R&D Update

April 10, 2014

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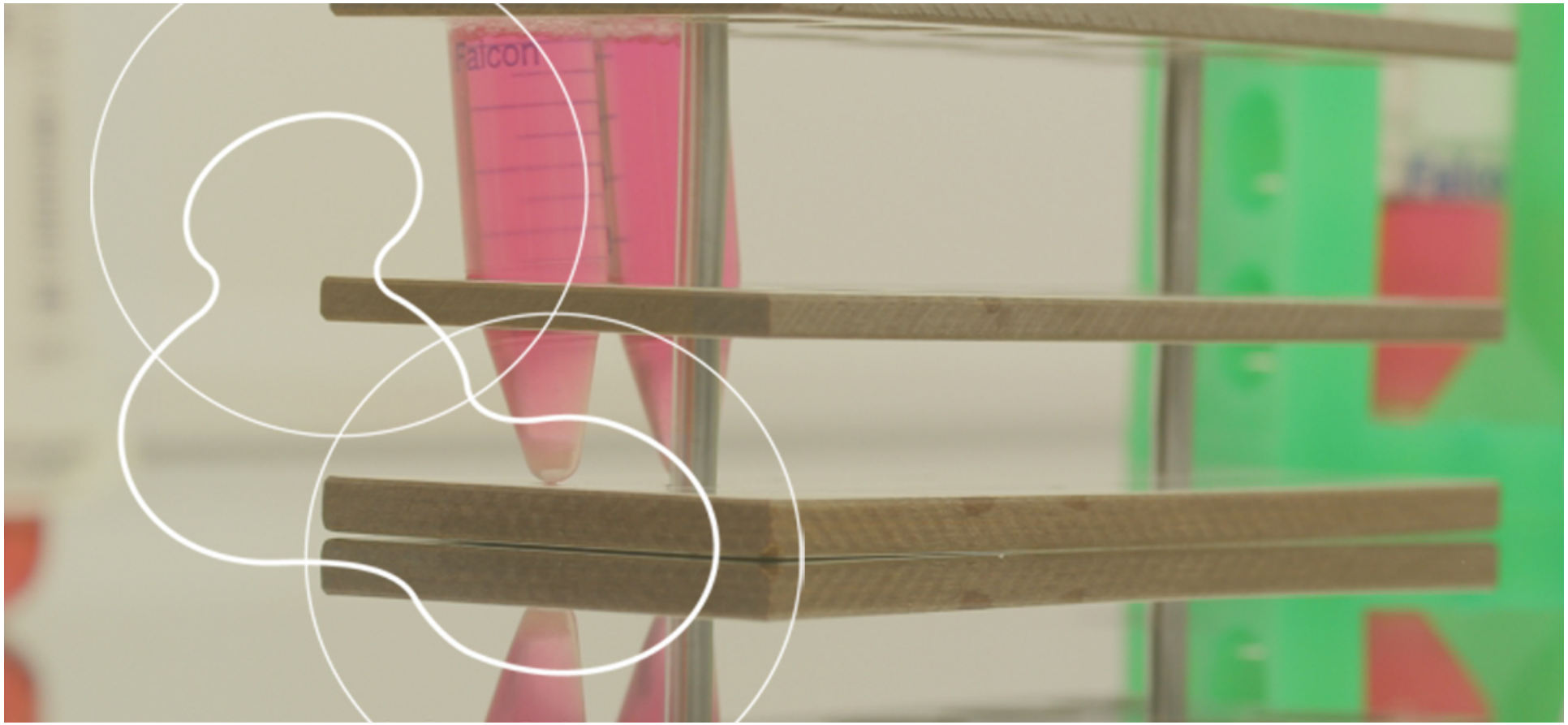
Agenda

- Introduction
  Hervé Brailly, CEO
- R&D portfolio update
  Nicolai Wagtmann, CSO
- Break
- Clinical development update
  Marcel Rozencweig, CMO
- Cash update
  Catherine Moukheibir, EVP Finance



Introduction

Hervé Brailly, CEO & Co-Founder

R&D Update, NY, April 10, 2014

Innate Pharma at a glance

innate pharma

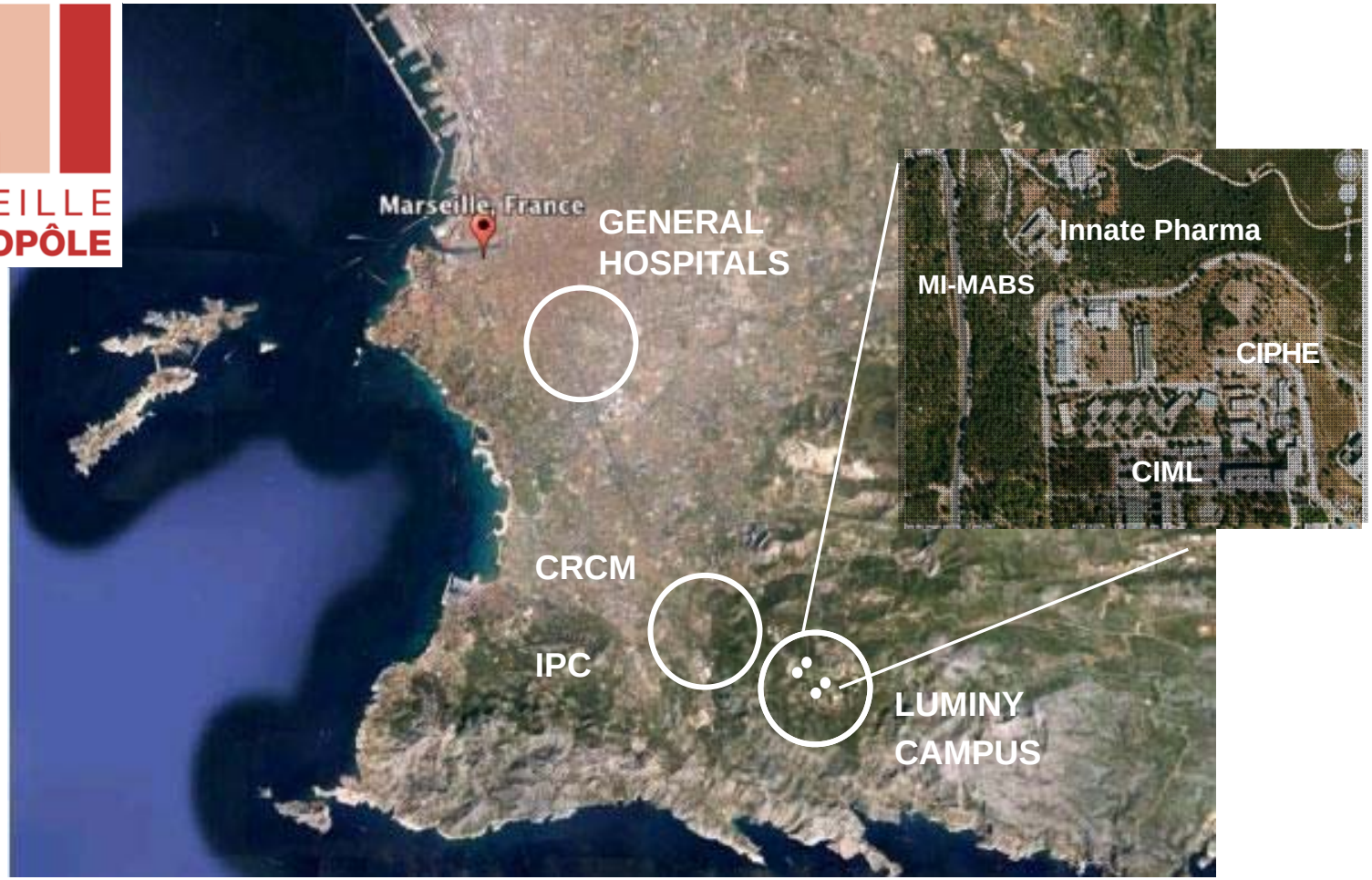
First-in-class immuno-modulating antibodies targeting innate immunity

- At the leading edge of innate immunity pharmacology
- Portfolio of first-in-class immunomodulating mAbs
- Primary focus in immuno-oncology
- Potential for development in chronic Inflammation

Founded in a leading European immunology cluster
A hot spot for immuno-pharmacology

