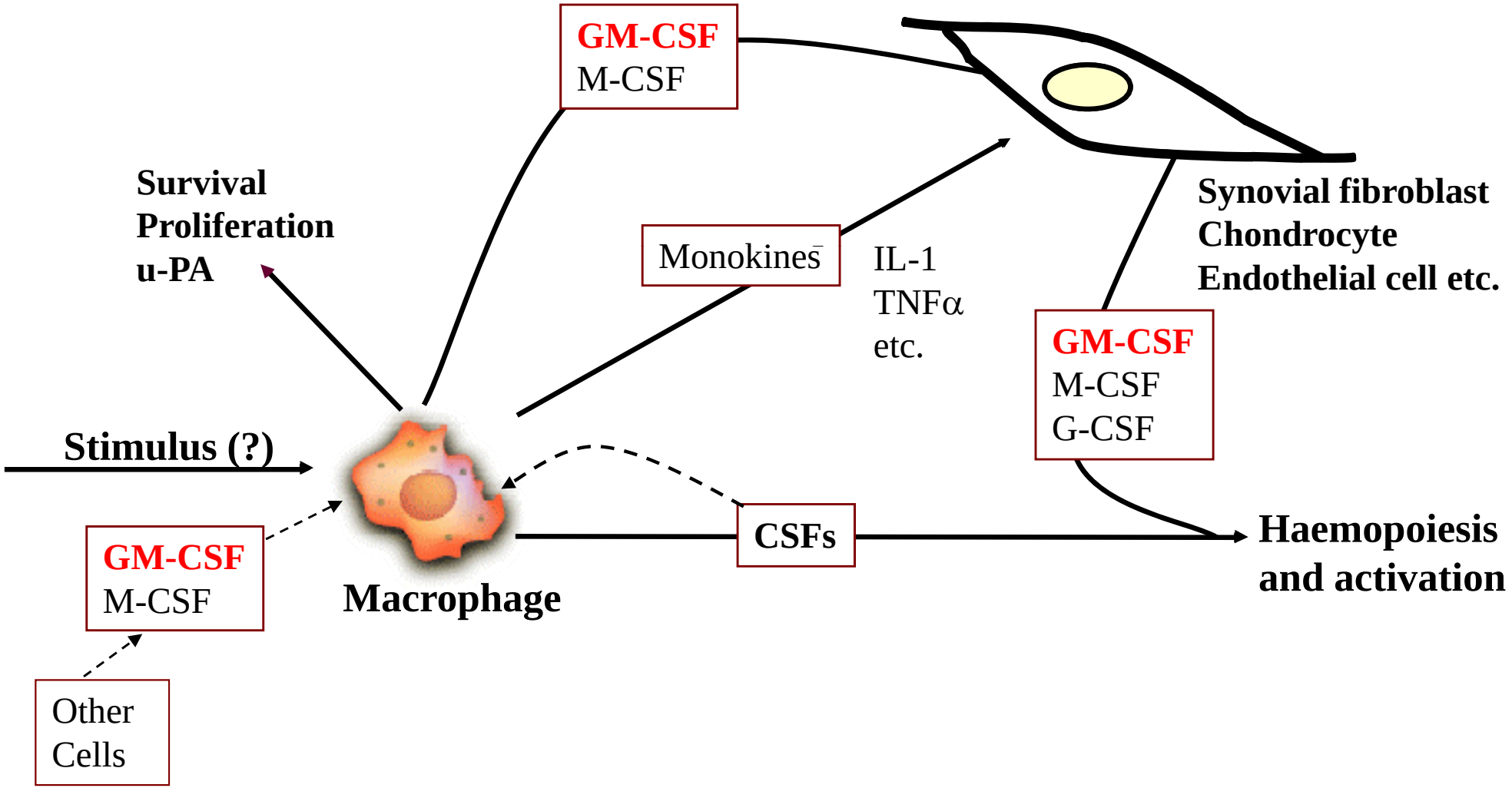
The main functions of GM-CSF



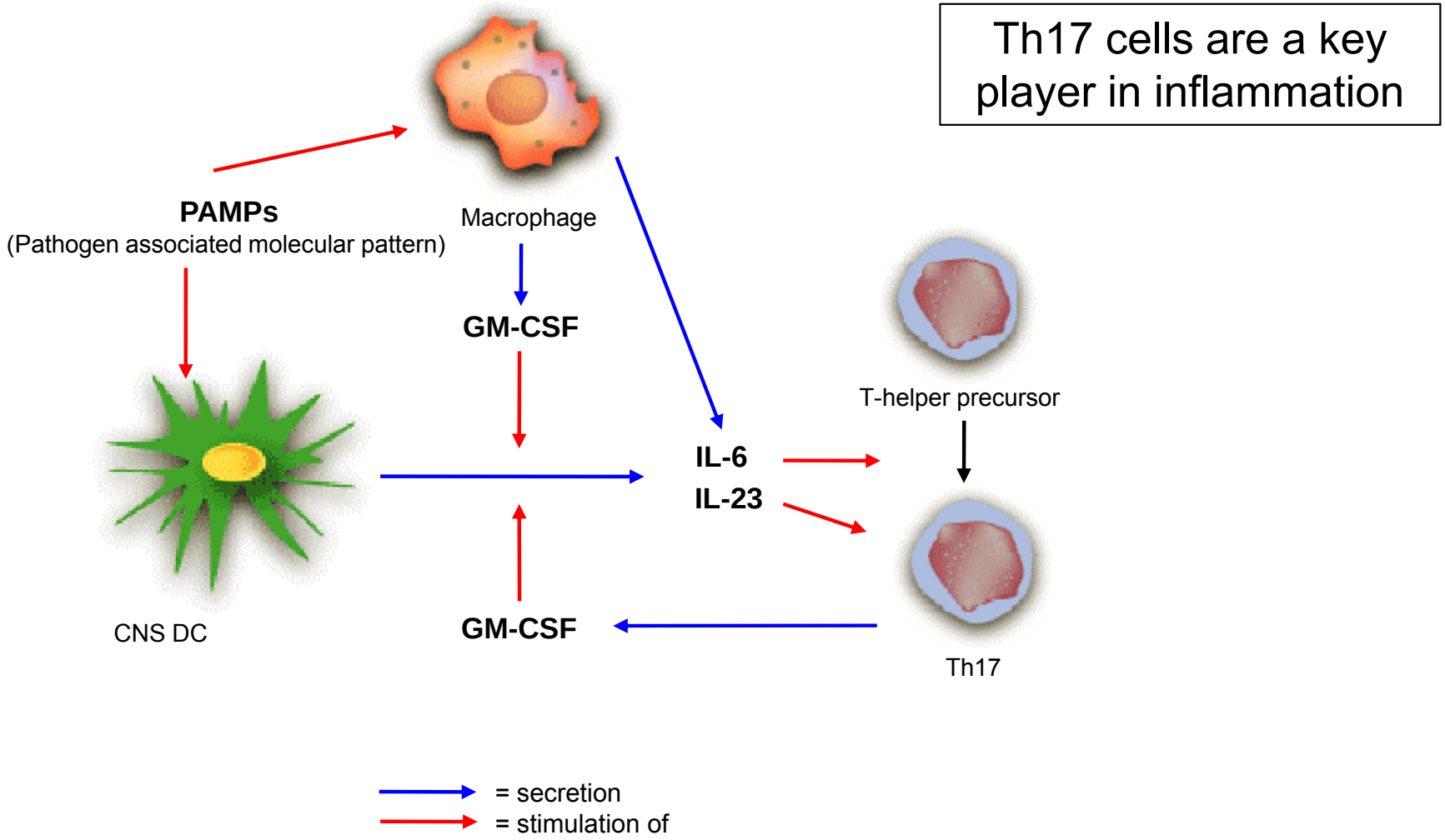
Adapted from: **Hamilton JA, (2008)** ***Nat Rev Immunol. 8:533-44***

GM-CSF controls inflammatory cell numbers, activation and is part of a cytokine network & feedback loop



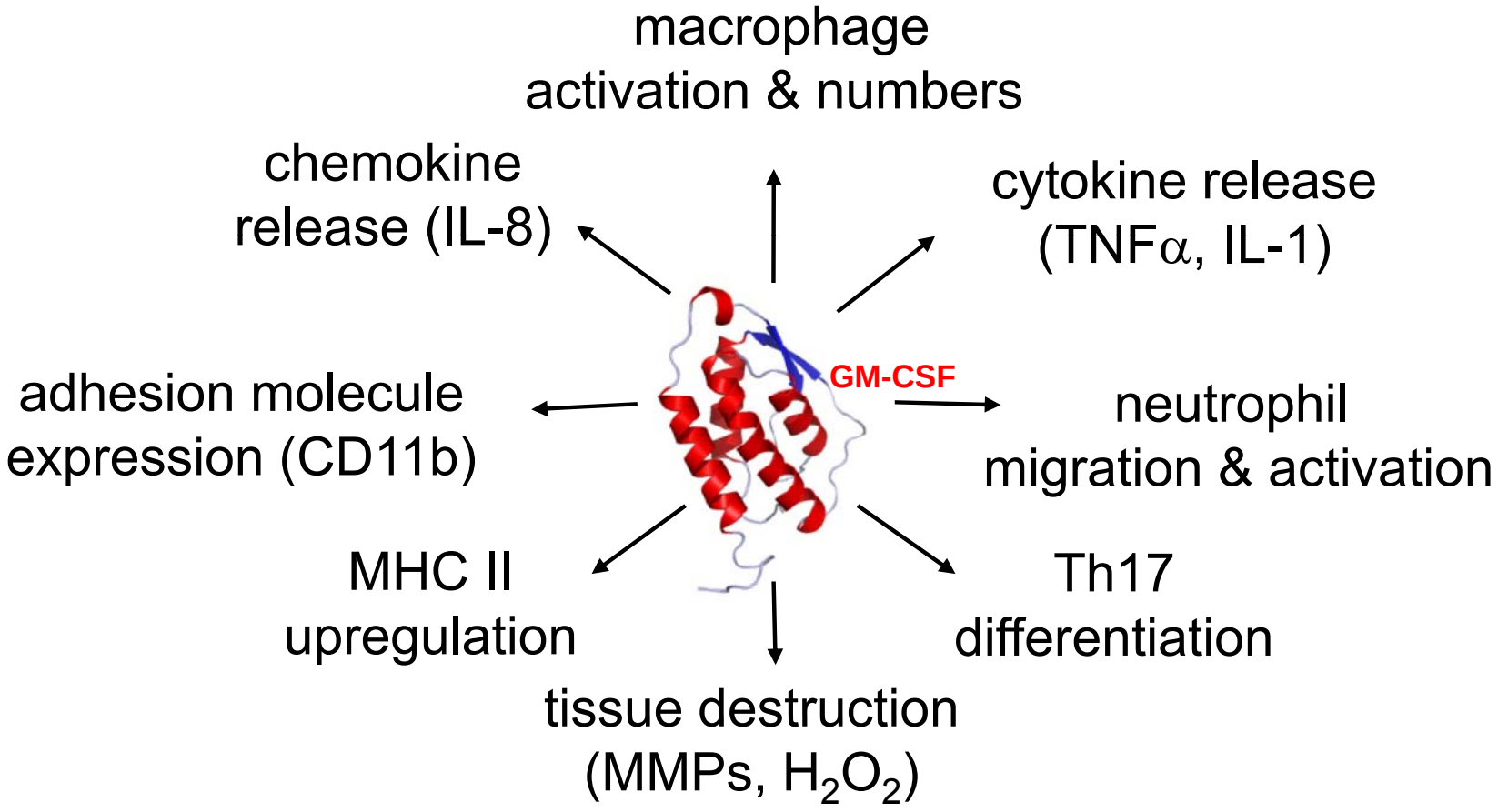
Adapted from Hamilton JA, The Lancet (1993) 342: 536 - 539

Novel role for GM-CSF in the generation and maintenance of Th17 cells by regulation of IL-6 and IL-23



adapted from Sonderegger I et al. J Exp Med (2008), 205:2281-2294

Biologic actions of GM-CSF in the pathogenesis of inflammatory diseases



Topics

- Biology of GM-CSF
- GM-CSF as a proinflammatory cytokine
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- Advantages vs caveats

In vivo validation of GM-CSF as target for inflammation

- Exacerbation of disease by GM-CSF
- Suppression of disease in GM-CSF-knockout mice
- Suppression of disease by neutralizing antibodies to GM-CSF

Involvement of GM-CSF in many pathologies (I)

Indication	Experimental model	Reference
Rheumatoid Arthritis	CIA mouse model	Campbell et al (1998) *J. Immunol 161:3639-3644* Cook et al., (2001) *Arthritis Res 3:293-298*
Rheumatoid Arthritis	SCW induced arthritis in mouse	*Plater-Zyberk et al., (2007) Ann Rheum Dis 66:452-457*
Rheumatoid Arthritis	SCW induced arthritis in rat	*MorphoSys (2008); HAH conference; www.morphosys.com*
Multiple Sclerosis (MS)	PLP / MOG induced EAE in mouse	McQualter et al., (2001) *J Exp Med. 194:873-882* King et al., (2009) *Blood 113: 3190-3197*
Multiple Sclerosis	MBP specific T-cell transfer EAE in mouse	Ponomarev et al., (2007) *J Immunol 178:39-48.*
Multiple Sclerosis	MOG induced EAE in rat; transgenic MS mouse model	*MorphoSys; unpublished data*

Involvement of GM-CSF in many pathologies (II)

Indication	Experimental model	Reference
COPD	4d smoke exposure model	Vlahos et al., (2010) *Am J Respir Crit Care Med 182, 34–40*
Asthma	Ovalbumin sensitization / airway hyperresponsiveness	Yamashita et al., (2002) *Cell Immunol 219, 92–97*
Psoriasis	Flaky skin mice	Schön et al., (2009) *J. Invest Dermatol 114, 976–983*
Bone Cancer Pain	Mouse sarcoma model of bone tumor–induced pain	Schweizerhof et al., (2009) *Nat Med 15, 802-7*
Artherosclerosis	Artherosclerotic lesions in Ldlr-/- mice	Zhu et al, (2009) *J Exp Med 206, 2141-2149*
Osteoarthritis (OA)	Collagenase induced OA	Studies currently ongoing

CIA Collagen induced arthritis
SCW Streptococcus cell wall
PLP Proteolipid protein
MOG Myelin oligodendrocyte glycoprotein

EAE Experimental autoimmune encephalomyelitis
MBP Myelin basic protein
COPD Chronic obstructive pulmonary disease
Ldlr-/- Low density lipoprotein receptor null mice

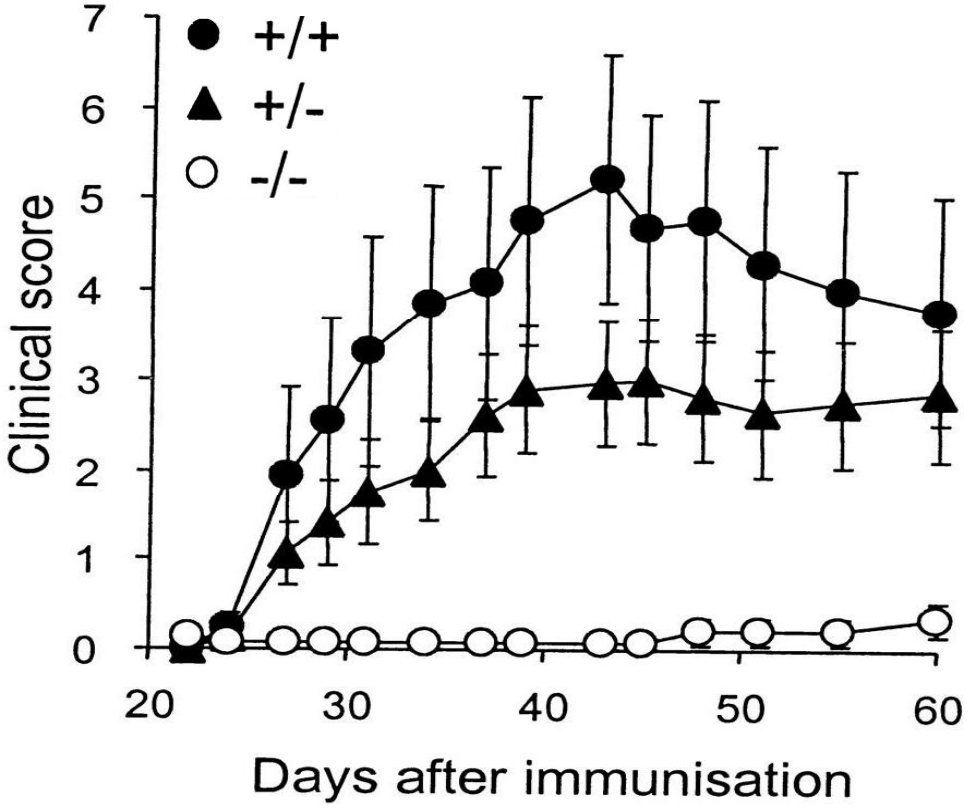
GM-CSF is a promising target for the treatment of rheumatoid arthritis

- Elevated levels of GM-CSF found in RA synovial fluid
- GM-CSF treatment led to flares of RA in patients with Felty syndrome
- Macrophage numbers in RA synovial tissue
  - increased numbers correlate with disease severity
  - decreased numbers observed after treatment of RA

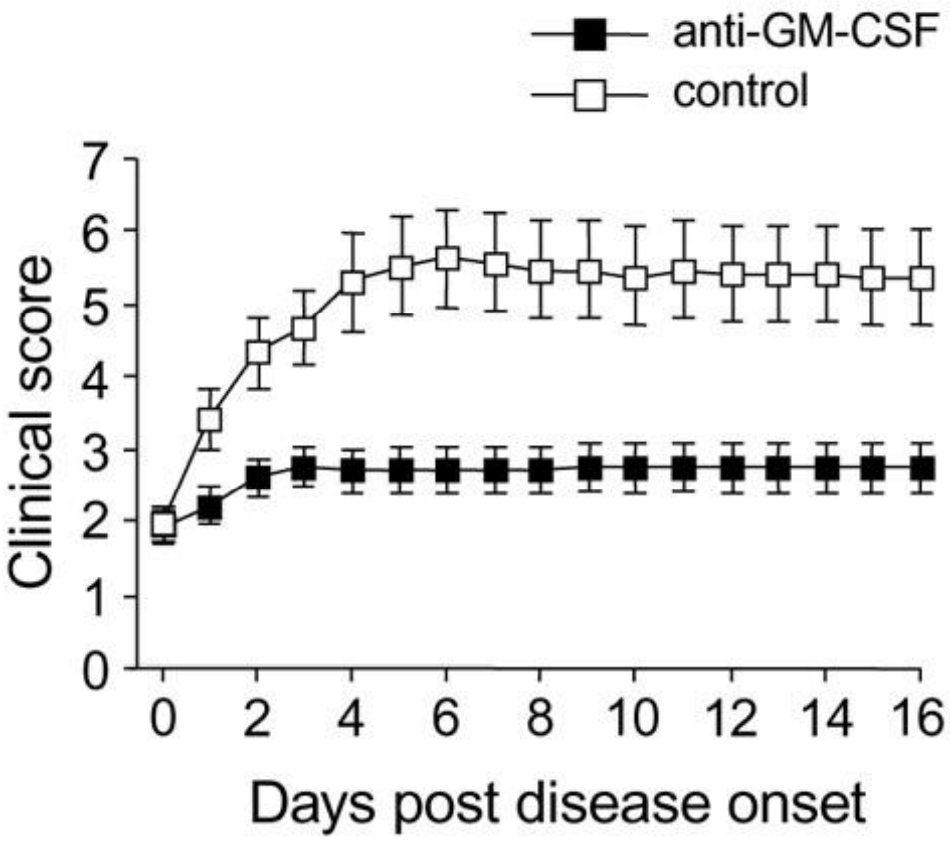
  (*Hamilton JA & Tak PP.Arthritis Rheum. 2009 May;60(5):1210-21*)
- Anti-GM-CSF mAb is an effective treatment in RA disease models