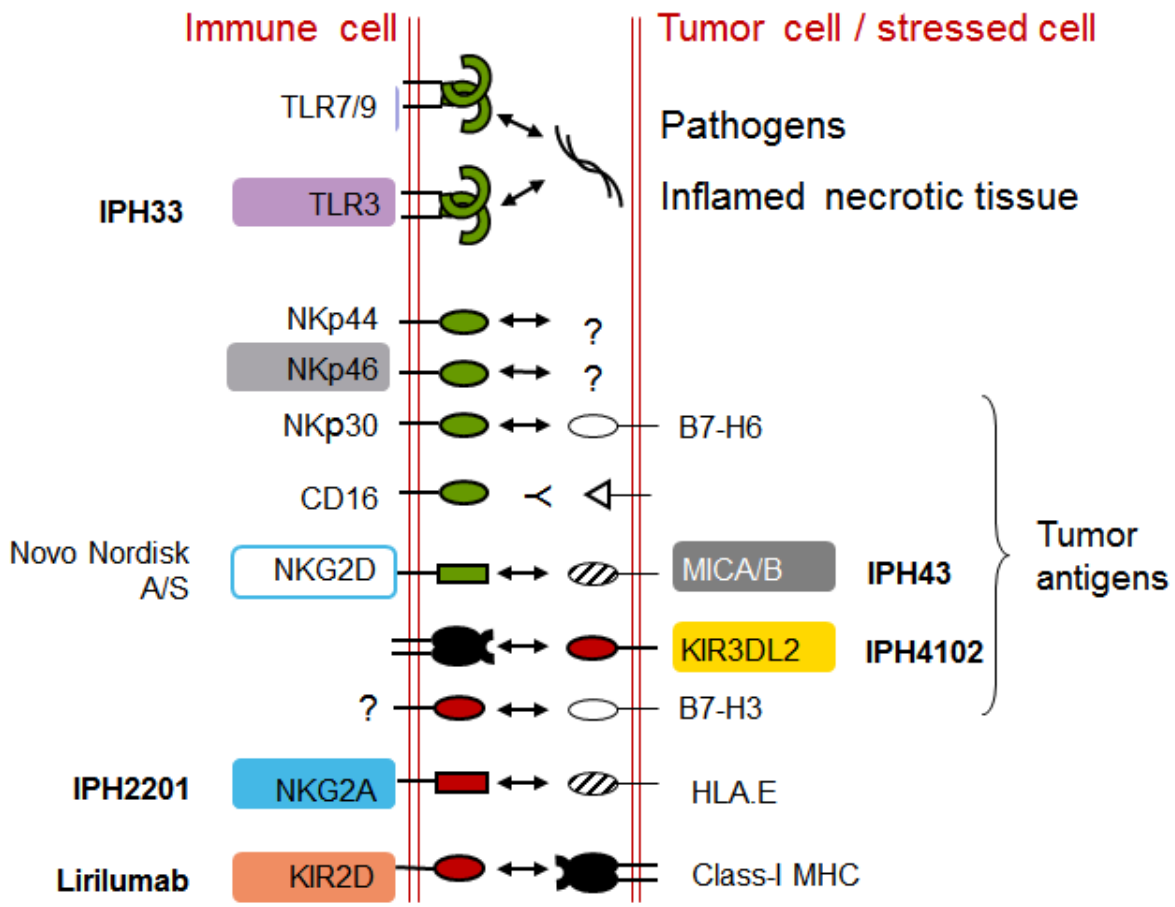
From CTLA-4 cloning (Golstein et al, 1987) to the deciphering of NK cell activation pathways (Vivier et al, late 90's) to anti-KIR first-in-man (2007)

MARSEILLE IMMUNOPÔLE



Addressing innate immunity checkpoints
A unique scientific positioning

- Dual role of NK cells in tumor immunity
  - > Direct killing, control of residual disease
  - > Breaking tolerance to yield T cell response
- Role of NK cells in the maintenance of inflammation



Innate Pharma's pipeline
First-in-class I-O assets

PROGRAM	TARGET	INDICATION	Valid	PC	PI	PII	PIII
Lirilumab (IPH2102/BMS-986015) licensed to **Bristol-Myers Squibb**	KIR2DL1,2,3	Acute Myeloid Leukemia					
		Solid tumors, comb. with ipilimumab					
		Solid tumors, comb. with nivolumab					
IPH2201	NKG2A	Cancer					
IPH4102	KIR3DL2	Cutaneous T-cell lymphomas					
IPH33	TLR3	Inflammation / Autoimmunity					
IPH43	MICA	Cancer					

Innate Pharma positioning in immuno-oncology

	TARGET	PHASE I	PHASE II	PHASE III	MARKET
Checkpoint inhibitors	**CTLA-4**		AZN		BMS
	PD-1 / PD-L1	Merck KGaA, Amplimmune /GSK, BMS	AZN	BMS, Merck, Roche	
	KIR		IPH/BMS		
	LAG-3	BMS	IMP		
	NKG2A	IPH			
Activating receptors	**CD137**	Pfizer	BMS		
	CSF-1R	AMG, LLY, Roche			
	B7-H3	Servier/Macrogenics			
	CD40	Roche			
	OX40	AZN			
	GITR	GITR Inc			
	CD27	Celldex			

Ongoing combination trials of immunomodulating mAbs		
Phase III	CTLA-4 + PD-1	BMS
Phase I	CTLA-4 + KIR	IPH/BMS
Phase I	PD-1 + KIR	IPH/BMS
Phase I	PD-1 + CTLA-4	AZN, Merck
Phase I	CD40 + CTLA-4	U. Penn.
Phase I	PD-1 + LAG-3	BMS

Management team

Hervé Brailly
PhD, **CEO** Co-founder

Immunotech SA, Beckman-Coulter

Nicolai Wagtmann
PhD,
Chief Scientific Officer

Novo Nordisk A/S
>10 years in development in cancer and inflammation

Catherine Moukheibir
MBA, **Sr Adv. Finance**

13 years of experience in strategy and finance for the healthcare industry

Movetis, Zeltia, Morgan Stanley

Marcel Rozencweig
MD, **Chief Medical Officer**

>30 years of experience in clinical drug development

Bristol-Myers Squibb

Jérôme Tiollier
PhD,
Chief Development Officer

>25 years in drug development

Pasteur Merieux Sangstat

Yannis Morel
PhD, **Chief Business Officer**

Innate Pharma

R&D portfolio update

Nicolai Wagtmann, CSO

R&D Update, NY, April 10, 2014

Agenda

I. **Innate Pharma: Unique scientific positioning driving first-in-class pipeline**

II. Lirilumab: A checkpoint inhibitor built on translational clinical data

III. IPH2201: A novel checkpoint inhibitor targeting NK and T cells

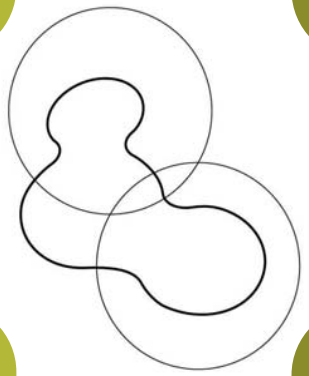
Innate Pharma

A unique combination of targets and expertise

Immuno-pharmacology

High throughput mAb platform

Differentiation



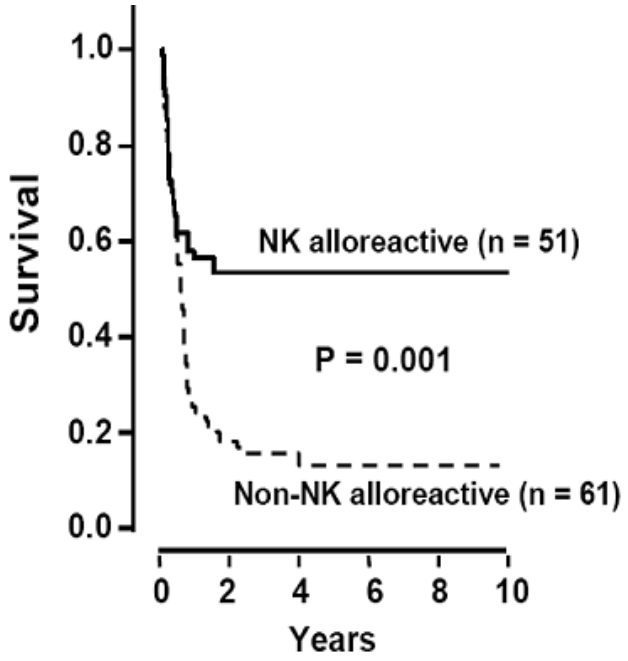
Integration

Innate Immuno-Receptors

Clinical development

Seminal observation on NK cells in tumor immunology

- NK cells can protect against tumor relapse, leading to improved survival in AML patients after stem cell transplantation



NK activation

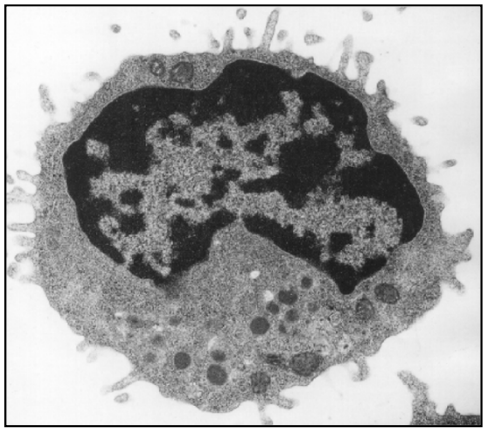
NK inhibition

Effects are:

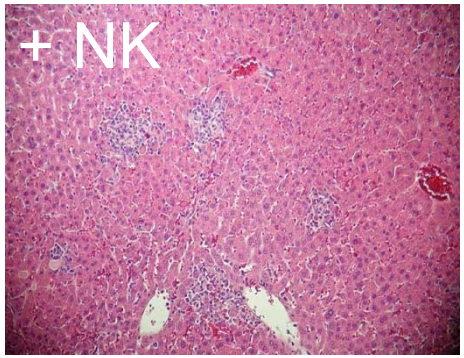
- Durable
- Safe
- Controlled by KIR
- Mediated by NK cells

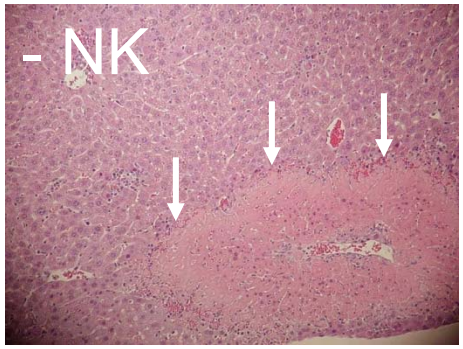
Ruggeri et al, Blood, 2007

NK cell functions



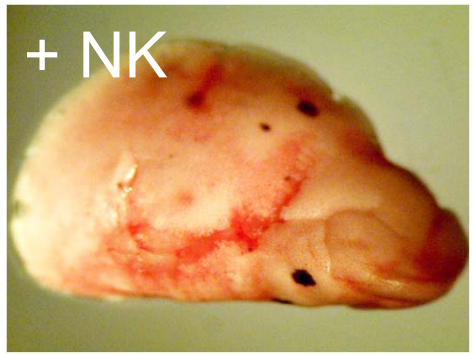
Virus

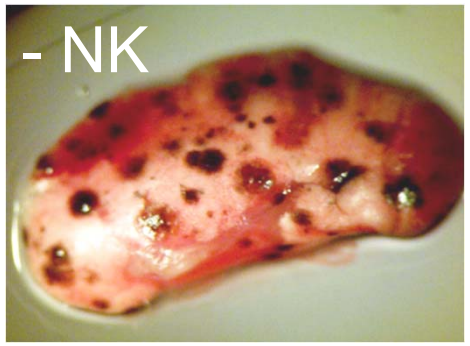




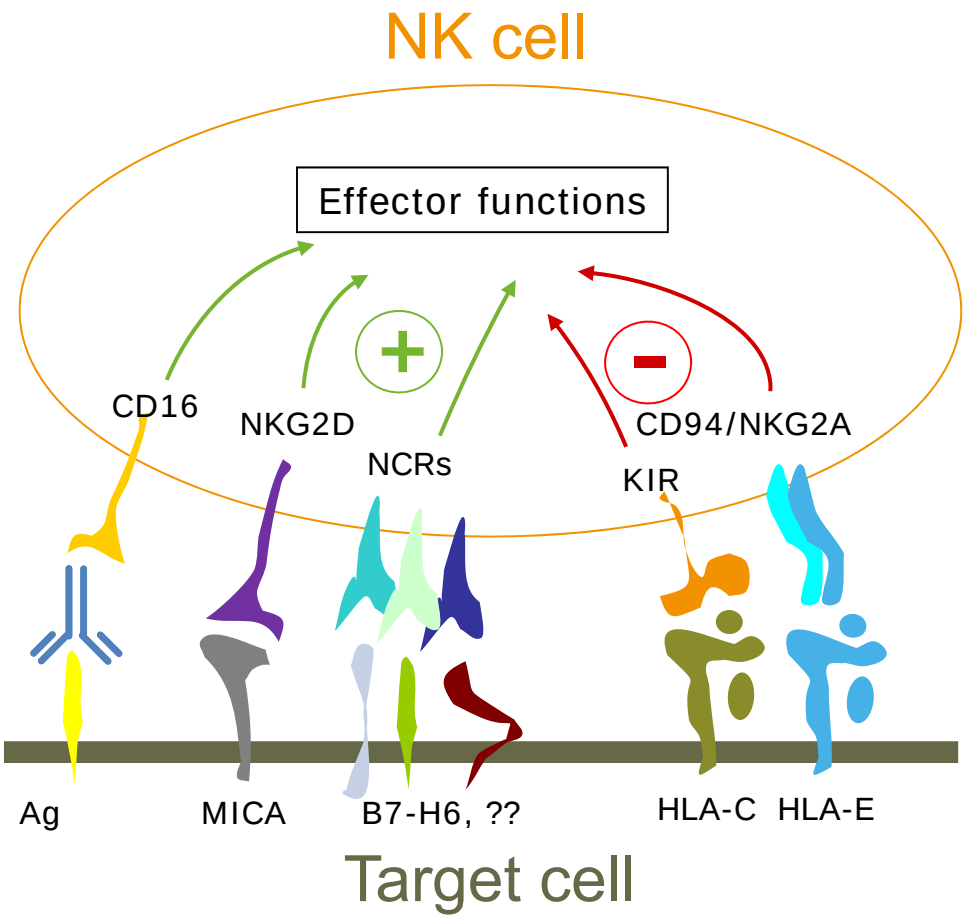
- Directly kill tumor and virally infected cells
- Produce cytokines (e.g. IFN-y) regulating adaptive immunity
- Rapidly activated & potent responses

Tumors



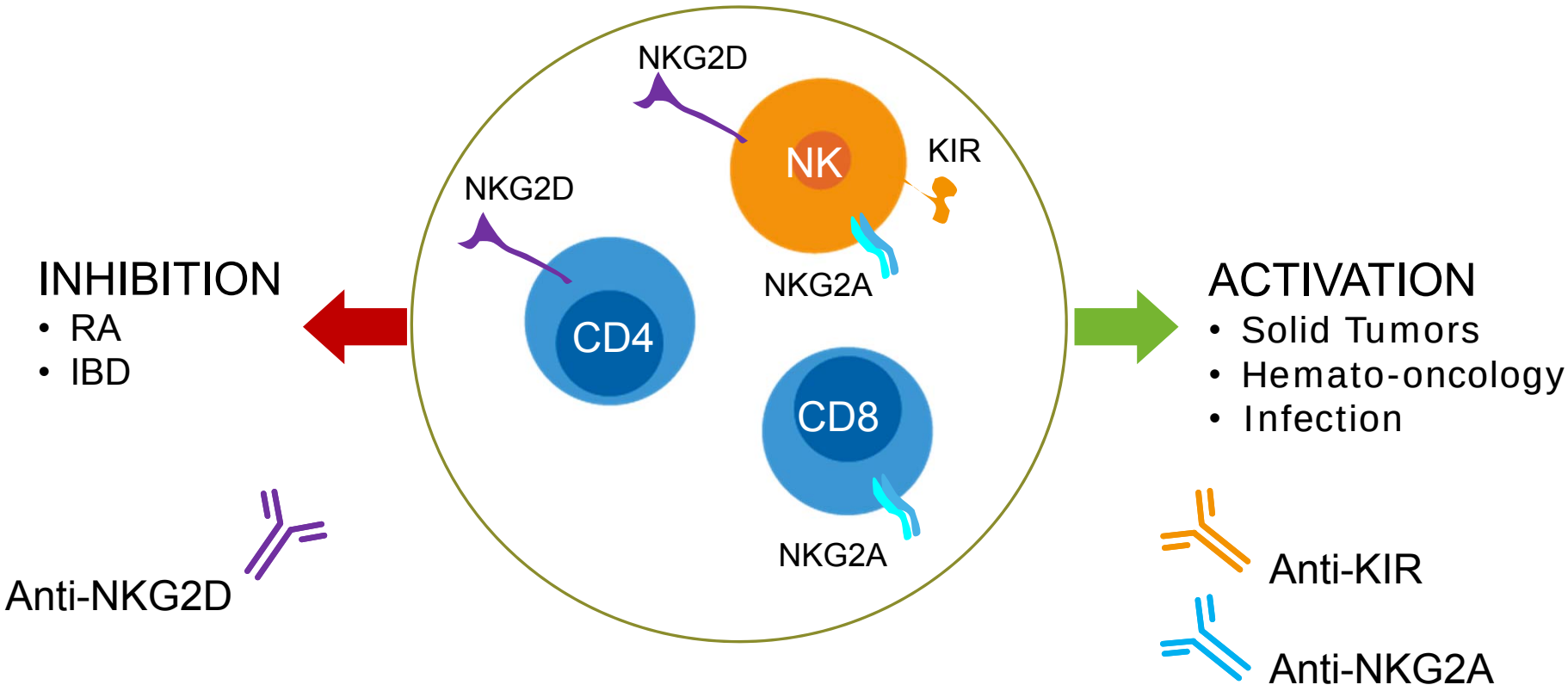


NK cells controlled by activating and inhibitory receptors



Innate Pharma

Immunomodulation with mAbs targeting “NK receptors”