GM-CSF depletion suppresses collagen induced arthritis

GM-CSF$^{-/-}$ mouse



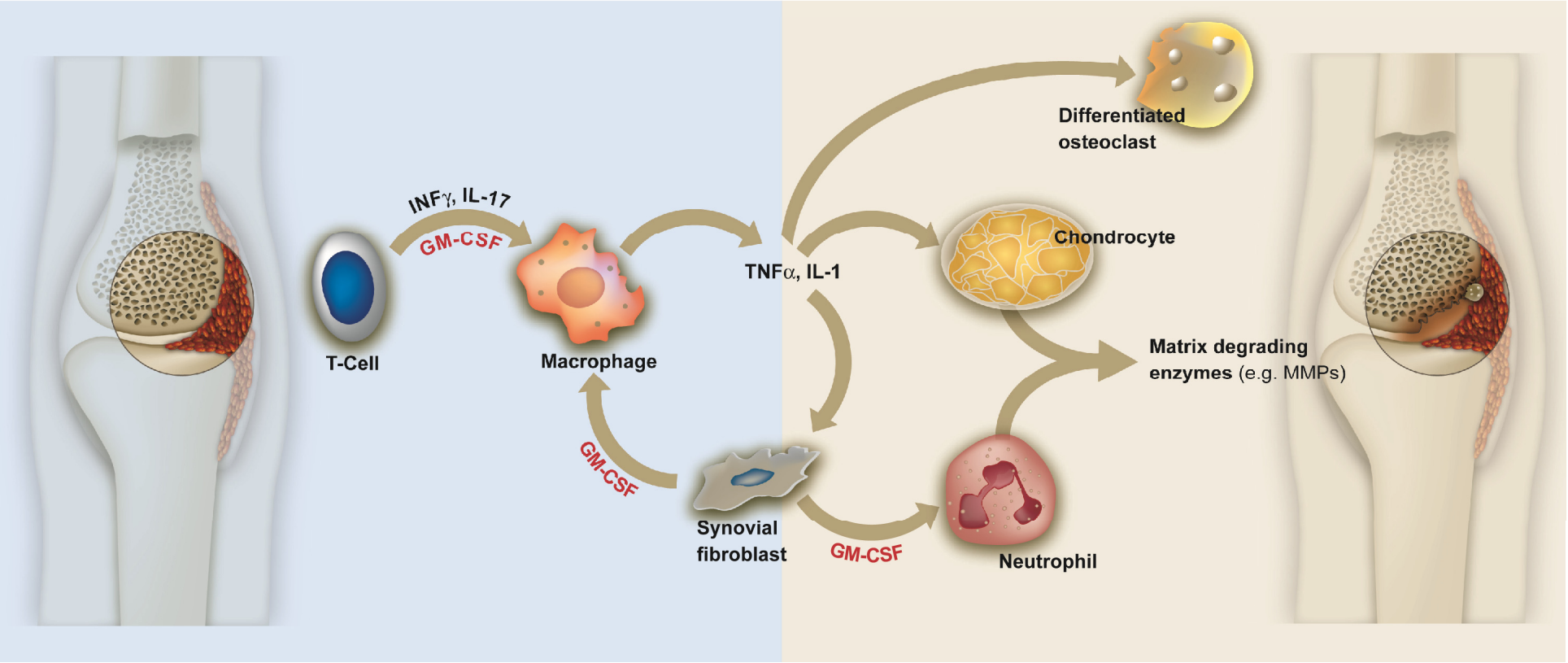
anti-GM-CSF mAb (mouse)



Campbell IK, Rich MJ, Bischof RJ, Dunn AR, Grail D, Hamilton JA. J. Immunol. (1998) 161: 3639

Cook AD, Braine EL, Campbell IK, Rich MJ, Hamilton JA. Arthritis Res. (2001) 3: 293

GM-CSF is strongly implicated in the pathogenesis of rheumatoid arthritis



Inflammation & Synovitis

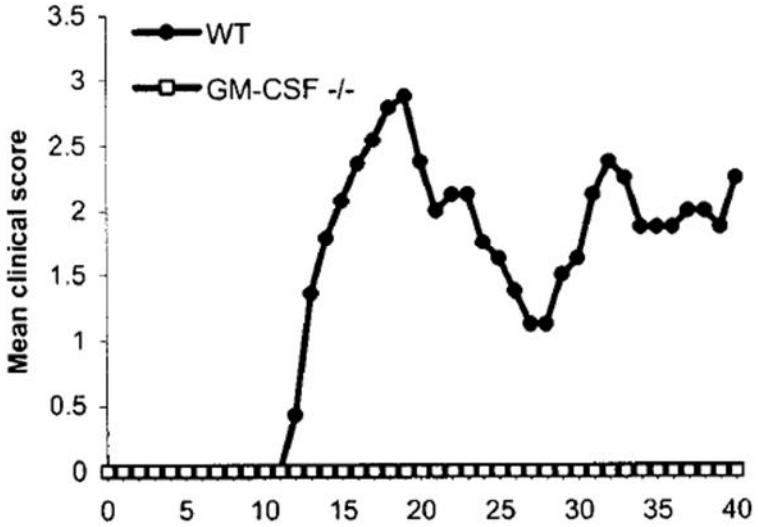
Structural Damage – Bone Erosion

GM-CSF is a promising target for the treatment of multiple sclerosis

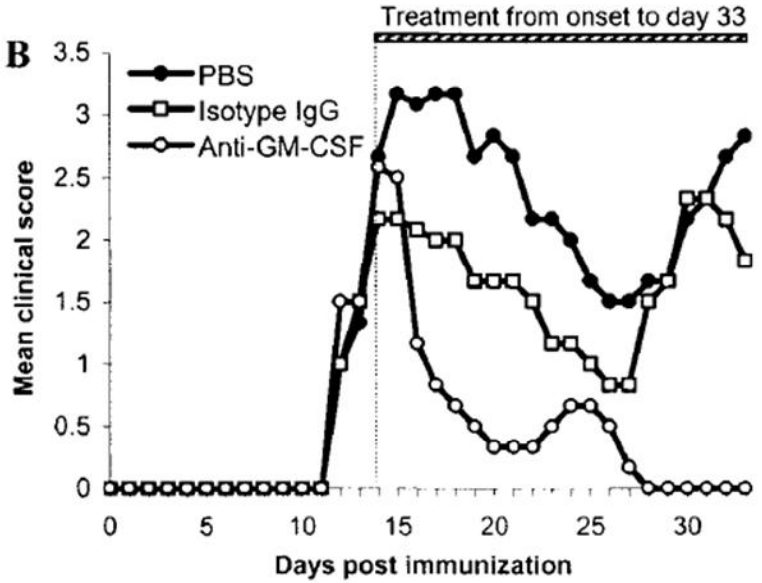
- Elevated levels of GM-CSF correlate with the active phase of MS (*Carrieri et al., 1998*)
- Recombinant GM-CSF exacerbates EAE in GM-CSF^{wt} mice (*McQualter et al., 2001*)
- Anti-GM-CSF mAb is an effective treatment in MS disease models

GM-CSF depletion suppresses experimental autoimmune encephalomyelitis (EAE)

GM-CSF$^{-/-}$ mouse

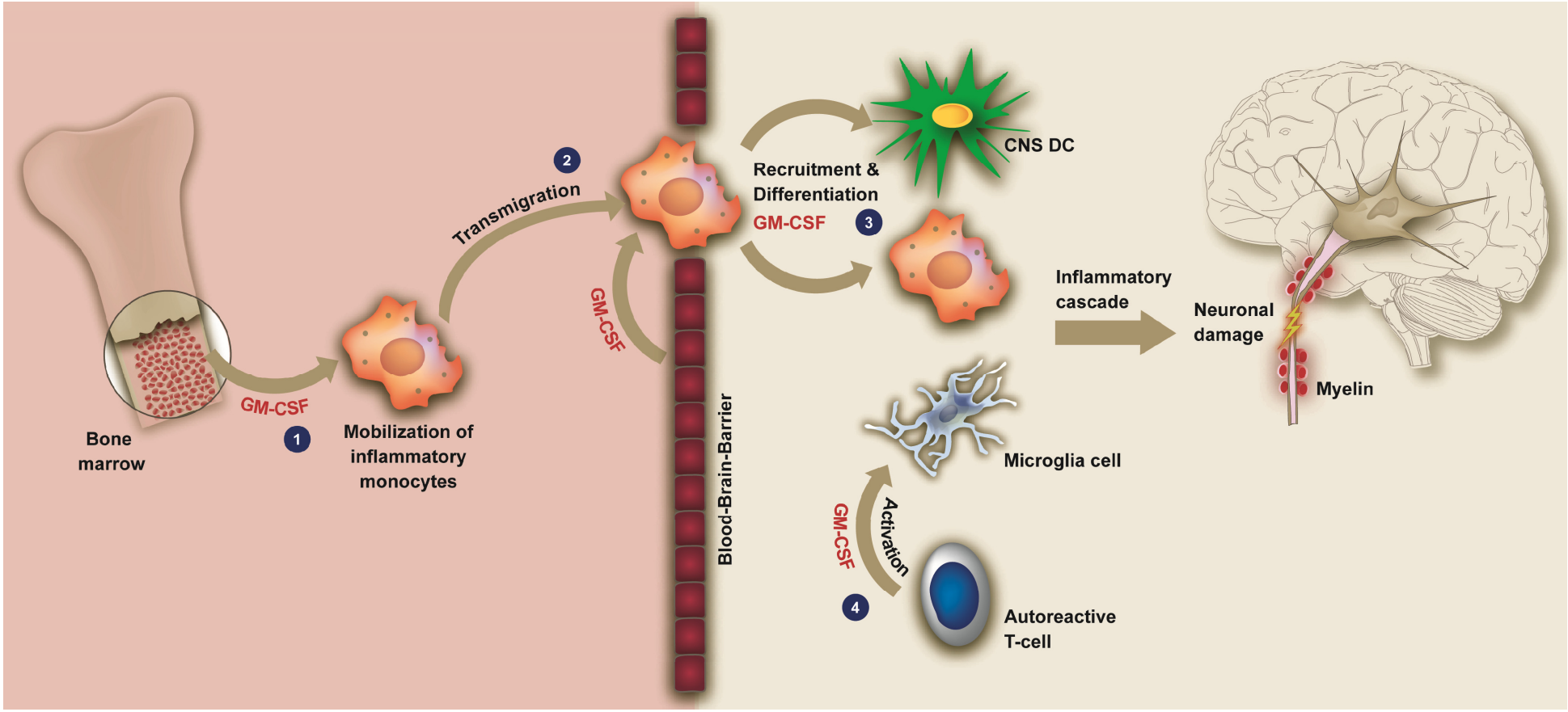


anti-GM-CSF mAb (mouse)



McQualter JL, Darwiche R, Ewing C, Onuki M, Kay TW, Hamilton JA, Reid HH, Bernard CC. J Exp Med. (2001) 194:873-82

GM-CSF is strongly implicated in the pathogenesis of multiple sclerosis



1 King et al., (2009) *Blood* 113:3190-7
2 Ifergan et al. (2008) *Brain* 131:785-99
3 Hesske et al., (2010) *Brain* 133:1637-54
4 Ponomarev et al., (2007) *J Immunol* 178:39-48

Topics

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- GM-CSF as a proinflammatory cytokine
- Indications for anti-GM-CSF therapy
- Advantages vs Caveats

GM-CSF targeting caveats

GM-CSF $^{-/-}$ mouse phenotype

- Steady state hematopoiesis – unimpaired
- Accumulation of phospholipids and surfactant proteins in the lung: resembling pulmonary alveolar proteinosis (*Stanley et al 1994; Dranoff et al 1994)*
- GM-CSF $^{-/-}$ mice are more susceptible to infections, when experimentally challenged with high doses of pathogens

Pulmonary Alveolar Proteinosis (PAP)

- Clinical features: dyspnea, dry cough
- In the largest cohort of PAP patients investigated (n=248) intercurrent illnesses, including infections, were infrequent (Inoue et al 2008)
- Manifestation of clinical PAP seems to require long-term GM-CSF deficiency caused by high levels of GM-CSF autoantibodies; clinically manageable

➢ **Pulmonary function and infection risk should be monitored in clinical trials**

GM-CSF targeting advantages

- "Upstream" mediator
  - dramatic therapeutic effect in disease models of RA and MS
  - central player in inflammatory processes
  - links to TNFα, IL-1, IL-23 / Th17 biology
- Unlikely a steady state hematopoietic growth factor
  - no impact on hematopoiesis expected for anti-GM-CSF mAb
- Control of myeloid numbers and activation at sites of inflammation
  - therapeutic effect expected at site of pathology (e.g. RA joints)

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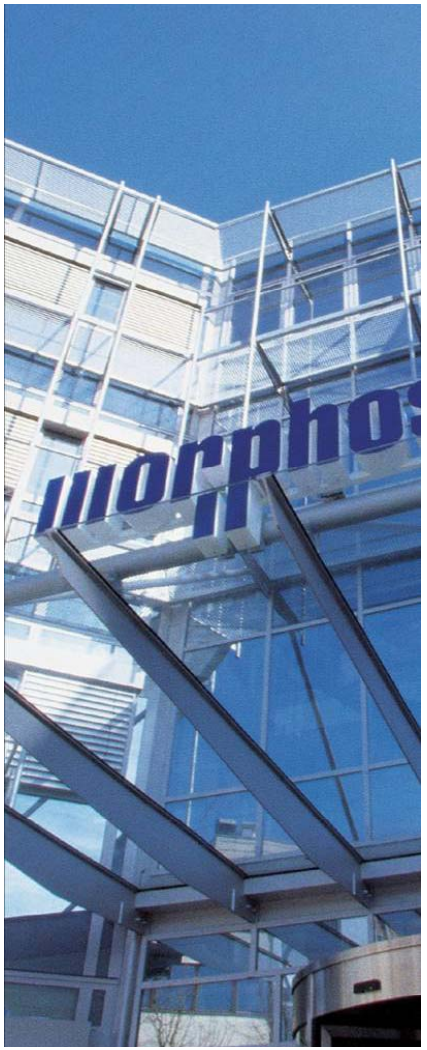
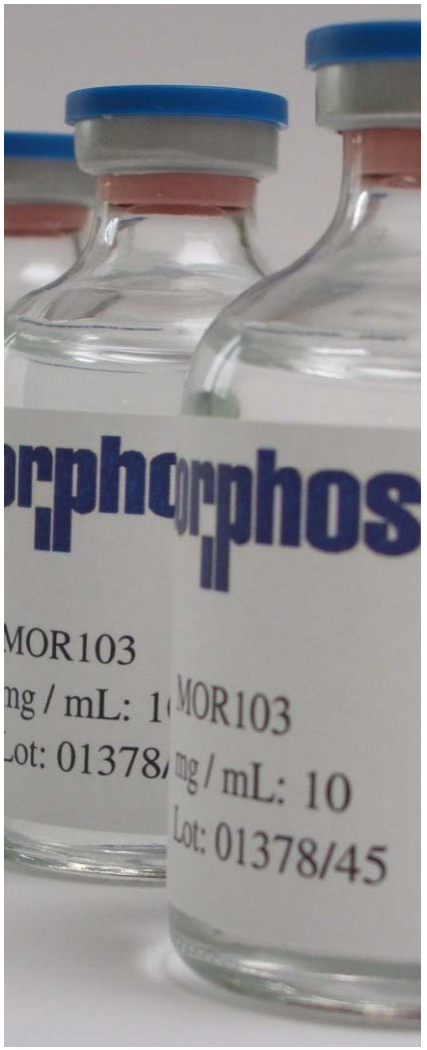


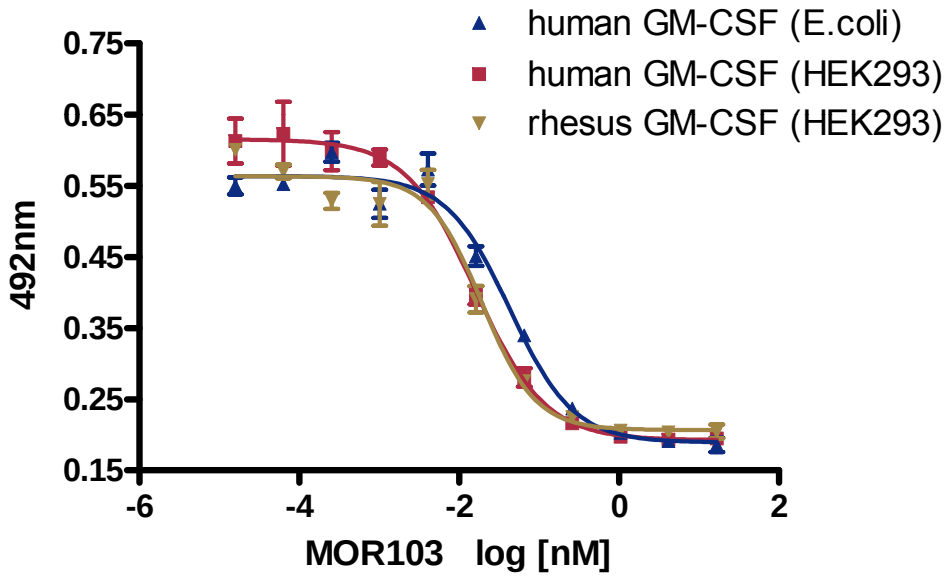
MOR103 Antibody Features

Description	■ HuCAL-derived, fully human, monclonal IgG1 antibody directed towards human GM-CSF
Affinity	■ Human and rat GM-CSF (K_D= 0.4 pM and 1100 pM)
Function	■ Inhibition of binding of GM-CSF to GM-CSFRα ■ Antibody inhibits downstream signaling of GM-CSF receptor
Potency	■ Neutralizes human and rhesus GM-CSF (IC50 = 11 pM to 48 pM) ■ Neutralizes rat GM-CSF (IC50 = 35 nM) ■ Approx. 1000-fold lower affinity and potency in rats vs. humans: weaker effects in rat models expected



MOR103 Potently Neutralizes GM-CSF

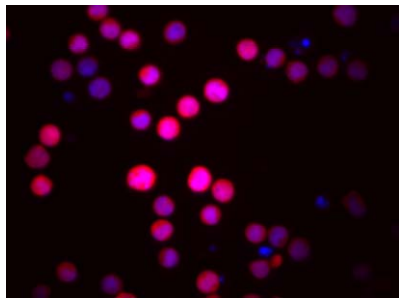
MOR103 inhibits proliferation of TF-1 cells



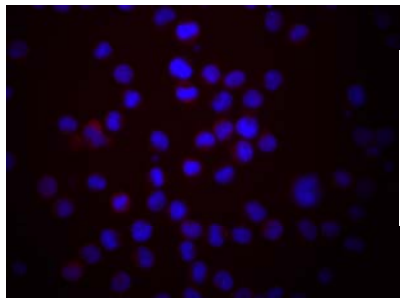
IC_{50}: 48 pM - human GM-CSF (*E.coli*)
11 pM - human GM-CSF (HEK293)
15 pM - rhesus GM-CSF (HEK293)