Pipeline

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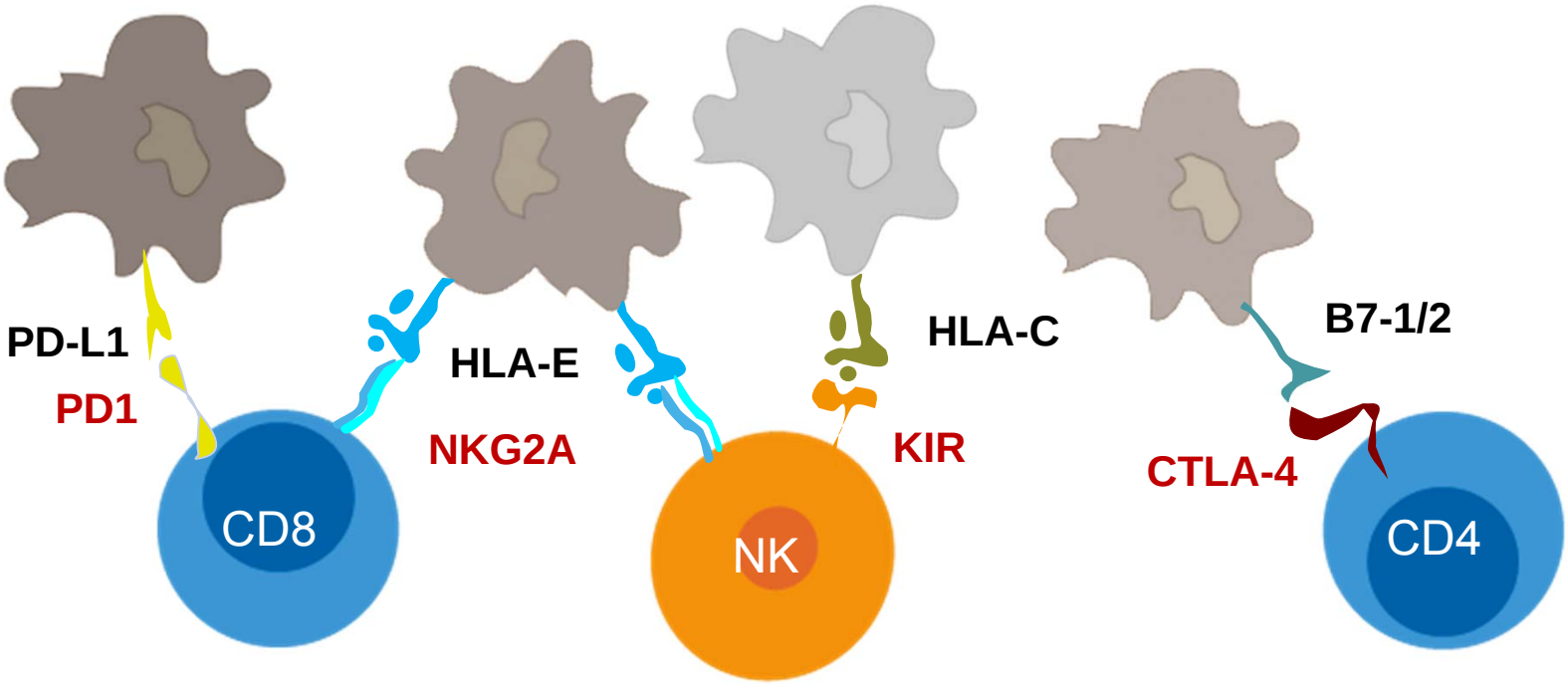
	NKG2A	IPH			
Activating receptors	**CD137**	Pfizer	BMS		
	CSF-1R	AMG, LLY, Roche			
	B7-H3	Servier/Macrogenics			
	CD40	Roche			
	OX40	AZN			
	GITR	GITR Inc			
	CD27	Celldex			

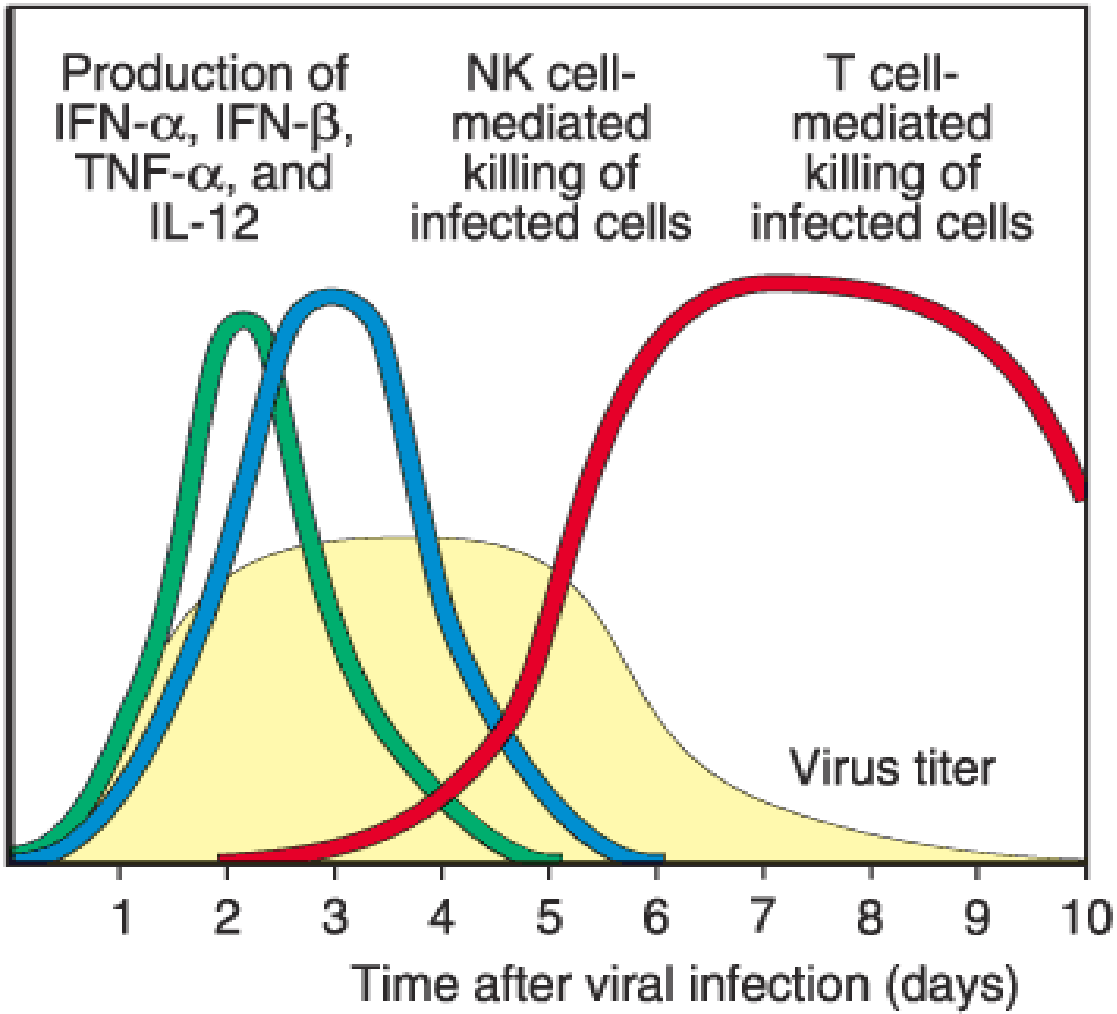
Heterogeneous expression profile of checkpoint receptors and ligands creates opportunity for differentiation