MOR103 inhibits downstream signaling of GM-CSF-Receptor

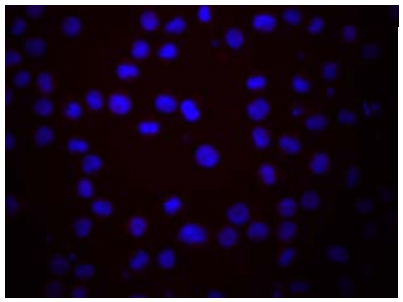
with GM-CSF



w/o GM-CSF

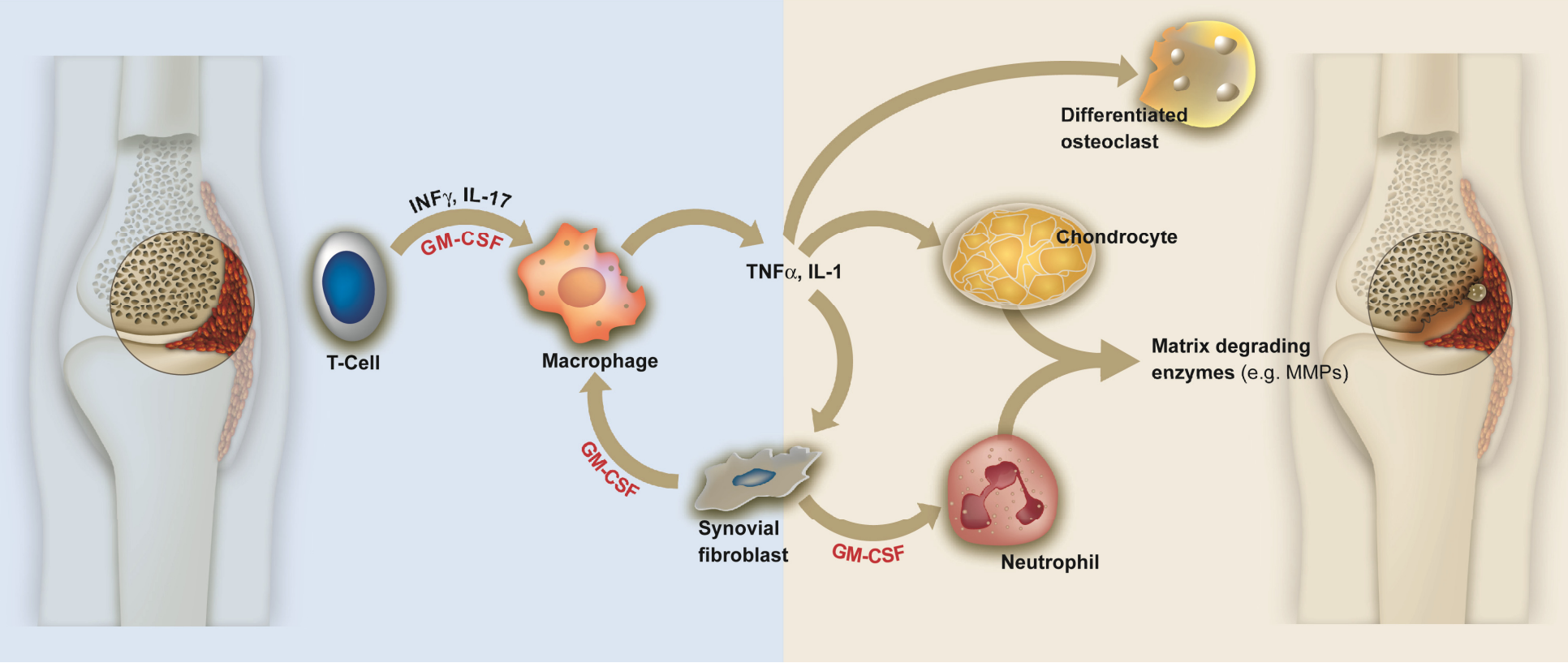


with GM-CSF + MOR103



Blocking of STAT5 phosphorylation:
TF-1 cells were stained with a phospho-specific STAT5 antibody (red)
Nuclei were stained with Hoechst33342 (blue)

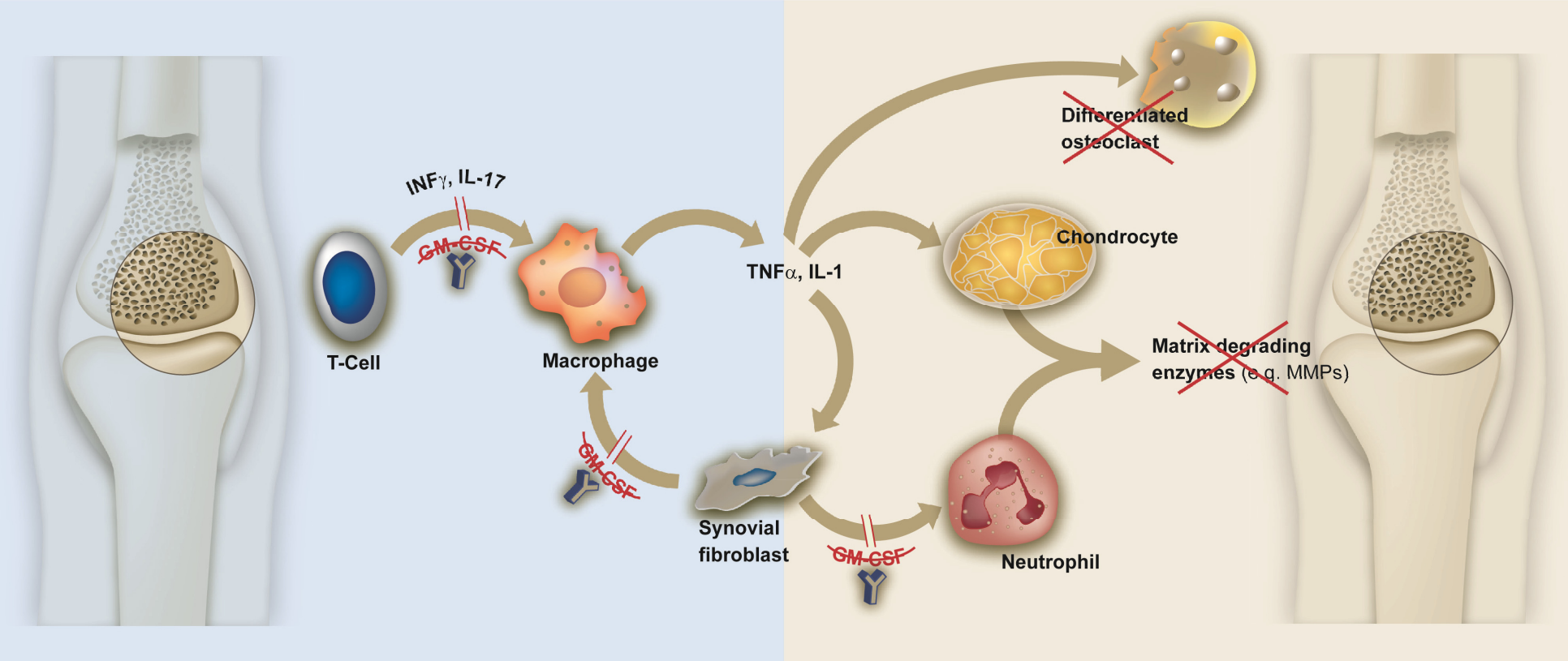
GM-CSF is Strongly Implicated in the Pathogenesis of Rheumatoid Arthritis



Inflammation & Synovitis

Structural Damage – Bone Erosion

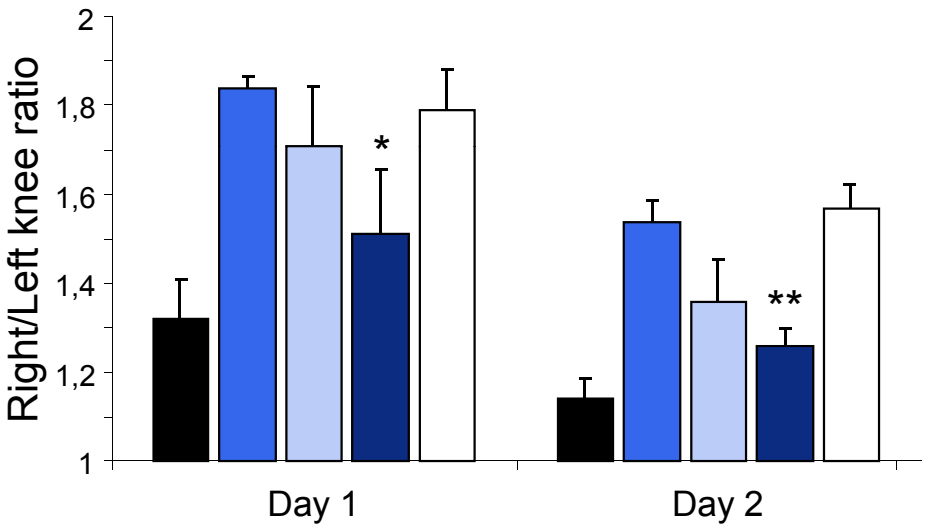
Expected Therapeutic Effects of Treatment with MOR103 in Rheumatoid Arthritis



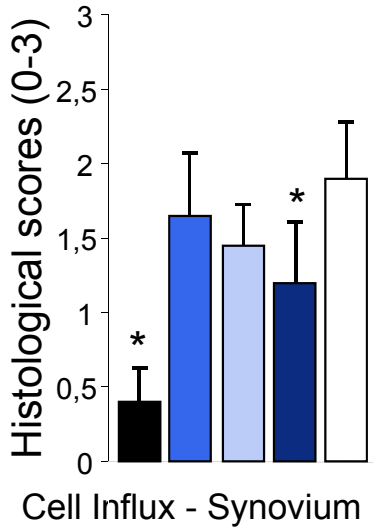
~~Inflammation & Synovitis~~ ~~Structural Damage – Bone Erosion~~

Results From SCW Induced Arthritis Rat Model

Knee joint swelling



Knee joint histopathology



Dexamethasone 3mg/kg
MOR103 10mg/kg
MOR103 20mg/kg
MOR103 50mg/kg
IgG1 control 50mg/kg

SCW: Streptococcal cell wall

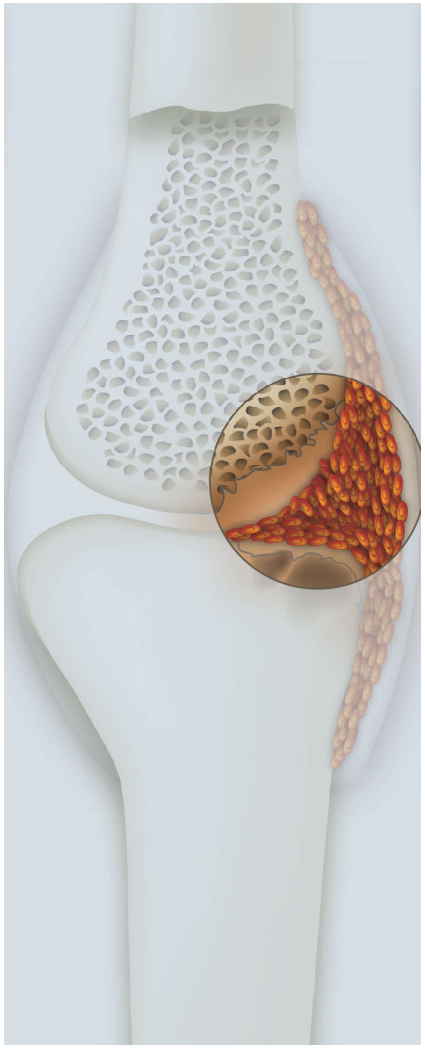
Error bars show: ±SD; n=5 rats/group; *P<0.03 and **P<0.008 compared to IgG1 control (Mann Whitney)

Treatment: MOR103 dosed at 10, 20 and 50 mg/kg once i.p. 2h before intra-articular administration of SCW fragments to male Wistar rats (right knee)
RA Parameters: Knee joint swelling (right/left knee ratio assessed by Technetium uptake) and histopathology (PMN cell influx)

Significantly decreased knee joint swelling and synovium cell influx following treatment with MOR103 compared to IgG1 control (at 50mg/kg)

Rationale for Selecting Rheumatoid Arthritis as Lead Indication for MOR103

- Strong scientific rationale based on literature and in-house data
  - Suppression of GM-CSF shows therapeutic effect in established RA animal models
  - GM-CSF-mediated flares in RA patients
  - Elevated presence of GM-CSF in synovium of arthritic joints
  - Suppression of base level GM-CSF in humans does not lead to changes in hematopoiesis
  - MOR103 ameliorates signs & symptoms of RA in established animal models
  - Novel mode of action, different from existing therapies
  - Links between GM-CSF and IL-23/Th17 biology
- Unmet medical need in RA
  - Fewer than 25% of RA patients adequately treated
  - 50% of TNF-responders stop responding within 2 years
- Commercial opportunity
  - RA Market >$13bn and growing
  - Interest from potential licensees



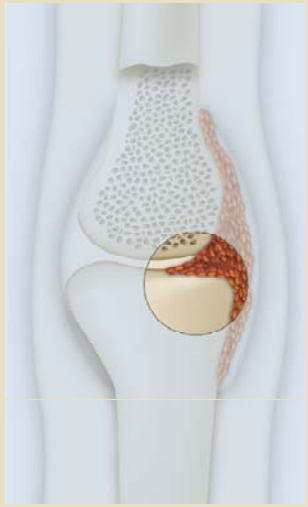
MOR103 – Phase 1b/2a (MSC-1001) in Rheumatoid Arthritis – Clinical Trial Update

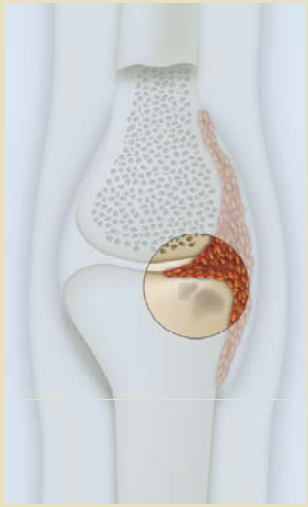
- Objectives: assess safety, signs of efficacy and immunogenicity of MOR103 in patients with active RA
- Trial design
  - Randomized, double-blind, placebo controlled
  - 135 patients with active RA
  - Four ascending doses i.v. 0.3, 1.0 and 1.5 mg/kg or placebo
  - Primary outcome measures: Adverse event rate and safety profile
  - Secondary outcome measures: DAS28, ACR core set measures and EULAR28 response criteria, cytokines, MRI (synovitis & bone edema), pharmacokinetics, immunogenicity and patient reported outcomes up to 16 weeks
  - Stable regimen of concomitant RA therapy
- Trial approved in Germany, Bulgaria, The Netherlands and Poland
  - Geographic mix provides access to full spectrum from biologics-pretreated to biologics-naïve RA patients

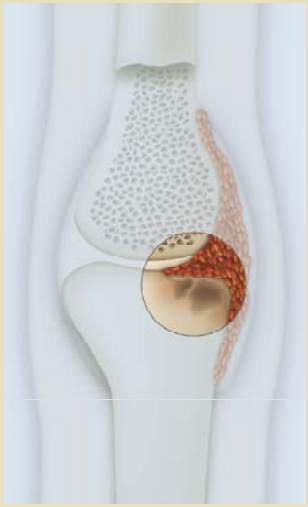
The Path to Bone Erosions in RA – Pre-erosive Changes Are Detectable by MRI

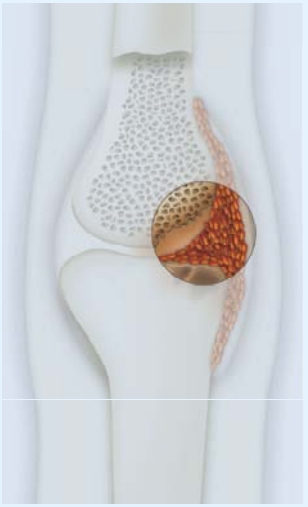
Natural progression of structural damage in RA: from synovitis, to osteitis, to bone erosions

Synovitis → Osteitis (Bone edema) → Microscopic bone erosion → Radiographic erosions









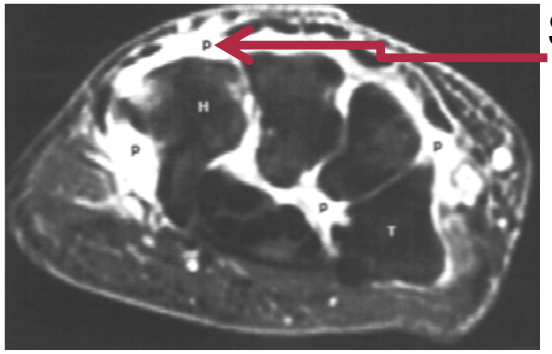
MRI can detect pre-erosive changes which cannot be detected by conventional x-ray

X-ray & MRI (MRI more sensitive)

MRI is the only imaging tool for early detection of synovitis & osteitis as risk factors for progressive destructive disease

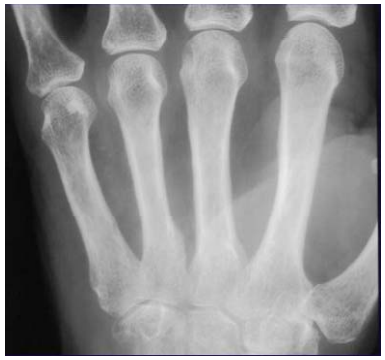
MRI – High Sensitivity Imaging Biomarker in RA

- MRI in RA is well validated:
  - MRI can detect reduction in synovitis and bone edema as early as 4-6 weeks after therapy
    (Lisbona MP et al. J Rheum 2008;35:394-397, Quinn MA et al. Arthr Rheum 2005;52:27-35)
  - MRI is more sensitive than X-ray for bone erosions
  - A validated MRI scoring system exists, thus MRI is suitable for clinical trials
- MRI imaging included in MSC-1001 protocol

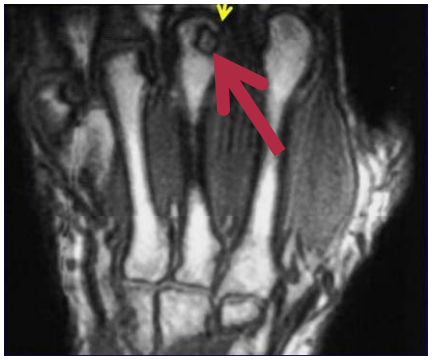


Synovitis (MRI)

Same hand of RA patient



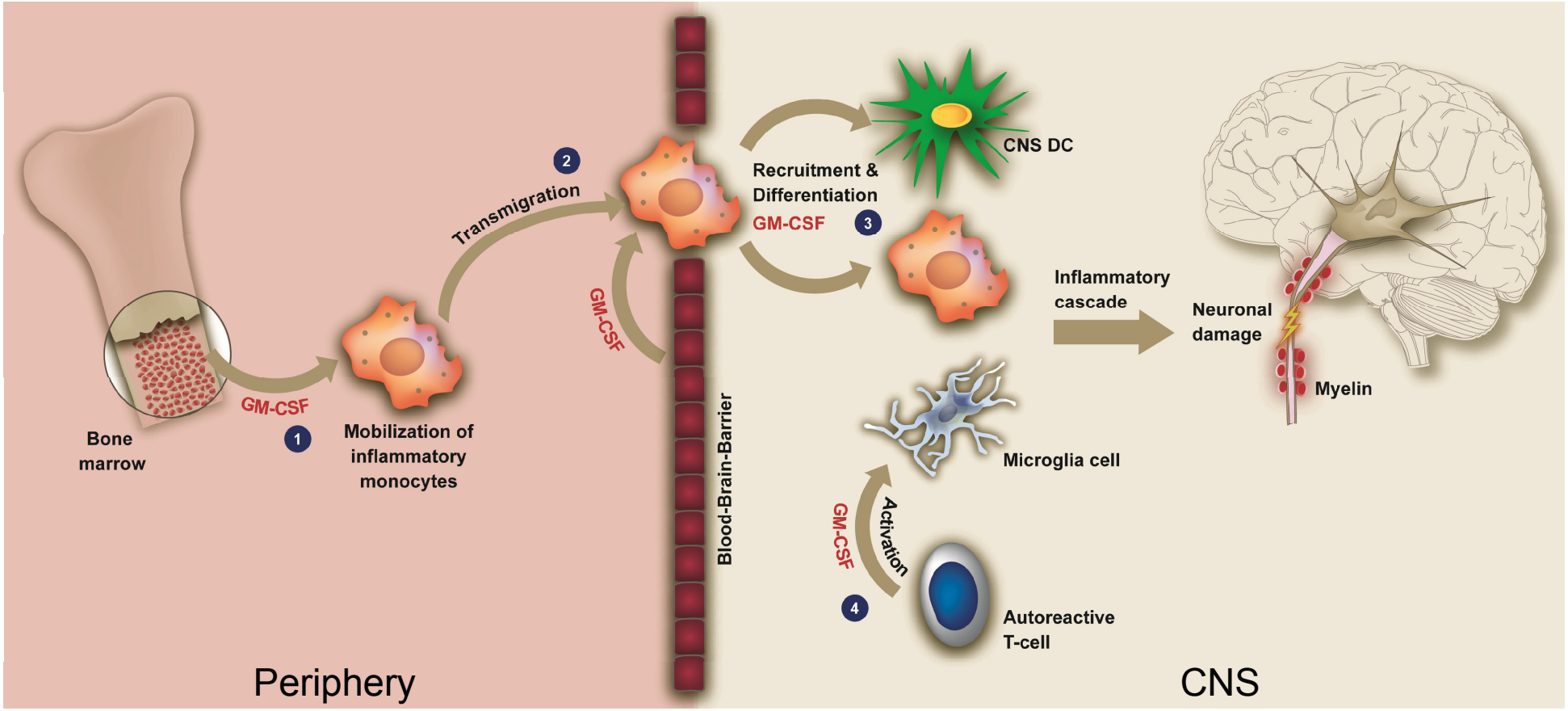
X-ray: bone erosion not visible (obscured)



MRI: Bone erosion visible

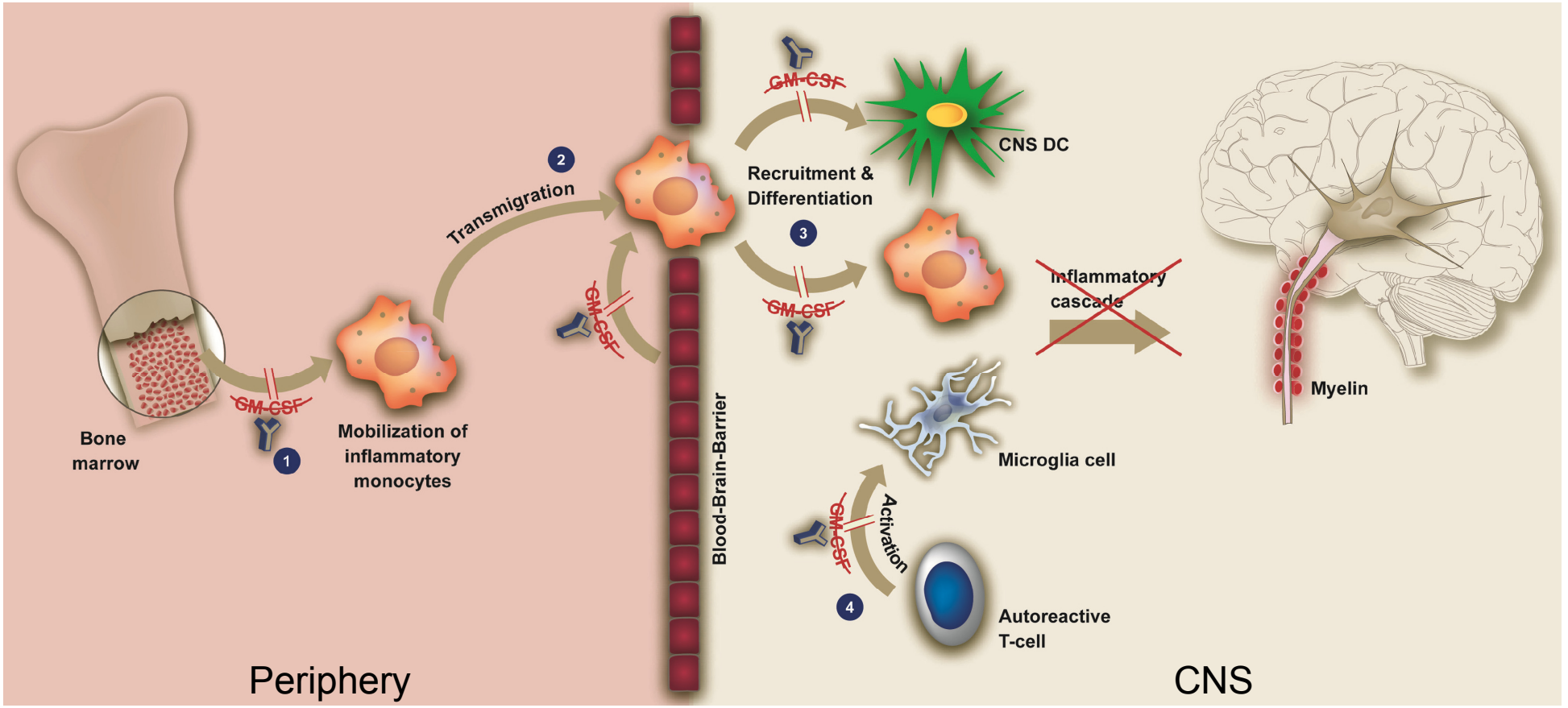
MRI – High Sensitivity Biomarker Capable of Demonstrating Early Responses to Therapy ➔ Enabling Early Proof of Concept

GM-CSF is Strongly Implicated in the Pathogenesis of Multiple Sclerosis



1. King et al., (2009) *Blood* 113:3190-7
2. Ifergan et al. (2008) *Brain* 131:785-99
3. Hesske et al., (2010) *Brain* 133:1637-54
4. Ponomarev et al., (2007) *J Immunol* 178:39-48

Expected Therapeutic Effects of Treatment with MOR103 in Multiple Sclerosis



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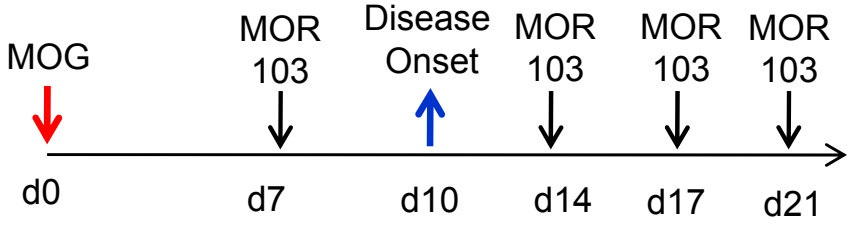
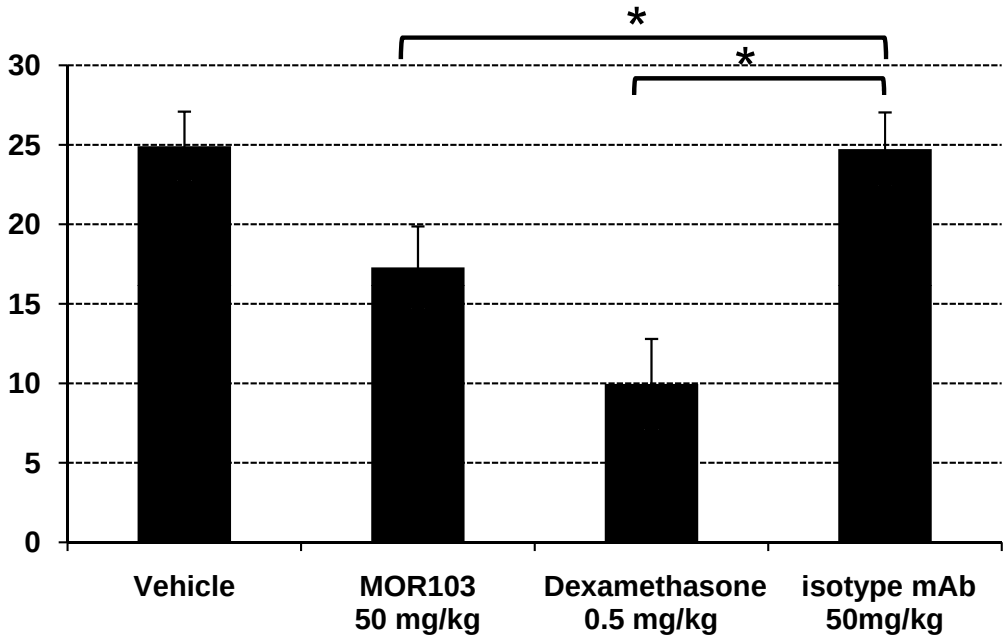
Results from MOG Induced Rat Model for Multiple Sclerosis

- Rats were injected with MOG[1] peptide on day 0
- MOR103 treatment after MOG immunization
- Disease onset around day 10
- Significant decrease in EAE score after preventive treatment on day 7
- Therapeutic treatment commencing on day 14 resulted in a trend of disease amelioration



Cumulative EAE[2] score (day 20)



* ANOVA LSD posthoc analyses vs. isotype (p <0.05); SEM is shown

[1] MOG: myelin oligodendrocyte glycoprotein
[2] EAE: experimental autoimmune encephalomyelitis

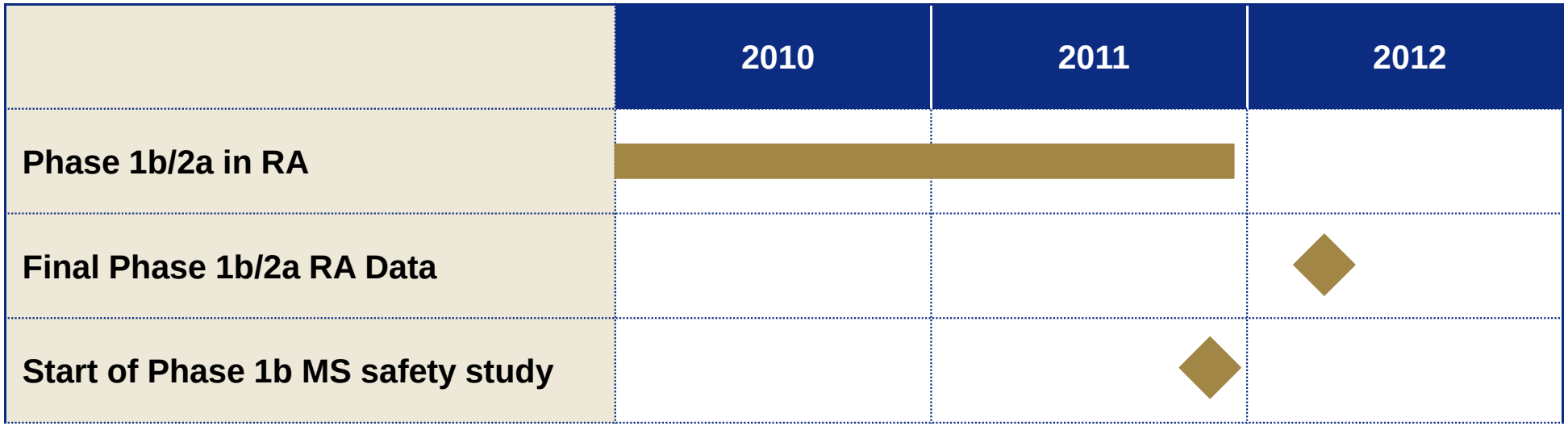
Significantly decreased cumulative EAE score following treatment with MOR103 cf. IgG1 control (at 50mg/kg)

Rationale for Selecting Multiple Sclerosis as Second Indication for MOR103

- Strong scientific rationale based on literature and in-house data
  - Elevated levels of GM-CSF correlate with active phase of MS
  - GM-CSF$^{-/-}$ mice do not develop EAE
  - Anti-GM-CSF ameliorates EAE in GM-CSF wt mice
  - MOR103 reduces cumulative EAE score in GM-CSF wt rats
- Unmet medical need in MS
  - None of currently marketed MS drugs fully control and alleviate symptoms
  - Specifically high unmet medical need in progressive forms of MS
- Commercial opportunity
  - High-price and growing market
  - Drugs in MS with blockbuster potential
  - Interest from potential Licensees
- Next steps
  - Prepare protocol and CTA application for Phase 1b safety study
  - Initiate Phase 1b safety study in Q4/2011

MOR103 – Phase 1b/2a (MSC-1001) in Rheumatoid Arthritis – Clinical Trial Update

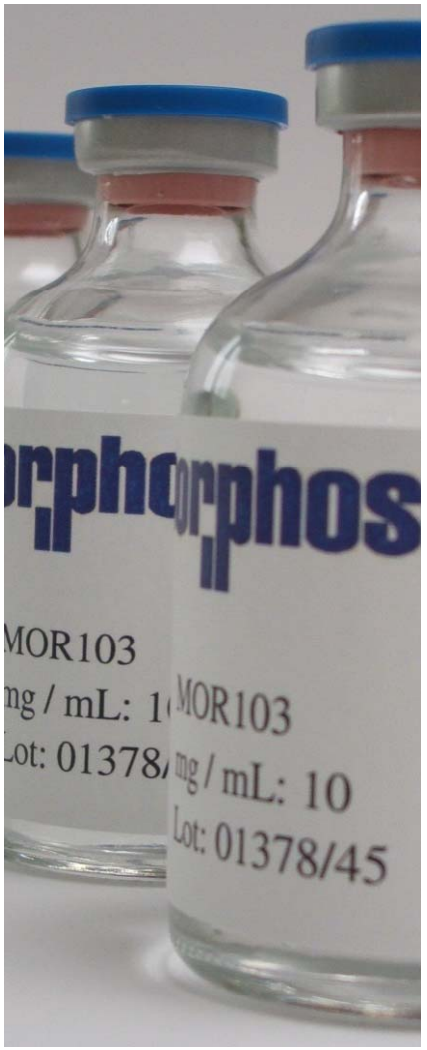
- Phase 1b/2a in patients with active rheumatoid arthritis ongoing
- Final Phase 1b/2a RA data expected in H1 2012
- Start Phase 1b safety study in multiple sclerosis planned in Q4 2011
- Subcutaneous administration development ongoing (SQ PK study in healthy volunteers planned in 2011)



MOR103 Has a Strong Competitive Profile

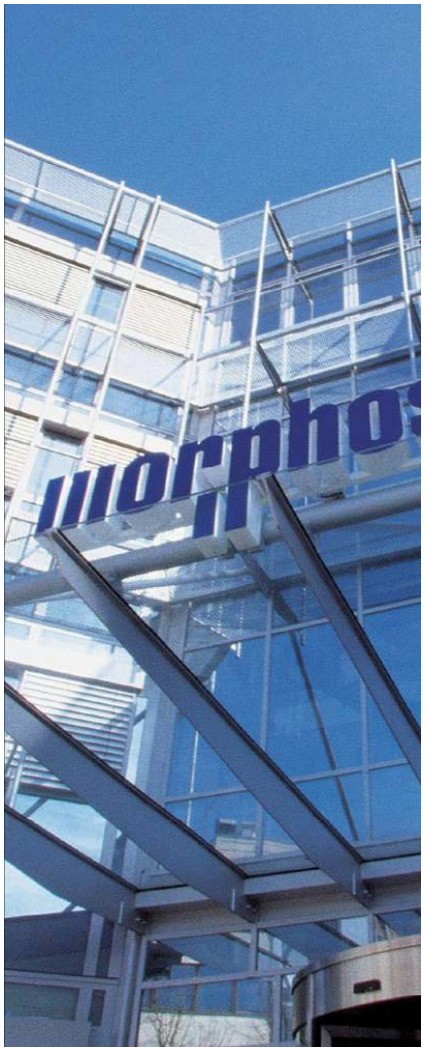
- MOR103 is a HuCAL antibody
  - Fully human, high affinity to GM-CSF, may lead to low cost of goods
  - Fully human antibodies have a lower risk of immunogenicity
  - Subcutaneous administration
- Neutralizing soluble GM-CSF instead of the receptor seen as an advantage
- MorphoSys has a solid patent position around the program
  - Exclusive access to a US patent from the University of Melbourne: Patent generically covers the treatment of chronic inflammatory disorders via GM-CSF antibodies
  - Worldwide patents and patent applications covering the antibody specifically

Blockbuster potential in multiple indications supported by a broad preclinical data package



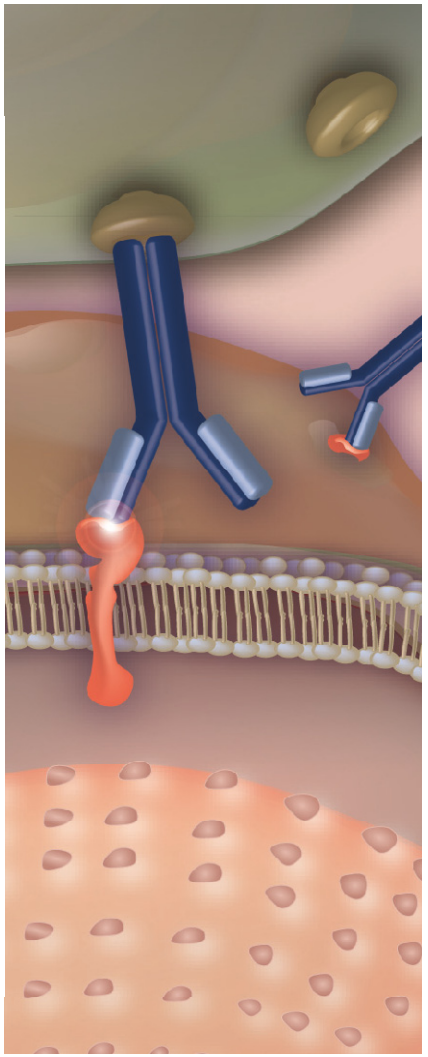
Agenda

09:30	Simon Moroney	Welcome and Introduction
09:35	Marlies Sproll	Latest Technology Developments
		Update Partnered Pipeline
10:05	Q&A	
10:20	Arndt Schottelius:	Introduction Proprietary Portfolio
10:25	John Hamilton	GM-CSF – Central Mediator in Inflammation
10:55	Arndt Schottelius	MOR103 in RA & MS
11:10	Q&A	
11:25	**Lisa Rojkjaer**	**MOR202 in Multiple Myeloma (MM)**
		MOR208 in Chronic Lymphocytic Leukemia (CLL)
11:55	Q&A	
12:15	Simon Moroney	Closing Remarks



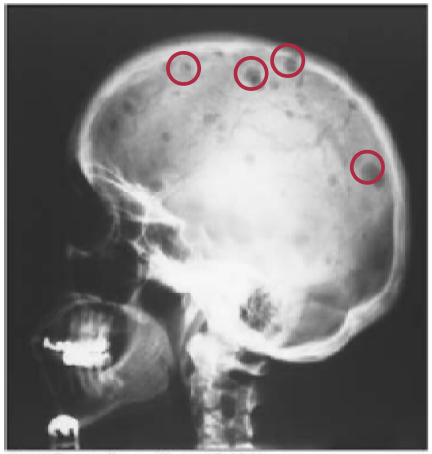
MOR202 Antibody Features

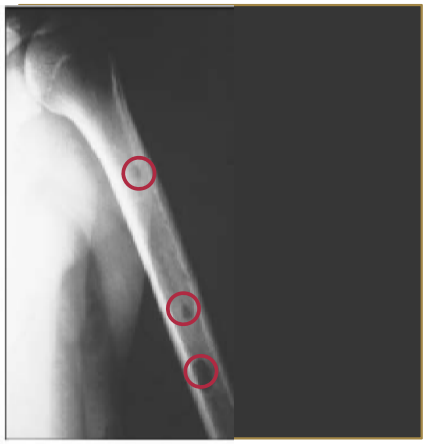
Description	■ HuCAL-derived, fully human, monoclonal IgG1 antibody directed towards human CD38
Potency	■ EC50 ~ 100-300 pM (in ADCC)
Preclinical Results	■ MOR202 induces killing of multiple myeloma cell lines and primary cells via ADCC ■ Demonstrates broad activity against myeloma cell lines *in vitro* and in numerous *in vivo* myeloma models ■ Comparable binding and activity in NHP enabled toxicology studies ■ Robust toxicology and PK data support entry into clinical trials
Development Status	■ CTA filed in November 2010



Multiple Myeloma: Plasma Cell Malignancy With High Unmet Medical Need

- Second most common hematological malignancy after NHL
- Estimated 5-year survival of 38%[1]
- Key disease features: anemia, kidney failure, bony lesions
- Characterized by disease relapses and increasing drug resistance
- Sales of leading therapies are combined €2.5 billion+ worldwide and are growing
- Malignant plasma cells highly express CD38, making MOR202 (anti-CD38) an ideal candidate to evaluate
- Competition: Two human CD38 molecules are in phase 1 development





[1]*Sirohi B, et al. Lancet 2004;363:875-878*

CD38 is a Key Target in Myeloma

What is CD38?