Multiple agents needed to address large population of patients

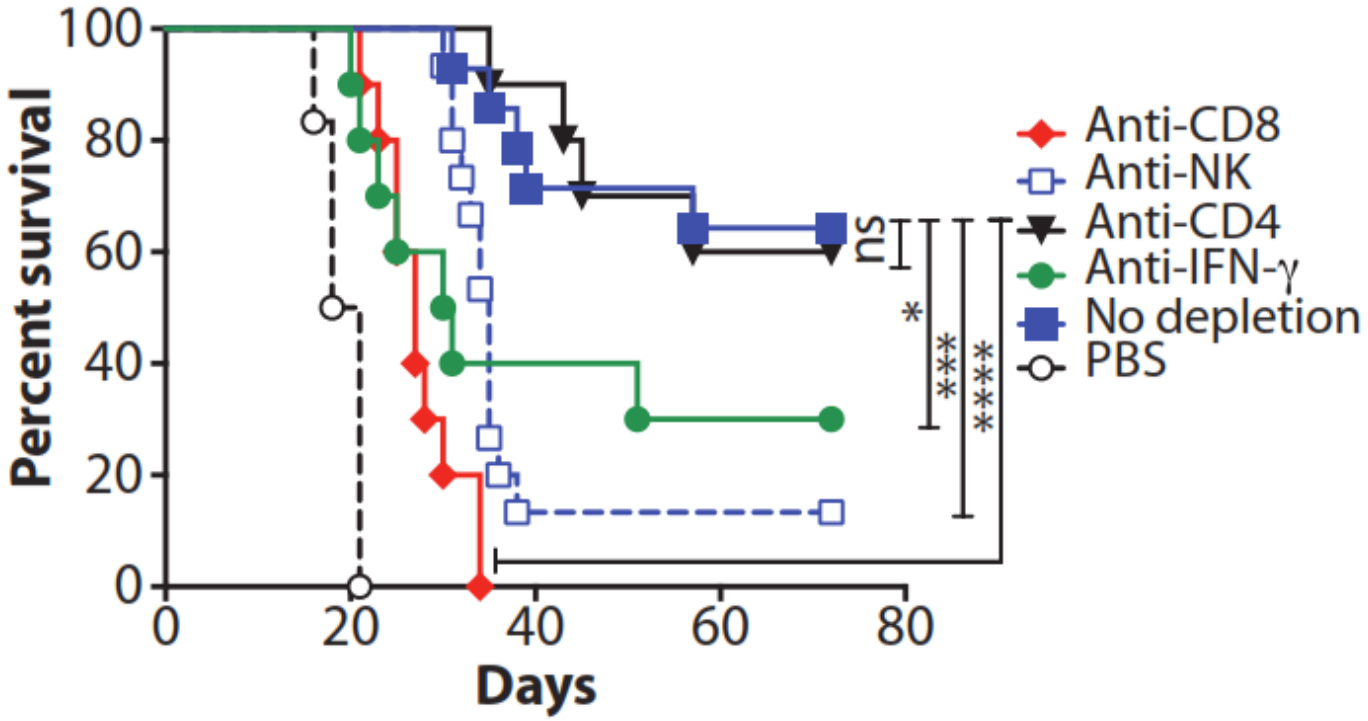
Maximize outcomes by patient stratification and/or combination therapy

NK cells protect and orchestrate



Janeway, Immunobiology

In mouse melanoma, NK and CD8+ T cells were required for therapeutic effect of anti-CTLA4



Zamarin et al. 2014. Science Translational Medicine

Innate Pharma's pipeline

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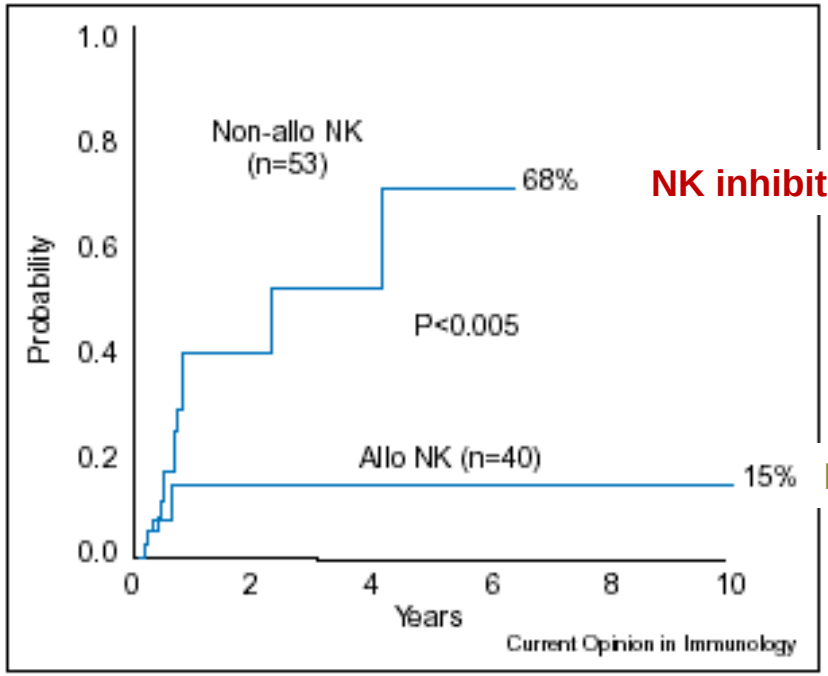
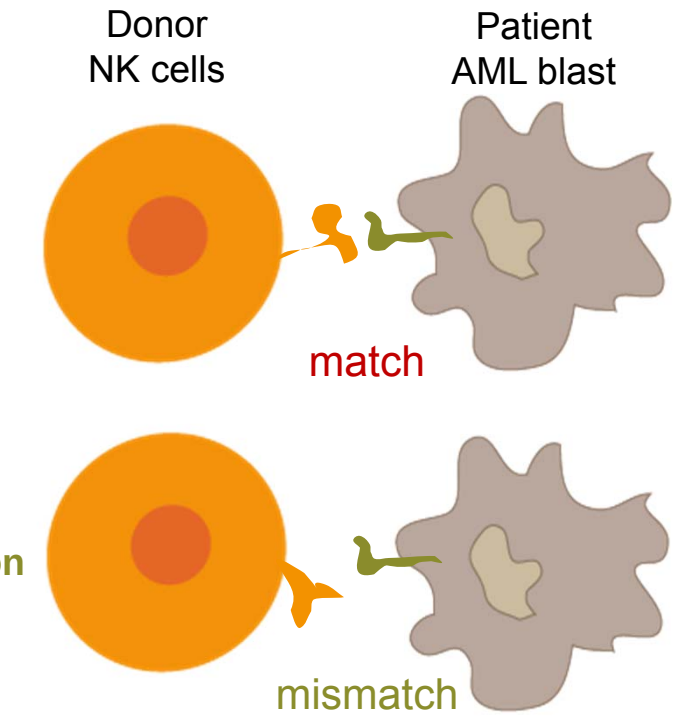
Clinical impact of KIR in AML patients