- Type II-transmembrane glycoprotein
- Belongs to family of ectoenzymes/receptors involved in catabolism of extracellular nucleotides

Function

- ADP-Ribosyl-cyclase/cADPR-hydrolase
- Receptor for CD31

Present on tumors and normal cells

- Plasma cells, B/T lymphocytes
- Monocytes/macrophages, thymocytes, NK cells

Highly expressed on MM cells

- Utilized to induce ADCC

CD38 Expression in Hematologic Disorders

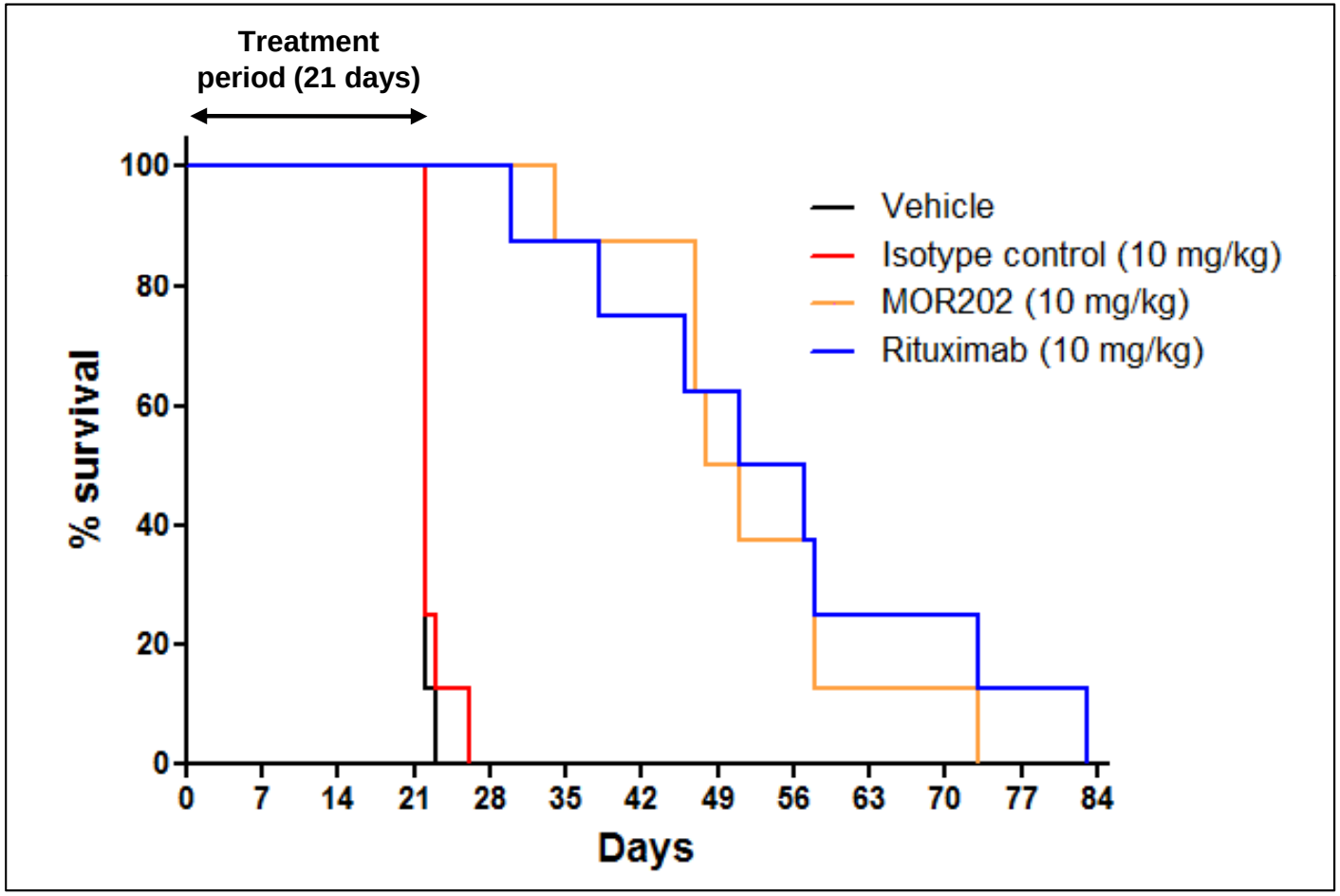
Indication	CD38+ patients	Incidence (7-MM)
MM	~ 98%¥	48,000
CLL	19 – 46%*	33,000
AML	0 – 75%*	27,000
ALL	75%*	12,000
CML	~ 60%*	11,000
Waldenstrom's (NHL)	48%*	2,000-4,000
PTLD**	High	3,000-6,000

* *considered CD38-positive when 20 or 30% of cells exhibit CD38-expression (Meta analysis; CLL: 13 publications; ALL; AML; CML: 2 publications; NHL: 1 publication)*

** *PTLD = post-transplant lymphoproliferative disorders*

¥ *>90% of MM cells are positive for CD38*

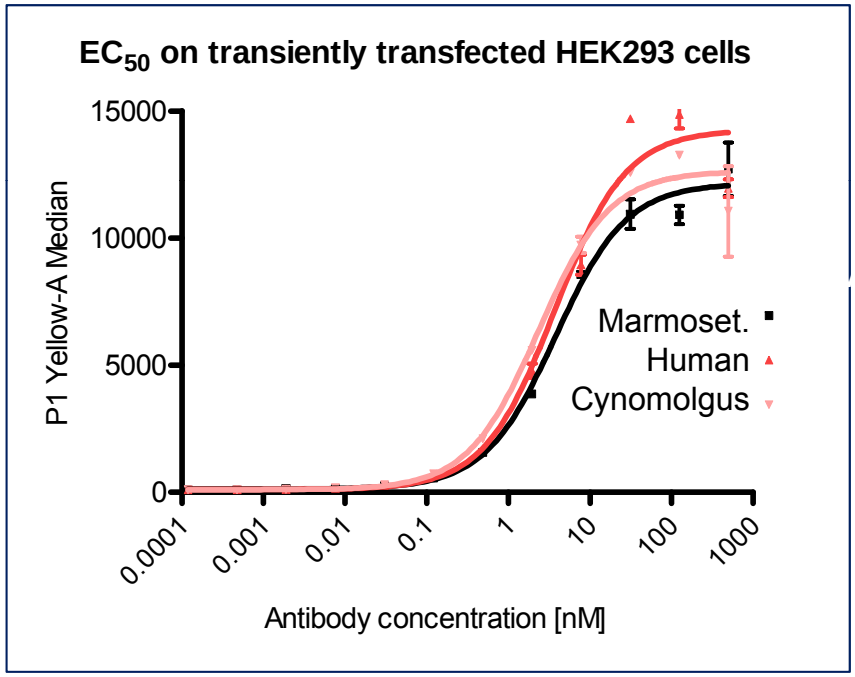
MOR202 Increases Survival in a Mouse Xenograft Model of Lymphoma



Ramos cells inoculated i.v. on day 0; 8 mice per group
Treatment started 3 days post inoculation

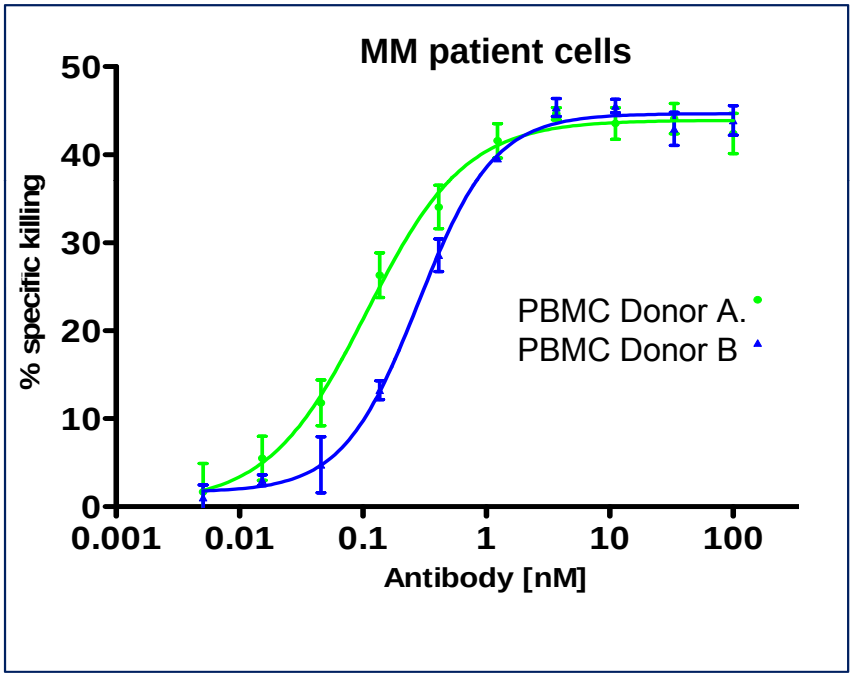
MOR202 Binds to CD38 and Induces ADCC-Mediated Lysis of Myeloma Cells

Cell Binding



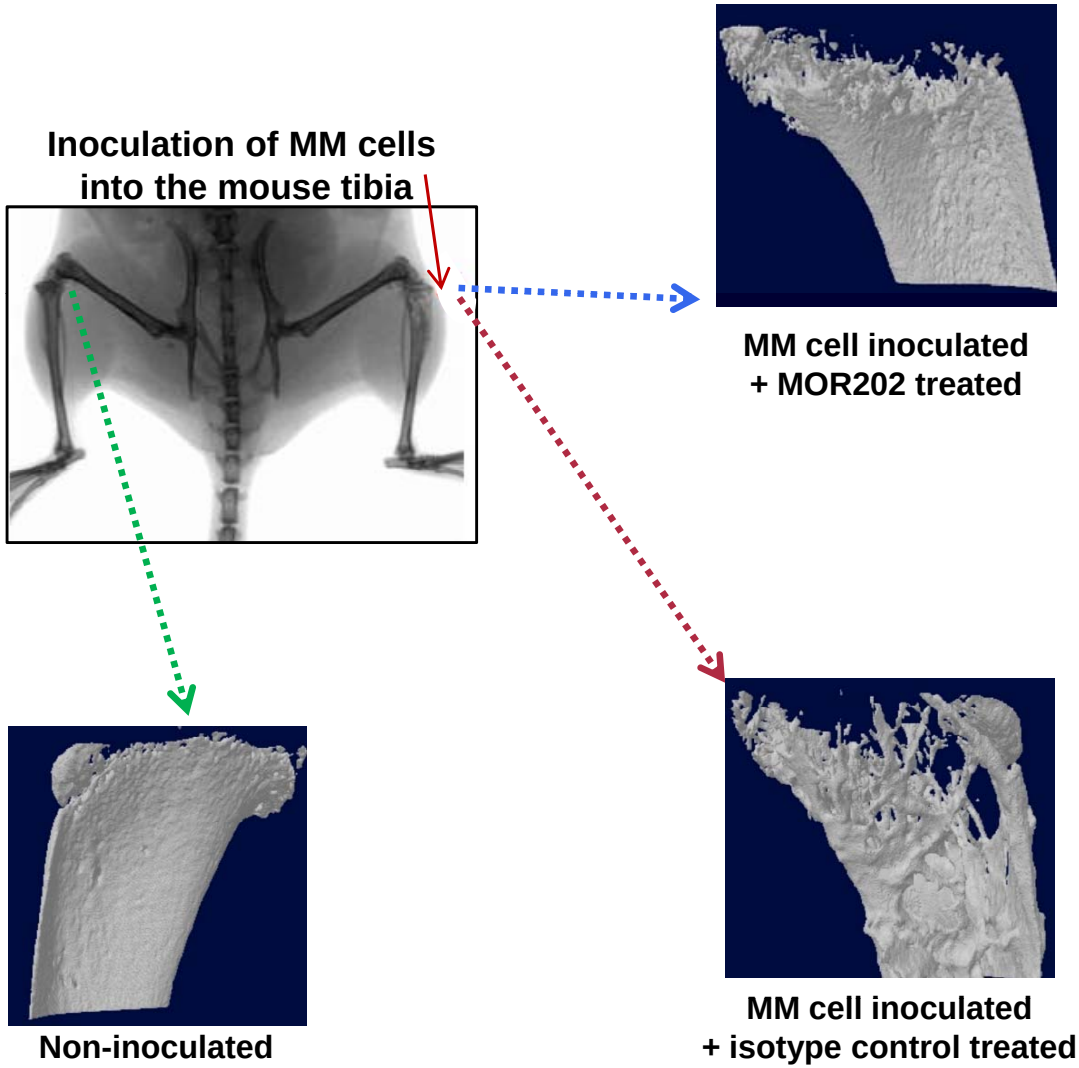
EC_{50}: ~ 3 nM

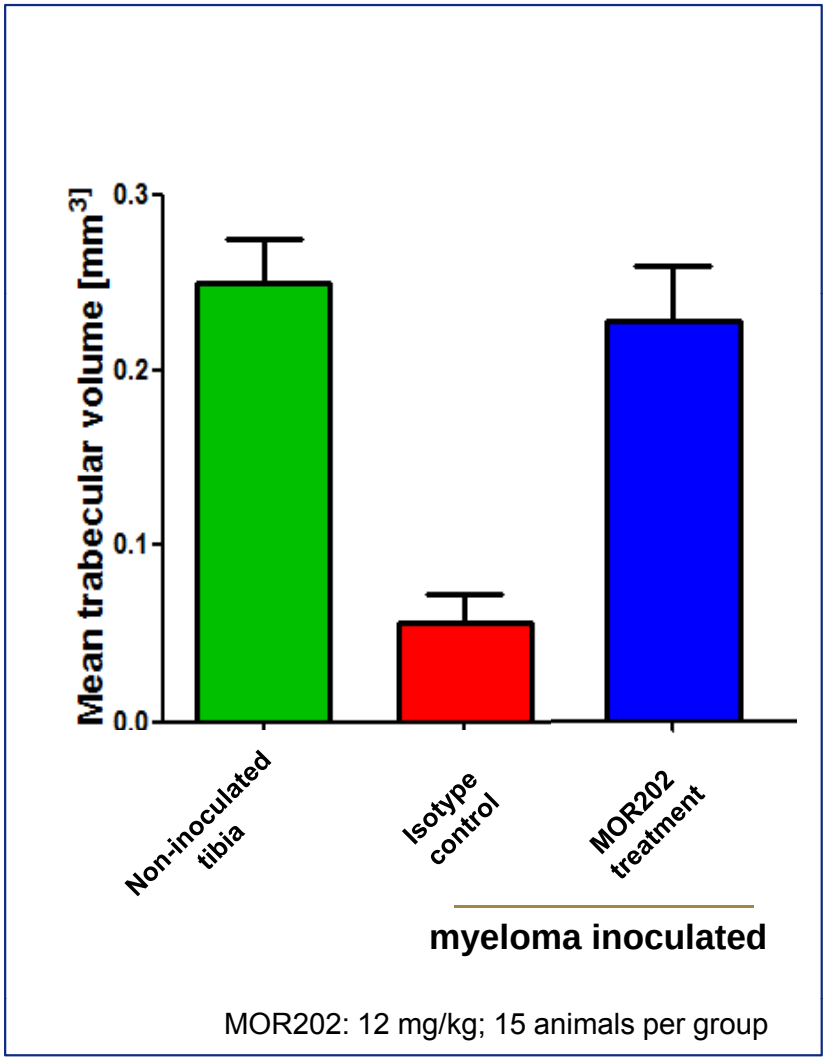
ADCC



EC_{50}: ~ 100 – 300 pM

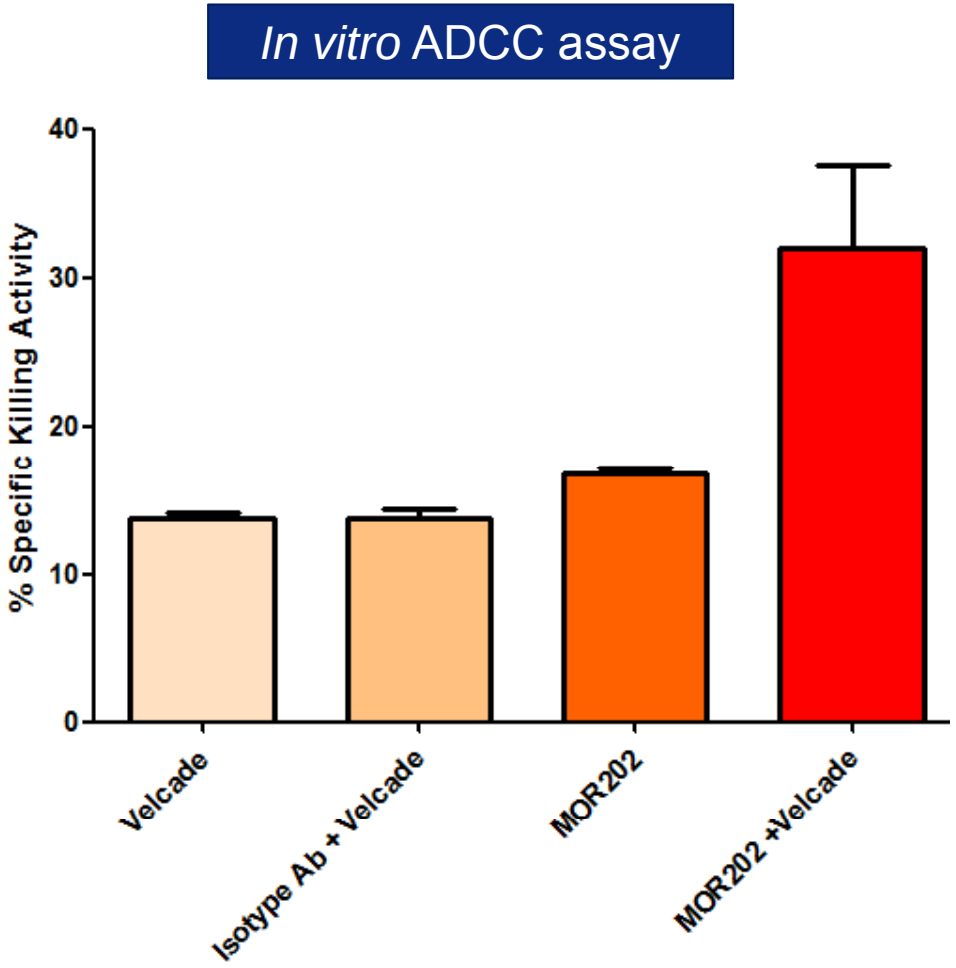
MOR202 Inhibits Bone Lysis in an Orthotopic Mouse Model of Multiple Myeloma





Cytotoxicity of MOR202 is Enhanced by Velcade

- Use of targeted therapy in combination with standard regimens in myeloma can:
  - Minimize adverse events while increasing efficacy through different mechanisms
  - Add flexibility to treatment regimens (e.g. in elderly patients)
- Preclinical investigations are on-going to identify potential combination partners for MOR202



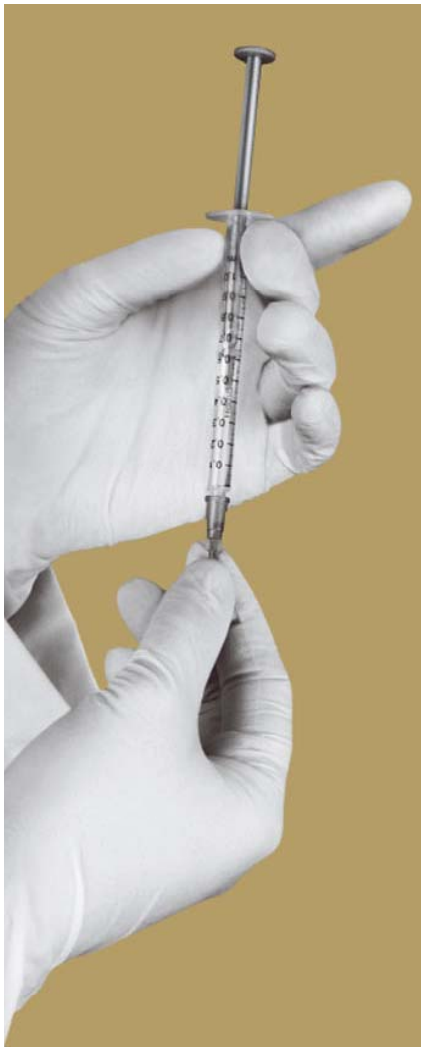
10µg/ml MOR202 and 4nM Bortezomib

MOR202 Well Tolerated in 12-week Repeated Dose Toxicity Study in Non-Human Primates

- Versatility of HuCAL technology enables generation of antibodies that are cross-reactive, allowing safety assessment in animals to optimize development
- Marmoset monkey identified as relevant NHP species
  - High target sequence identity
  - Similar binding of MOR202 to human and marmoset CD38 (shown)
  - MOR202 mode of action (ADCC) present in marmoset with similar activity *ex vivo*
- Tox study conclusions
  - Safety profile supports entry into clinical trials
  - Dose-proportionality of PK parameters observed

MOR202 – Phase 1/2a Trial

- Design
  - Multicentre, open-label, dose-escalation study (EU)
- Population
  - Patients with relapsed/refractory multiple myeloma; failure of at least 2 prior therapies
- Objectives:
  - Investigate maximum tolerated dose, safety and tolerability
  - Pharmacokinetics and immunogenicity
  - Assess preliminary activity
- **Enrollment of first patient expected in H1 2011**



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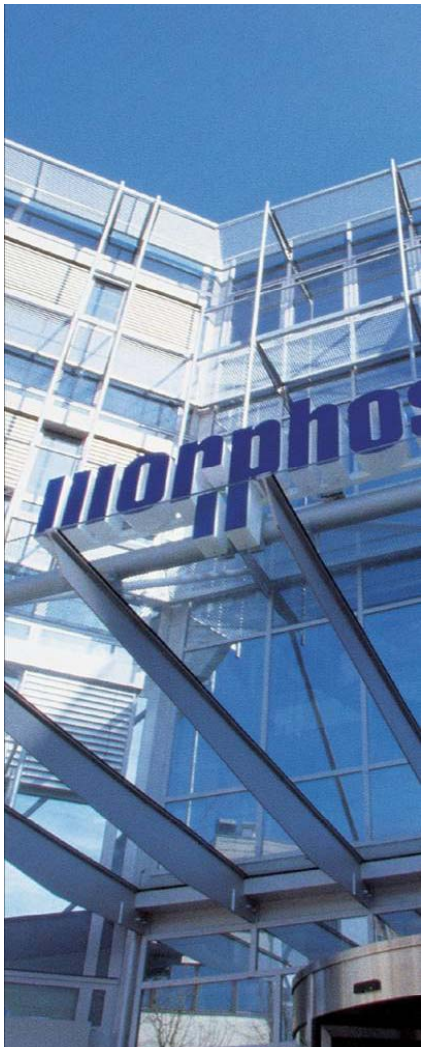
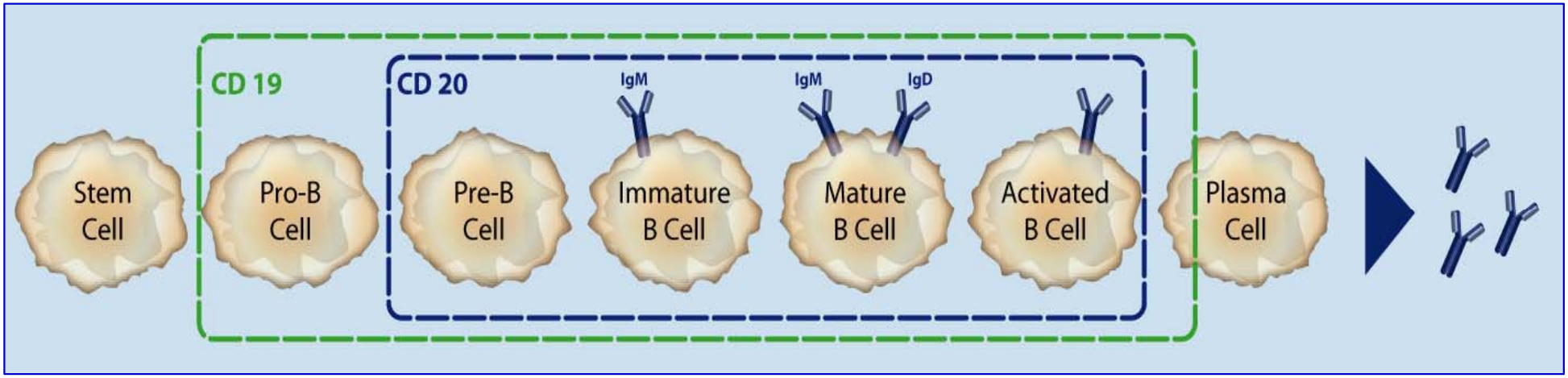


Anti-CD19 Antibodies Hold Promise for the Treatment of B-cell Malignancies

- Broader expression of CD19 in B-cell tumors compared to CD20
- Potential for use in patients with rituximab resistance
- Encouraging clinical data with CD19 bispecific antibody in NHL and ALL validates CD19 as a new target



Success of B-cell depletion in lymphoma opens many possibilities for novel regimens

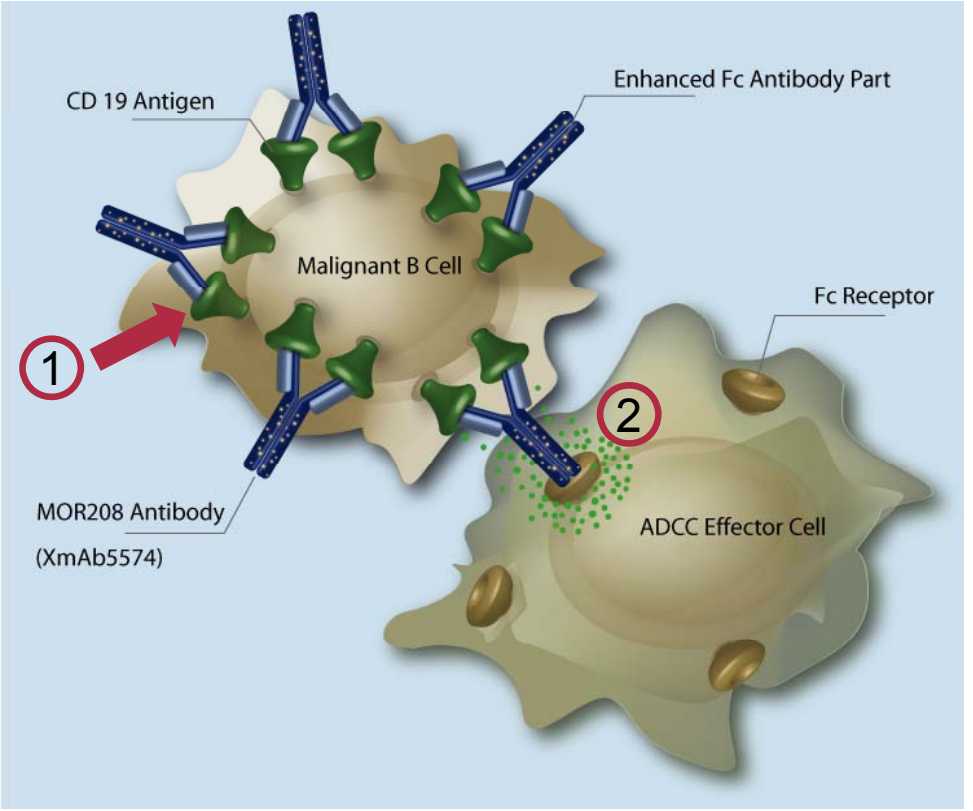
Antitumor Mechanisms are Mediated by IgG – Fc Receptor Interactions

Two interactions are key for mAb activity:

1. mAb binding to the tumor-associated antigen
2. Binding of Fc receptor (FcR) on effector cell (e.g. NK cell) to the Fc portion of the mAb

Immune effector-mediated mechanisms are important for mAb-mediated cytotoxicity:

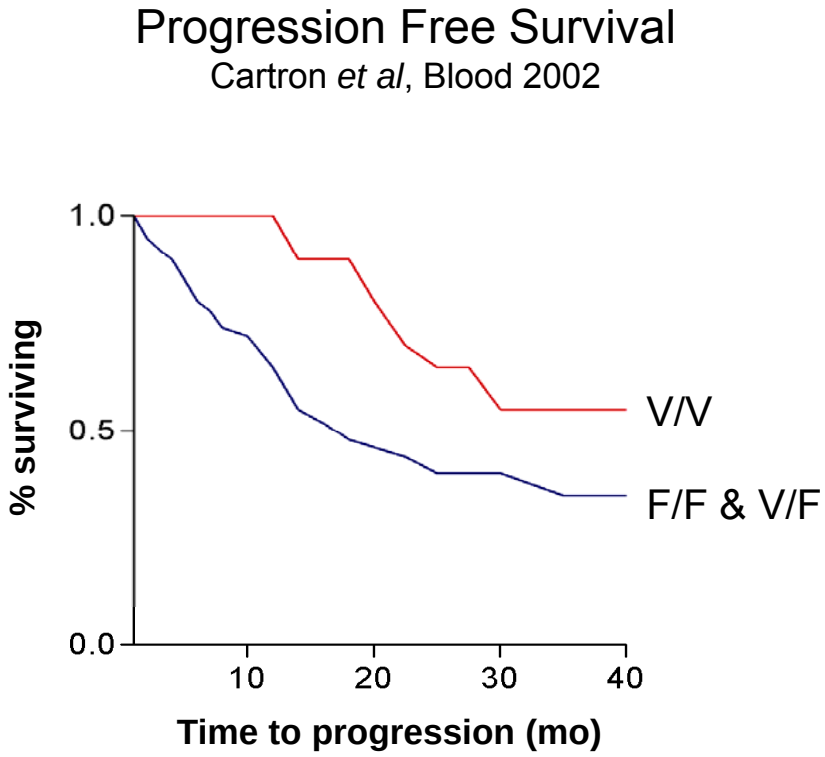
- Fc receptors on leucocytes (FcγR) mediate several effector functions (e.g. phagocytosis, ADCC) against Ab-bound targets



MOR208/XmAb5574: Fc Optimization for Increased Tumor Killing

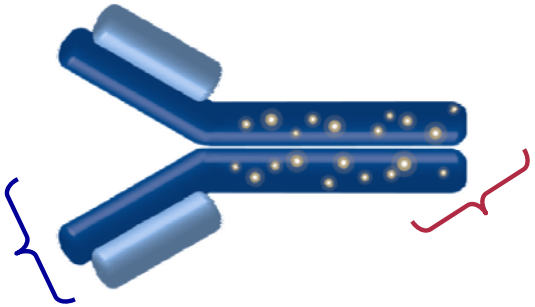
Higher FcγR Affinity Correlates with Better Clinical Response to Rituximab

- NK cell FcγRIIIa receptors are important for rituximab-dependent cytotoxicity against lymphoma cells
- Two polymorphisms in the FcγRIIIa gene exist: position 158, valine (V) or phenylalanine (F)
  - Affinity for IgG1: V = 5x F
- Response rates (clinical/molecular) to Rituxan were assessed in 49 previously-untreated patients with follicular NHL
- Data demonstrates that stronger IgG binding to the FcγRIIIa receptor improves ADCC



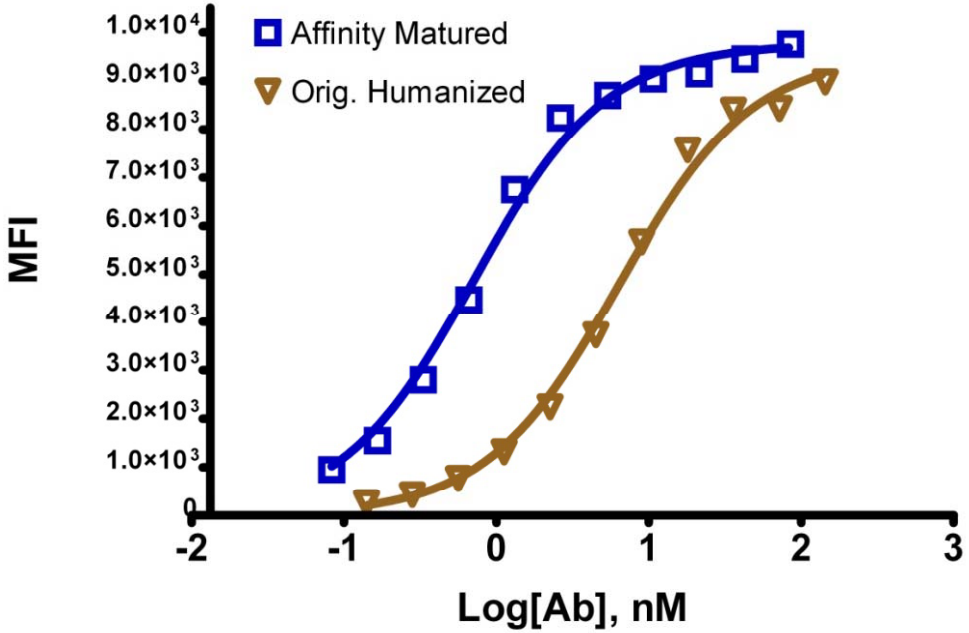
Higher FcγRIIIa affinity → Improved clinical efficacy

MOR208/XmAb5574 Has a Humanized, Affinity Optimized Fv Domain and Enhanced ADCC

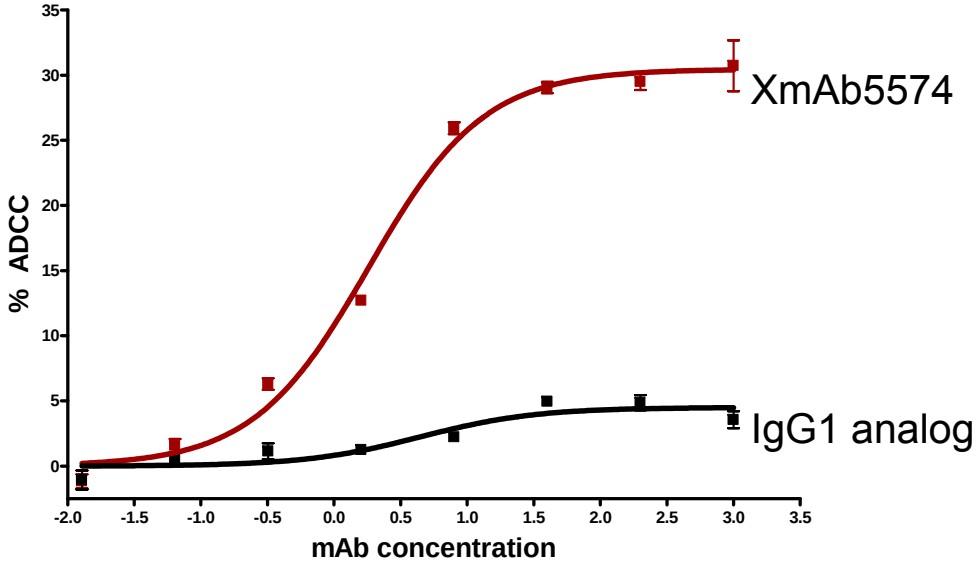


Potent Binding

Enhanced Killing

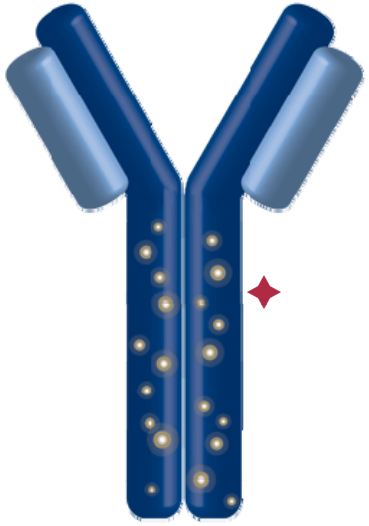


Anti-CD19 Binding to RS4;11 Cells



ADCC with RS4;11 Cells

MOR208/XmAb5574 Has a High ADCC Fc Domain: Enhanced Binding to FcγRIIa and FcγRIIIa