AML relapse after allo-transplantation



NK inhibition

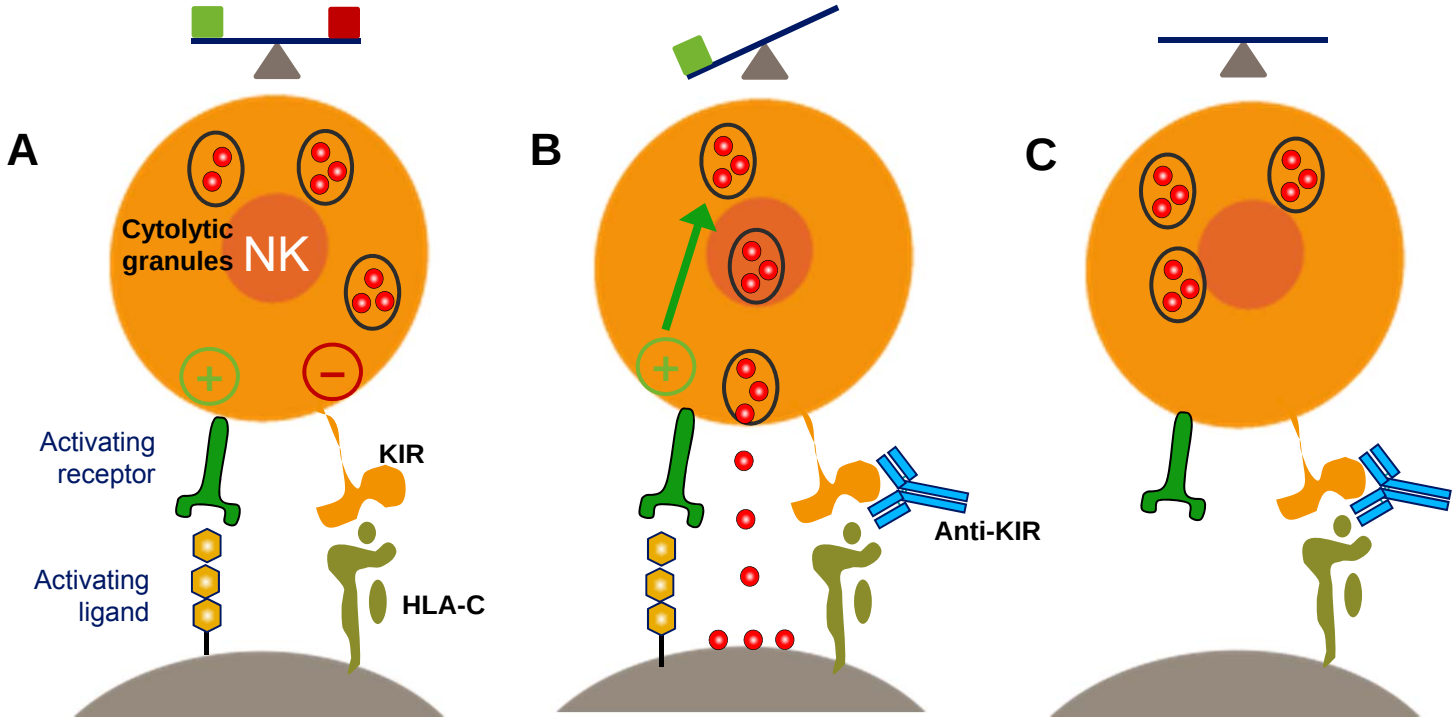
NK activation



- Three types of KIR2DL
- Need for a cross reactive mAb

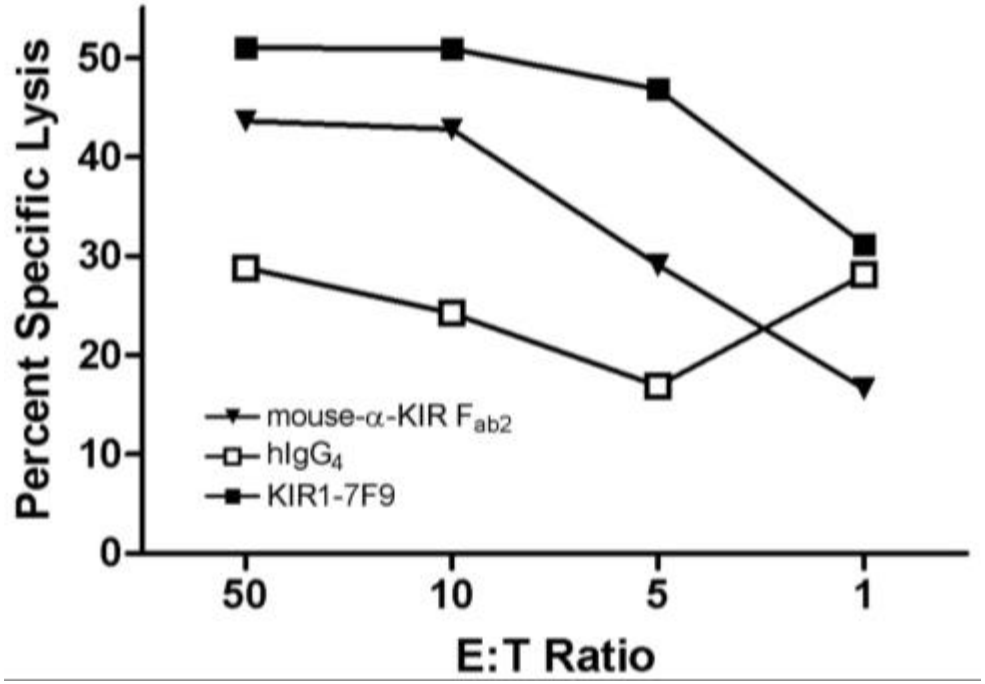
Ruggeri et al Curr. Opinion Immunol 2005

Lirilumab (anti-KIR): Mechanism of action



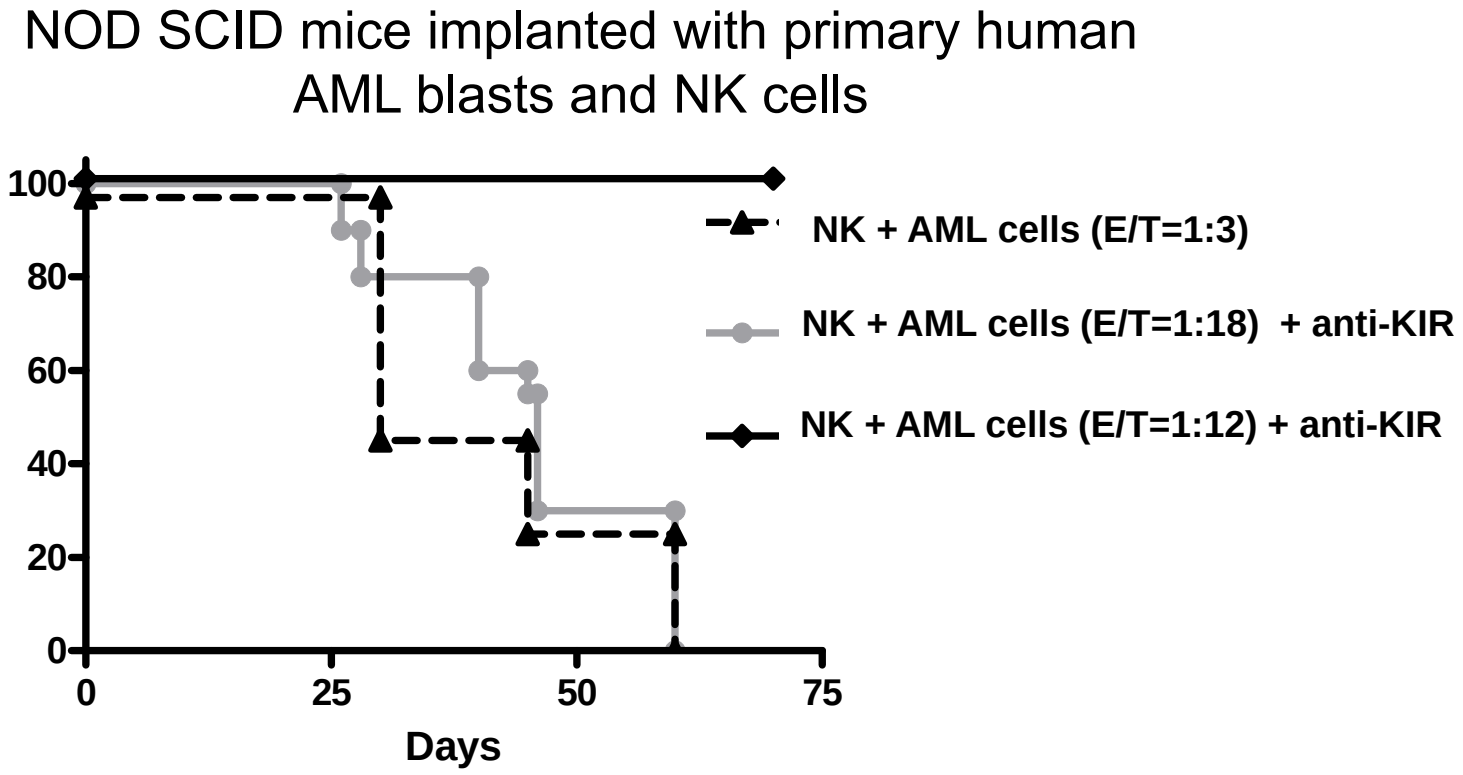
Anti-KIR induces killing of primary AML blasts *ex vivo*

Ex vivo killing of primary AML blasts by autologous NK cells from patient in remission