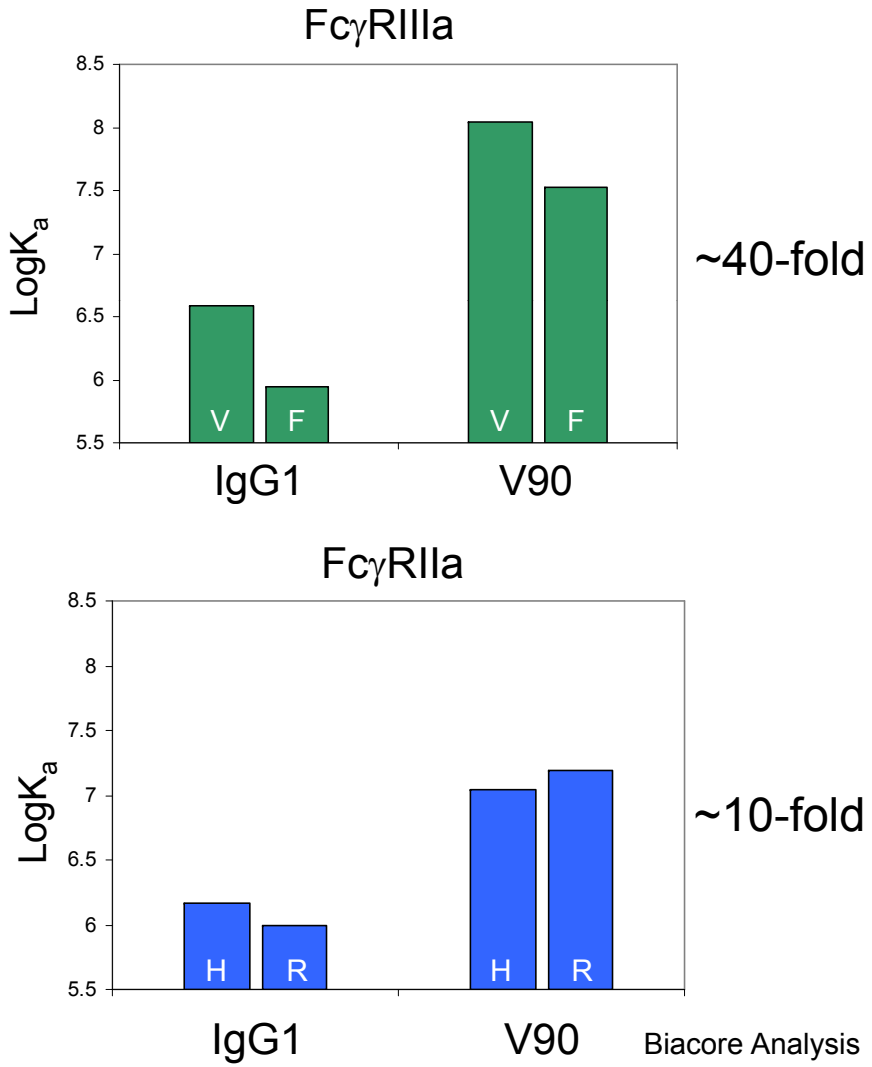


Fc variant

✦V90 – 239D/332E

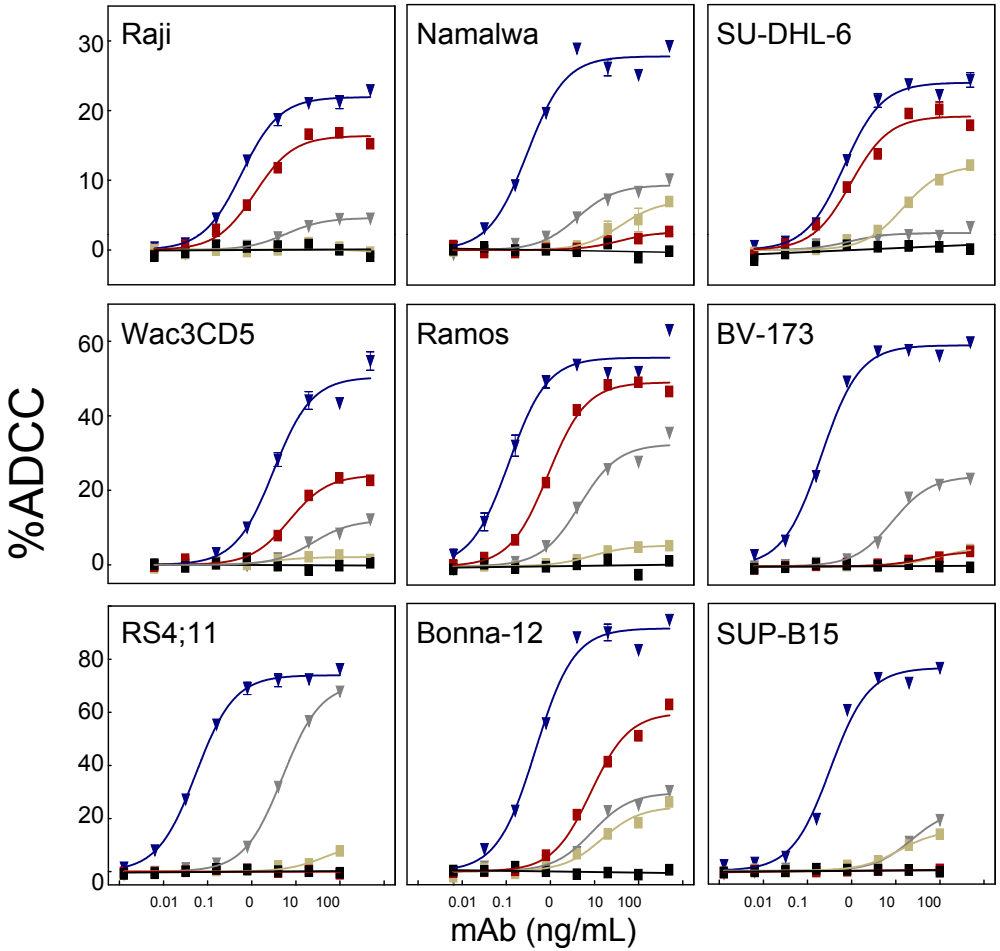
Human validation in XmAb2513 (anti-CD30)

- Well-tolerated (very few adverse events, no maximum tolerated dose)
- Low immunogenicity
- Objective responses in Hodgkin's disease



MOR208/XmAb5574 Has Superior Cytotoxicity

Higher potency of MOR208/XmAb5574 in ADCC



Horton HM et al. Cancer Res 2008;68:8049-57

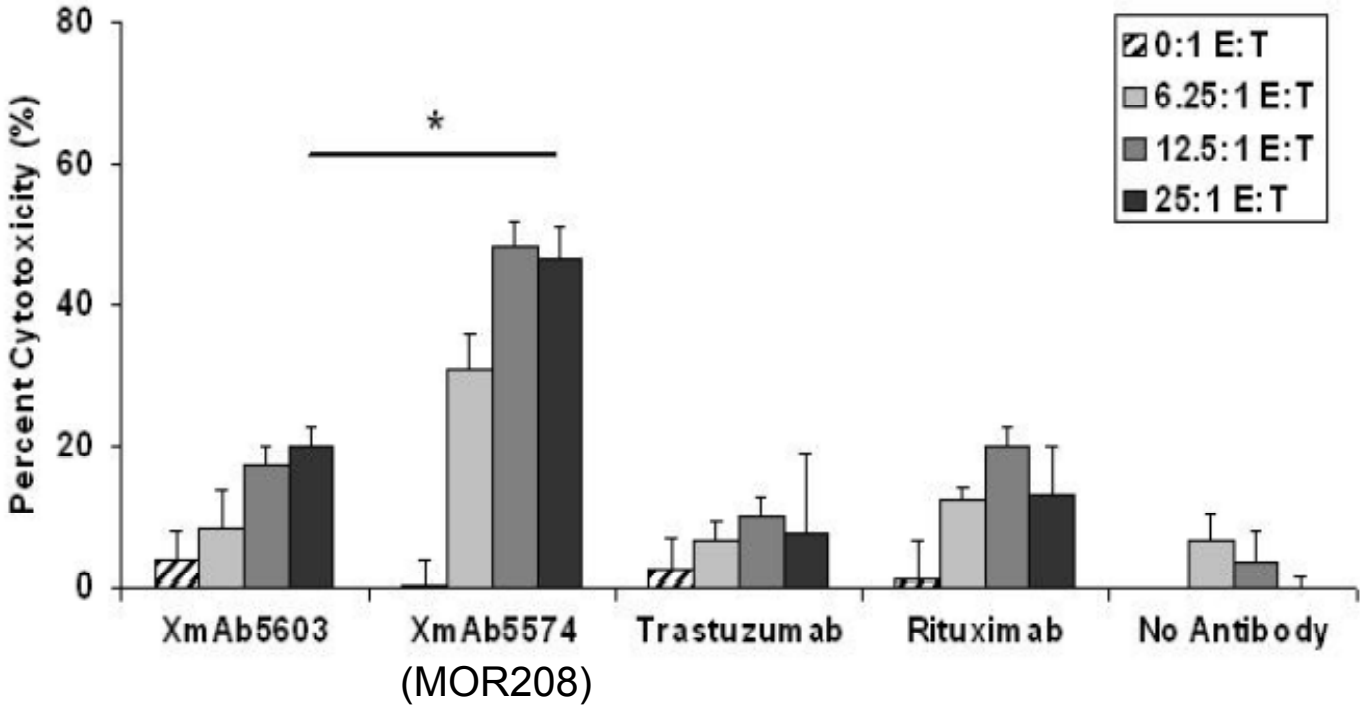
- Highly cytotoxic against a panel of lymphoma and leukemia cell lines
- Higher ADCC than rituximab & alemtuzumab
- Higher ADCC than IgG1 anti-CD19 (unmodified)

Lymphoma/Leukemia	Cell Lines
B-NHL	SU-DHL-6
Burkitt's Lymphoma	Raji, Ramos, Namalwa
CLL	Wa3CD5
CML	BV-173
HCL	Bonna-12
ALL	SUP-B15, RS4;11

MOR208/XmAb5574
Anti-CD19 IgG1 (unmodified)
XmAb (-) control
rituximab (CD20)
alemtuzumab (CD52)

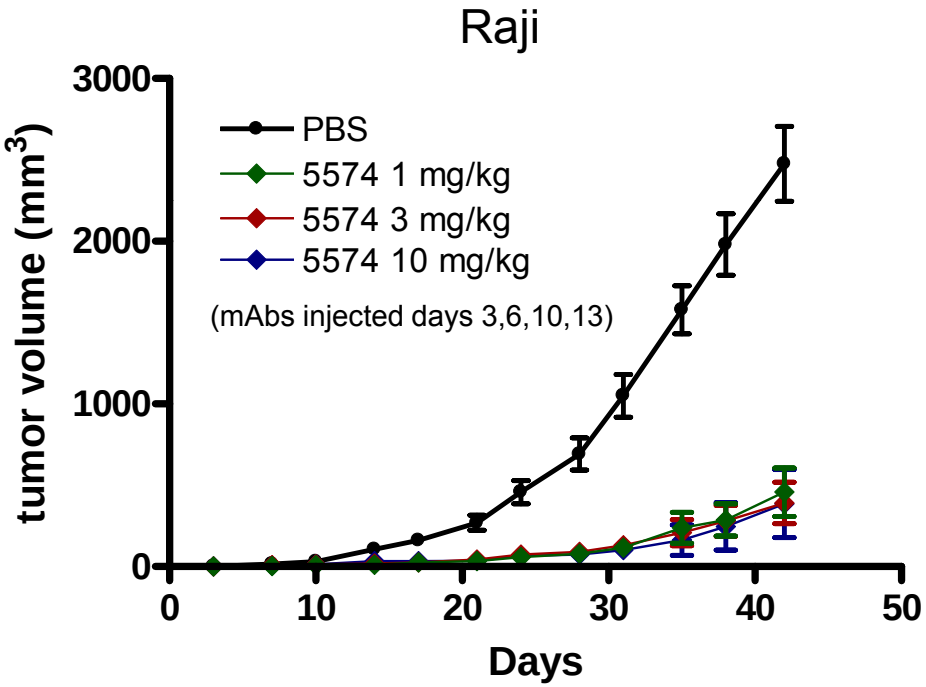
MOR208/XmAb5574 Induces Potent ADCC Against Patient-Derived B-CLL Cells

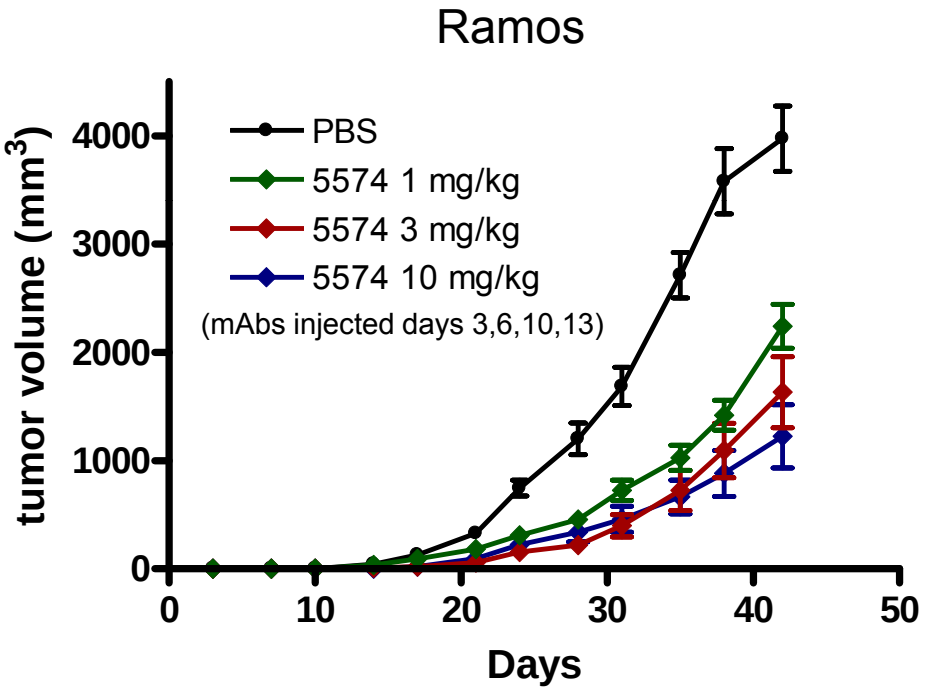
NK-mediated ADCC against patient CLL cells



Awan et al., Blood 2010

MOR208/XmAb5574 Prevents the Growth of Subcutaneous Lymphoma Tumors

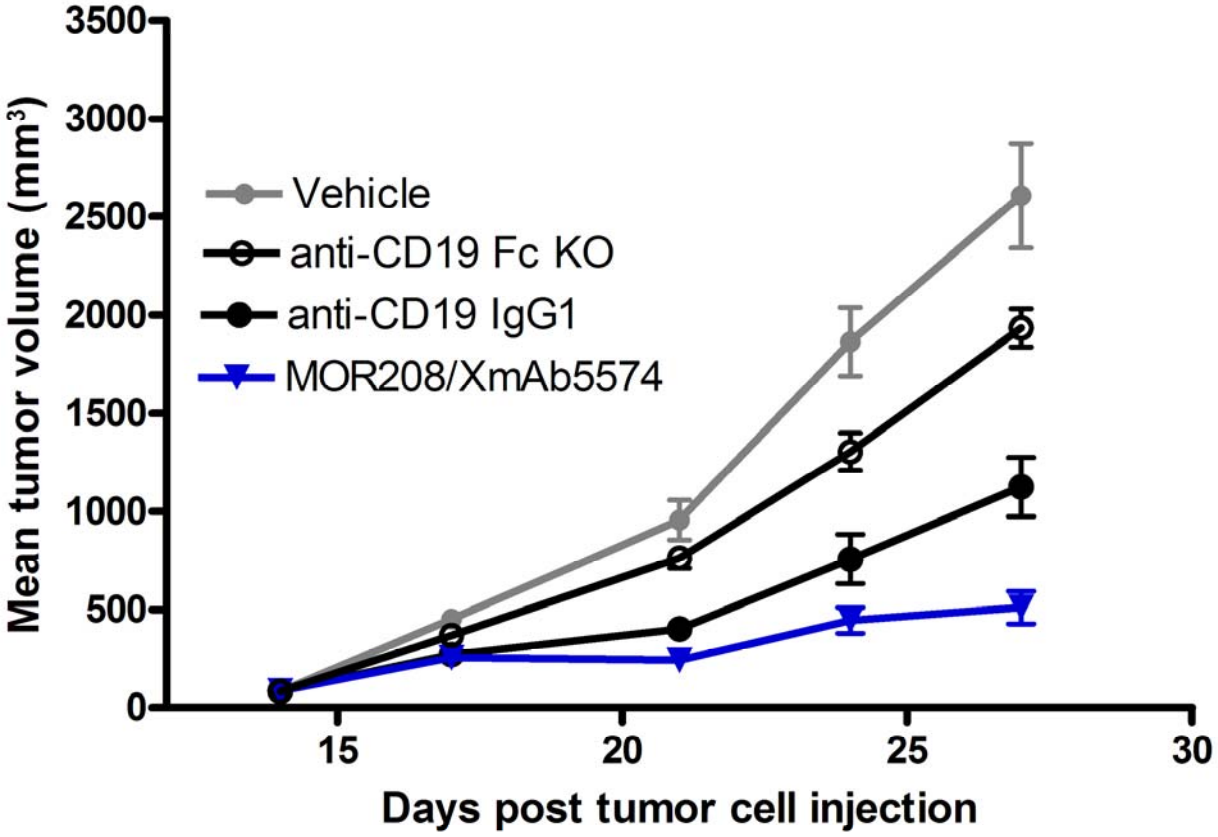




Horton HM et al. Cancer Res 2008;68:8049-57

Human tumor xenograft in SCID mice;
Tumor inoculation points = 0; 10 mice per group

Efficacy Against Established Tumors is FcγR-Dependent and Enhanced by Fc Engineering



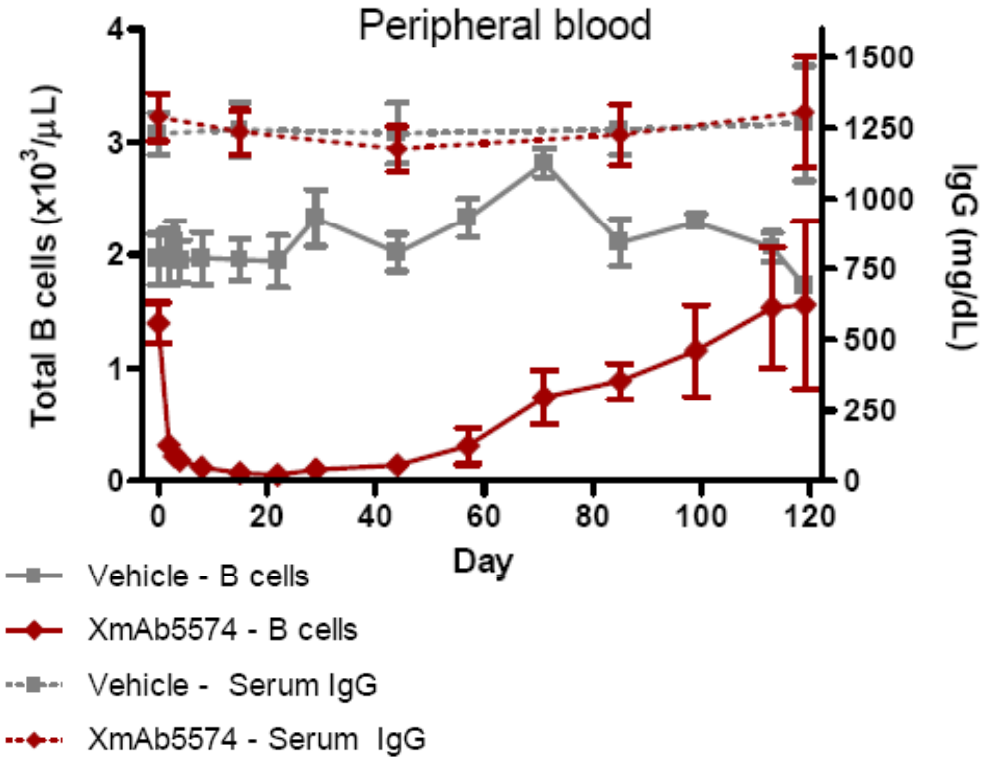
- Human tumor xenograft in SCID mice
- 40-120 mm^3 Ramos tumors at start of Ab treatment
- 10 mg/kg Ab, 2x/week for 3 weeks, 10 mice/group

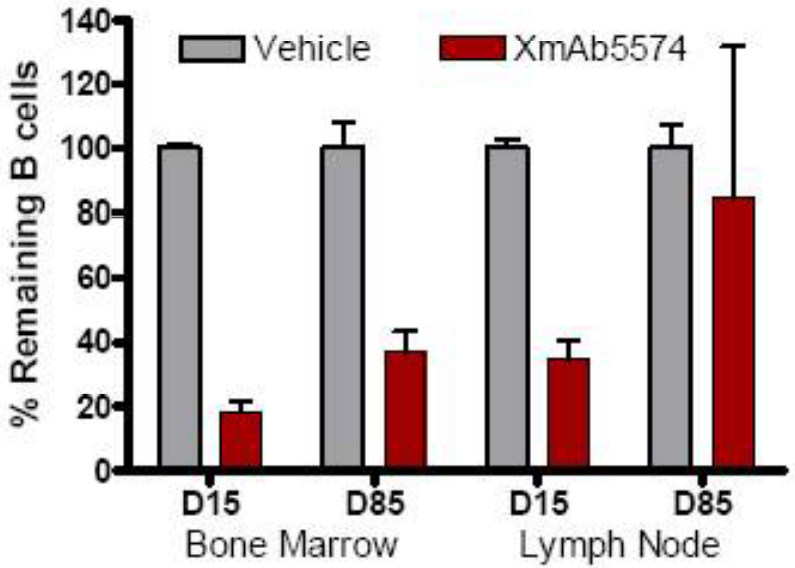
Horton HM et al. Cancer Res 2008;68:8049-57

anti-CD19 Fc KO:	full length anti-CD19 with two mutations that remove FcγR binding
anti-CD19 IgG1:	unmodified IgG1

MOR208/XmAb5574 Depletes B cells in Cynomolgus Monkeys

Single 10 mg/kg dose; N=6



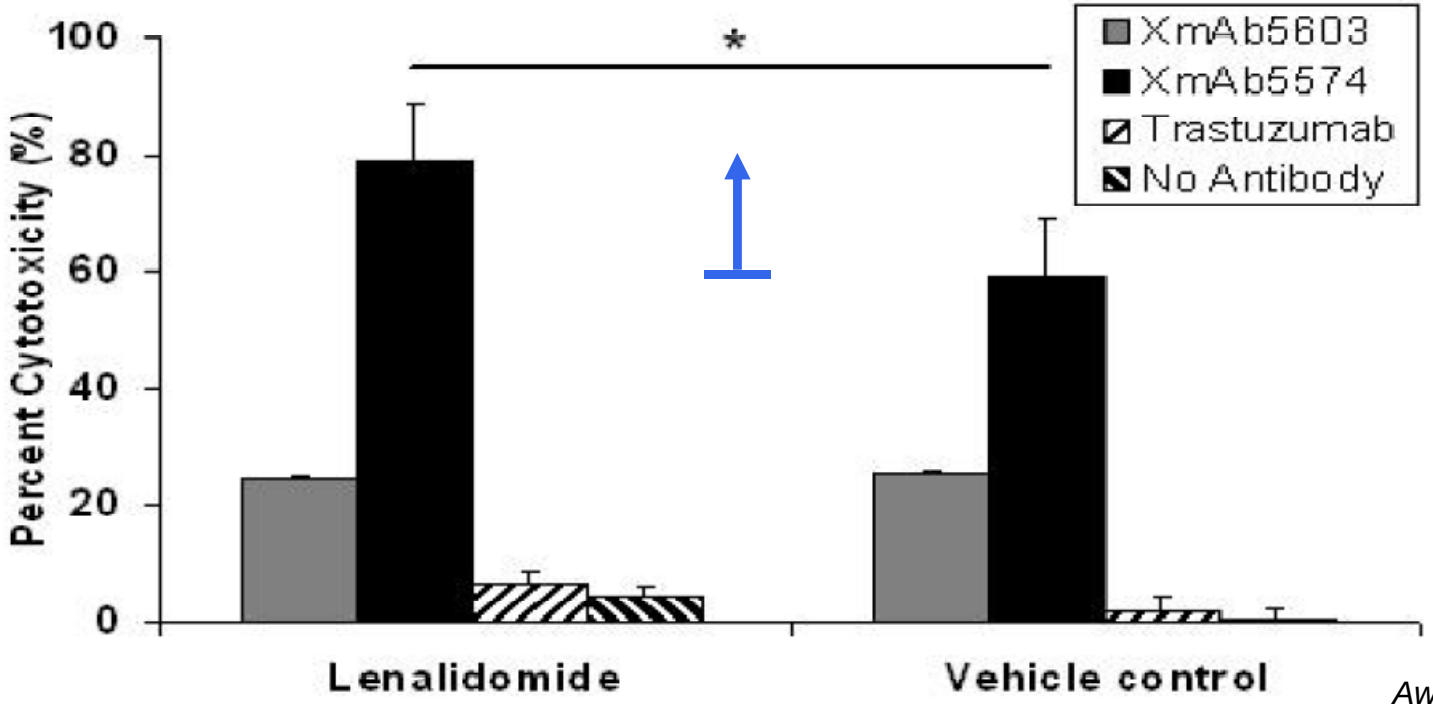


Zalevsky J et al. Blood 2009;113:3735-3743

MOR208/XmAb5574 showed efficient B cell depletion in peripheral blood and lymph nodes, while not decreasing serum IgG levels

Lenalidomide Significantly Enhances ADCC Activity of MOR208/XmAb5574

- Lenalidomide is an immunomodulating agent, approved for relapsed myeloma
- Reported to enhance T cell and NK cell activity
- Active in relapsed/refractory CLL

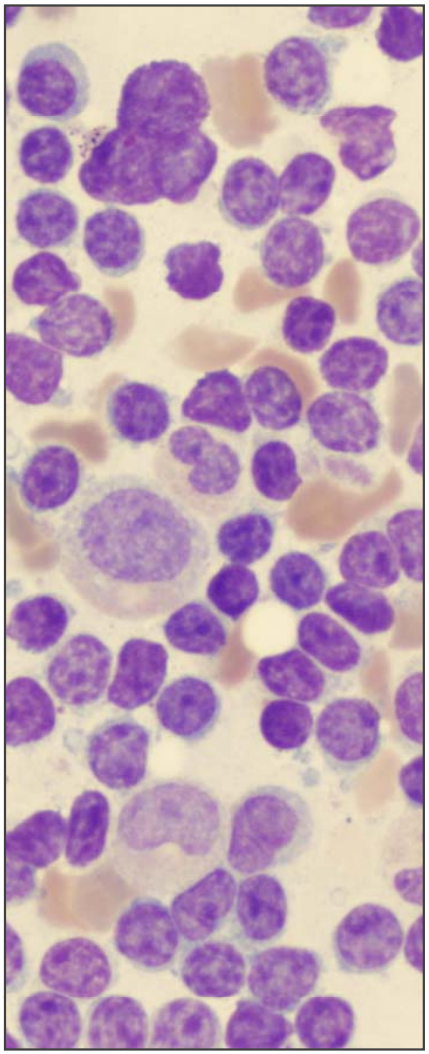


Awan et al., Blood 2010

Potential for novel combination regimens with MOR208/XmAb5574 in CLL therapy

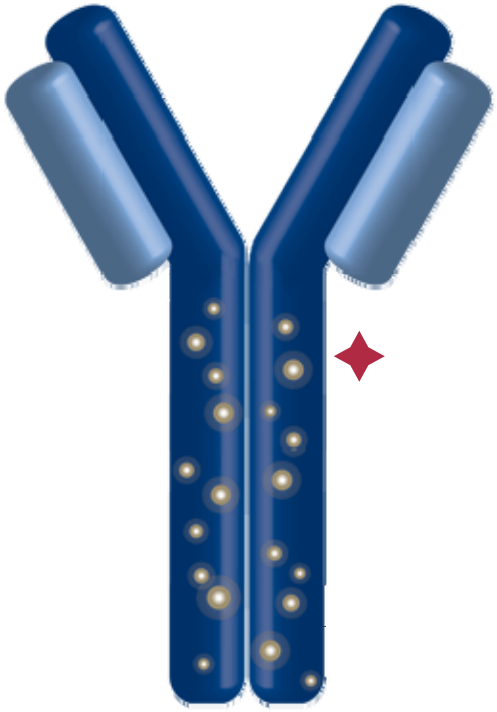
Potential Benefits of Targeting CD19

- In CLL: CD19 antigen density per cell is significantly higher than CD20
  (D'Arena G et al, Leuk Lymphoma 2001;42:649-654)
- Ability to target and deplete pre-B and immature B cells in the bone marrow, as well as peripheral mature B-cells
  (Yazawa N, 2005)
  - May translate into more durable peripheral B cell depletion
- B-cell depletion in lymph nodes demonstrated *in vivo*
  (Schroeder, 2003; Cioc, 2008, Cavallo, 2008)
- Loss of CD20 expression following Rituxan treatment is reported in up to 50%, with persistence of CD19 positivity
  (Chu, 2002; Seliem, 2006)
- Anti-CD19 antibodies have efficacy against Rituxan-resistant cell lines *in vitro*
  (Gerber, 2009)
- CD19 antibodies in development show preliminary efficacy in rituximab refractory/resistant patients



MOR208/XmAb5574 is Competitive in the CD19 Antibody Space

- Expected half life favoring convenient dosing schedule
- i.v. administration
- Straightforward manufacturing
- Potential for good safety profile
- Embraces a number of effector mechanisms
  - Significantly increased ADCC compared to rituximab *in vitro*
  - Enhanced FcγRIIa affinity may enhance macrophage function, and result in improved tissue B-cell depletion



Fc domain modification of MOR208/XmAb5574 embraces a broader range of effector mechanisms

MOR208/XmAb5574 – Phase 1 Trial

Design

- Multicentre, open-label, multi-dose, single-arm phase 1, dose-escalation study (USA)

Population

- Patients with CLL/SLL, who have not responded to or have become refractory to previous therapies

Objectives

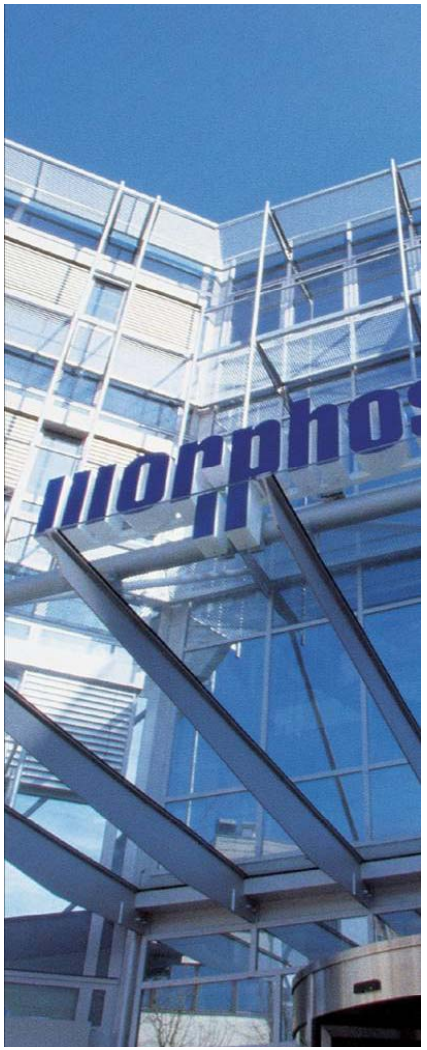
- Investigate maximum tolerated dose, safety and tolerability
- Pharmacokinetics and immunogenicity
- Assess preliminary anti-tumor activity

Clinical Data

- Final data will become available in 2012

Agenda

09:30	Simon Moroney	Welcome and Introduction
09:35	Marlies Sproll	Latest Technology Developments
		Update Partnered Pipeline
10:05	Q&A	
10:20	Arndt Schottelius	Introduction Proprietary Portfolio
10:25	John Hamilton	GM-CSF – Central Mediator in Inflammation
10:55	Arndt Schottelius	MOR103 in RA & MS
11:10	Q&A	
11:25	Lisa Rojkjaer	MOR202 in Multiple Myeloma (MM)
		MOR208 in Chronic Lymphocytic Leukemia (CLL)
11:55	Q&A	
12:15	**Simon Moroney**	**Closing Remarks**



Therapeutic Antibodies: A Star Still Rising

- Opportunity for therapeutic antibodies continues to grow
- Increasing understanding is guiding us to new ways of making superior therapeutic antibodies
- The future will see
  - Antibodies with much greater efficacy
  - …against new target classes
  - …being adopted in new indications
  - …with much lower cost-of-goods
  - and more…

MorphoSys aims to command and apply a range of technologies to deliver innovative new antibody-based drugs

Strategy and How it Relates to Today's Agenda

PROPRIETARY DEVELOPMENT

- Carefully selected programs in cancer & inflammation
- …taken by MorphoSys to clinical proof-of-concept
- …promise even greater upside

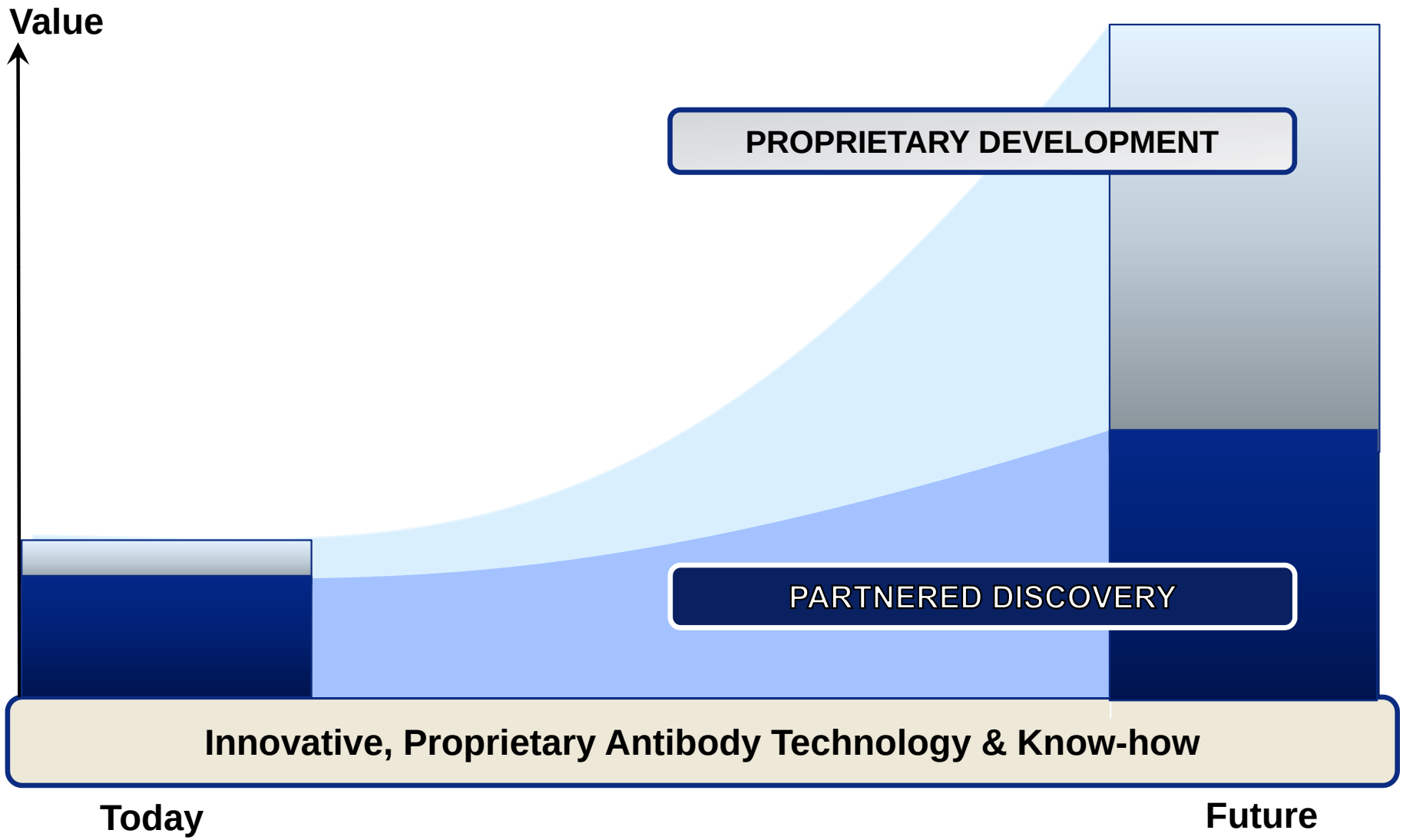
PARTNERED DISCOVERY

- A growing number of projects against partners' targets
- …promises to result in multiple marketed drugs
- …delivering a lucrative flow of milestone and royalty payments

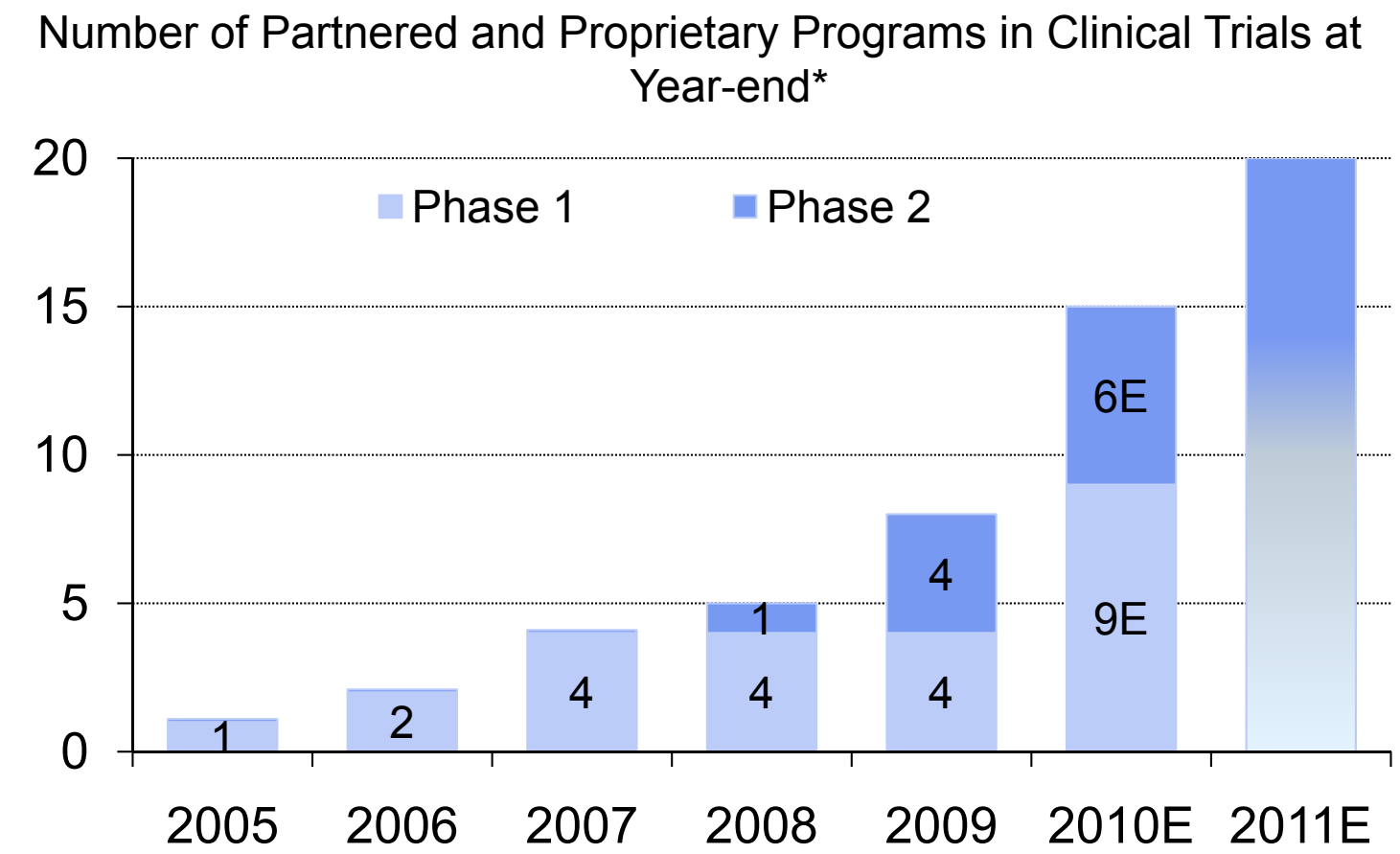
Innovative, Proprietary Antibody Technology & Know-how

- Fulfilling a growing demand for technologies which can deliver superior antibodies
- …more efficacious, against new target types, better producible
- …extending the therapeutic reach and performance of antibodies

A Strategy for Substantial Value Creation



Great Progress of Clinical Pipeline



Clinical Antibody Pipeline is a Key Value Driver

* assuming no attrition

Thank You

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