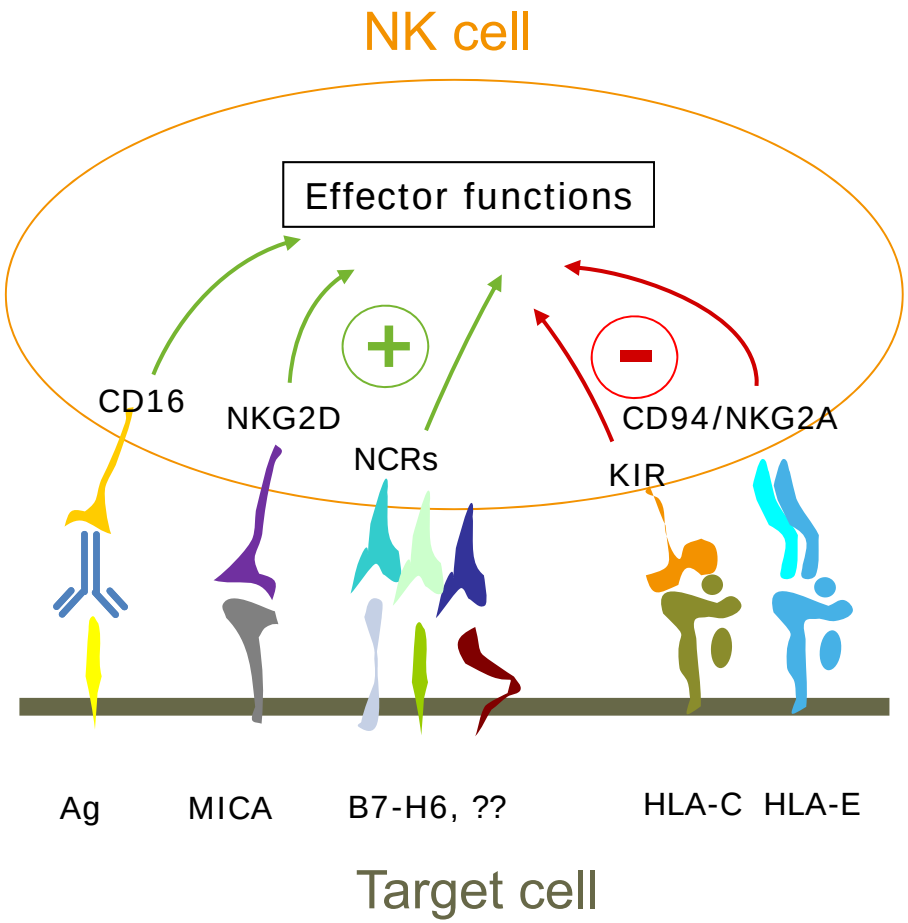
Romagne et al. Blood 2009

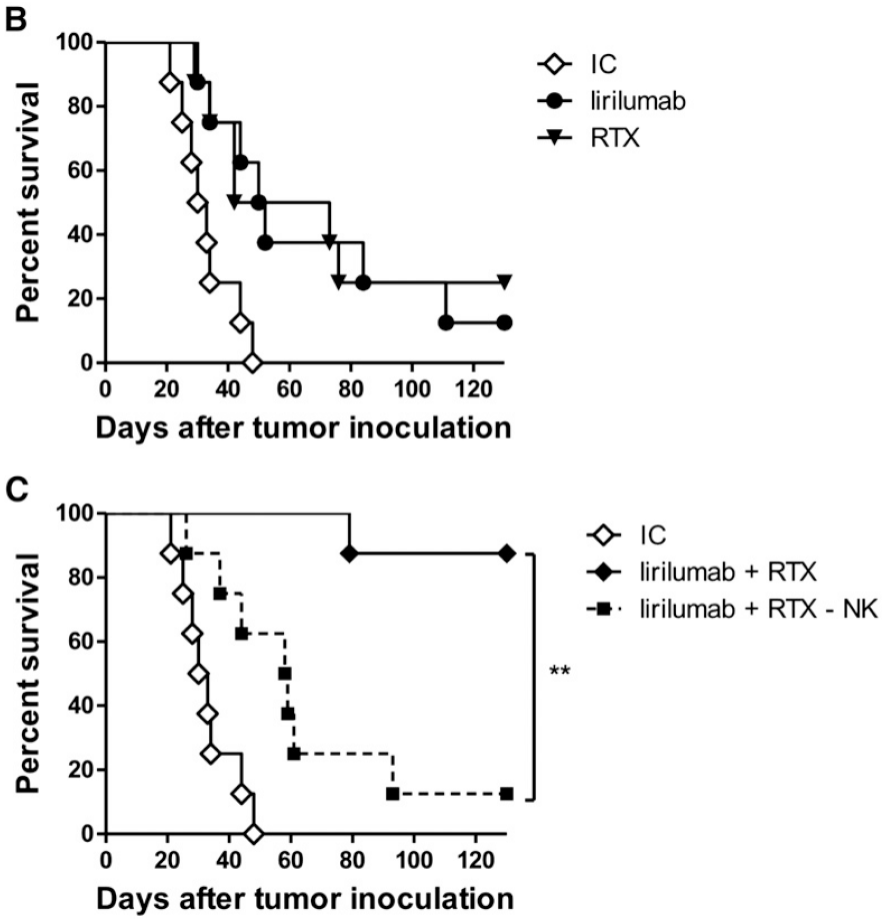
Anti-KIR induces killing of primary AML blasts *in vivo*



Romagne et al. Blood 2009

Anti-KIR enhances rituximab ADCC





Adapted from Kohrt et al. Blood 2014

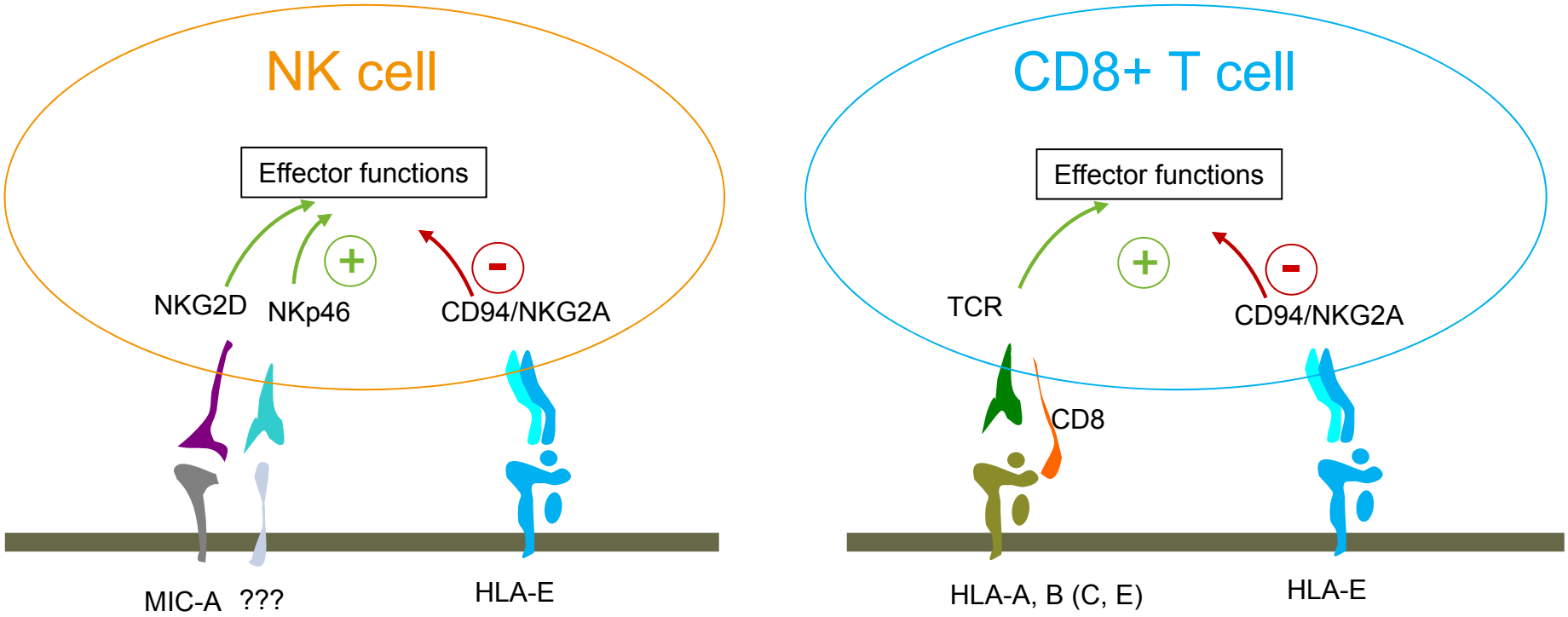
Agenda

I. Innate Pharma: Unique scientific positioning driving first-in-class pipeline

II. Lirilumab: A checkpoint inhibitor built on translational clinical data

III. **IPH2201: A novel checkpoint inhibitor targeting NK and T cells**

NKG2A: A checkpoint receptor on NK and T cells

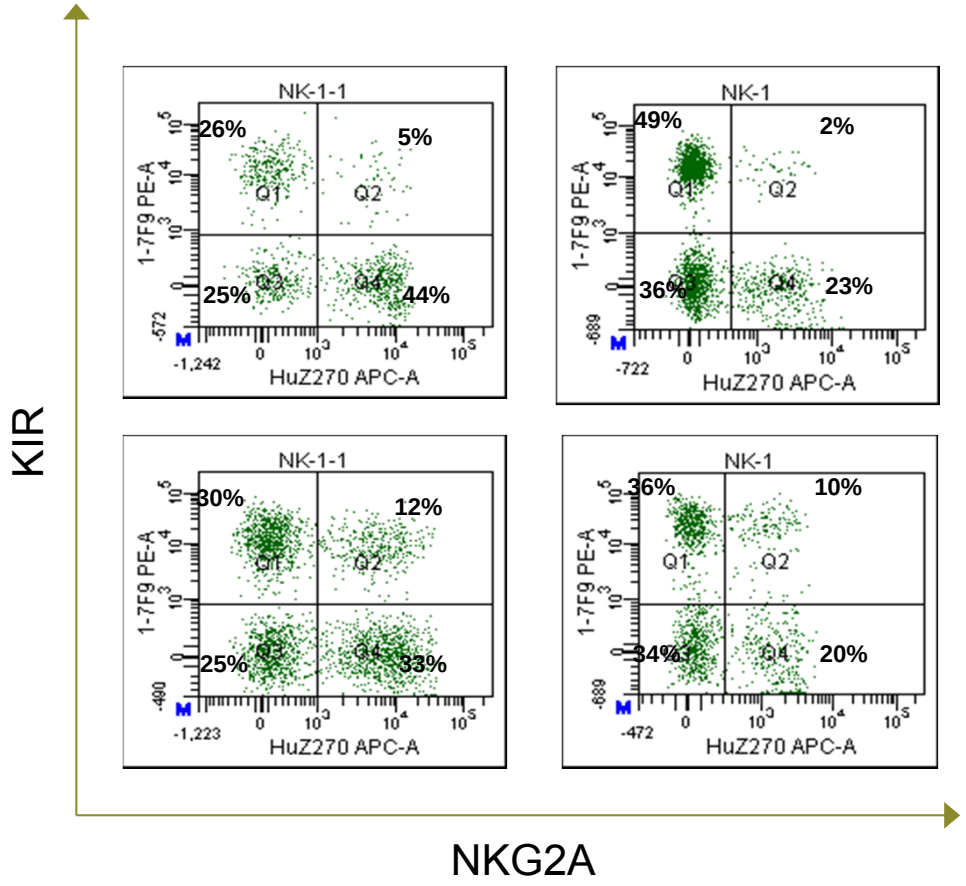


Target cell

(tumor or infected cell, stressed cell in chronic inflammation)

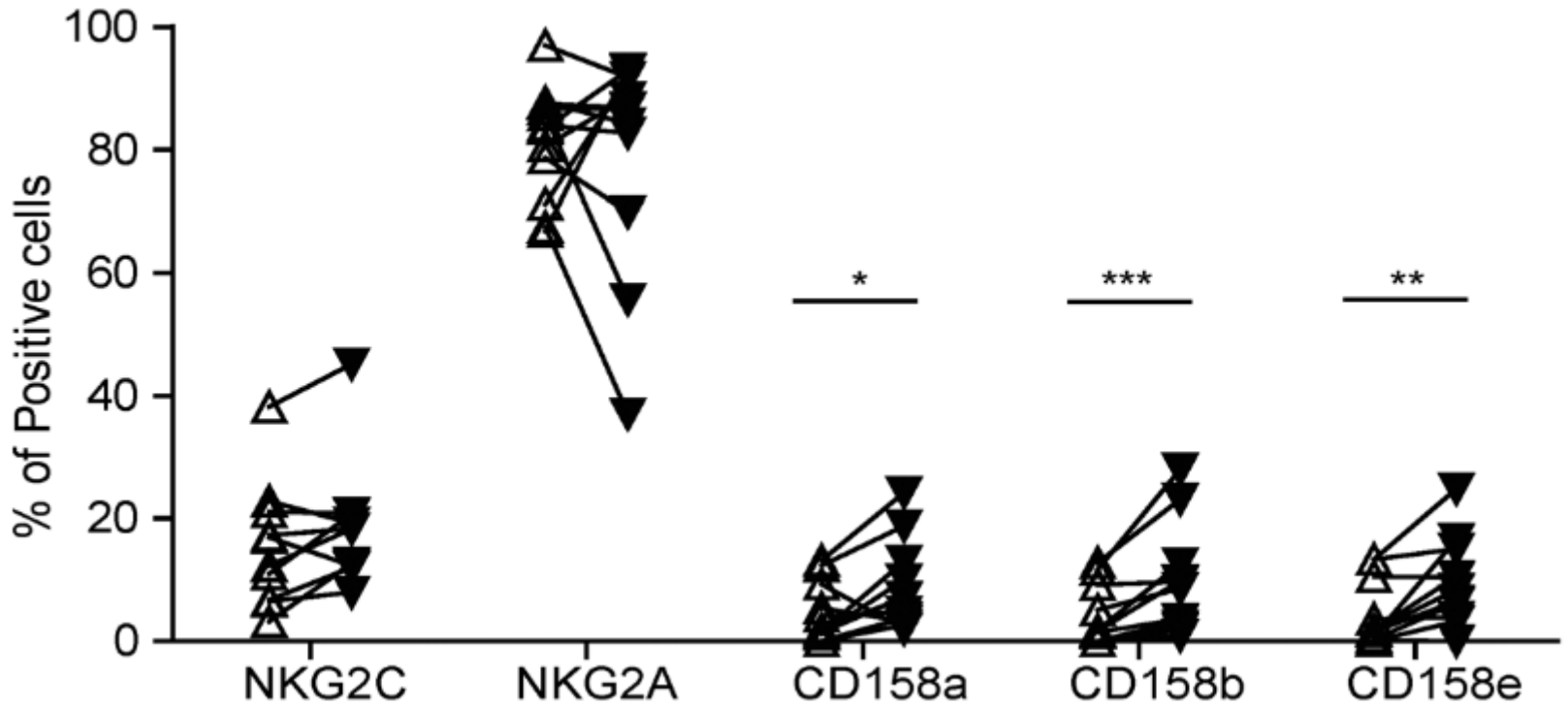
KIR and NKG2A are expressed on different subsets of NK cells

Blood NK cells from four healthy donors (one in each panel) stained for KIR and NKG2A



NKG2A expression on tumor infiltrating lymphocytes 1/2

Melanoma infiltrating NK cells

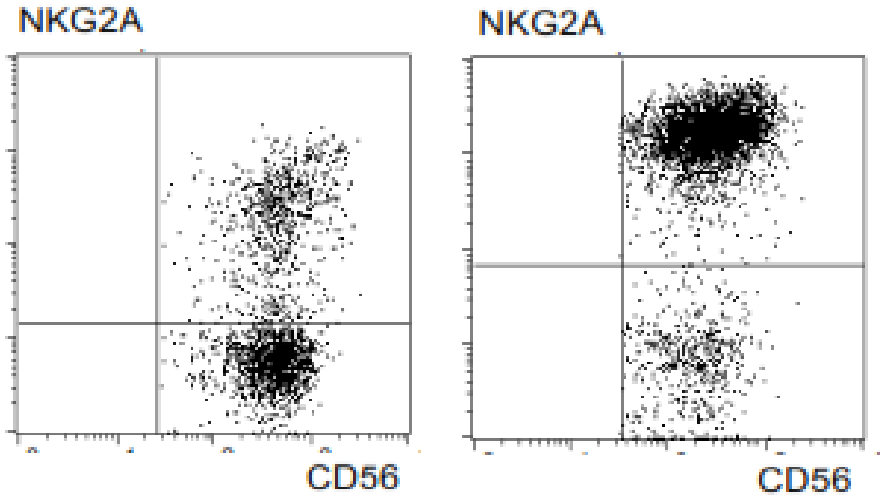


Messaoudene M et al, Cancer Res 2014

NKG2A expression on tumor infiltrating lymphocytes 2/2

Upregulation of NKG2A on NK cells inside tumors

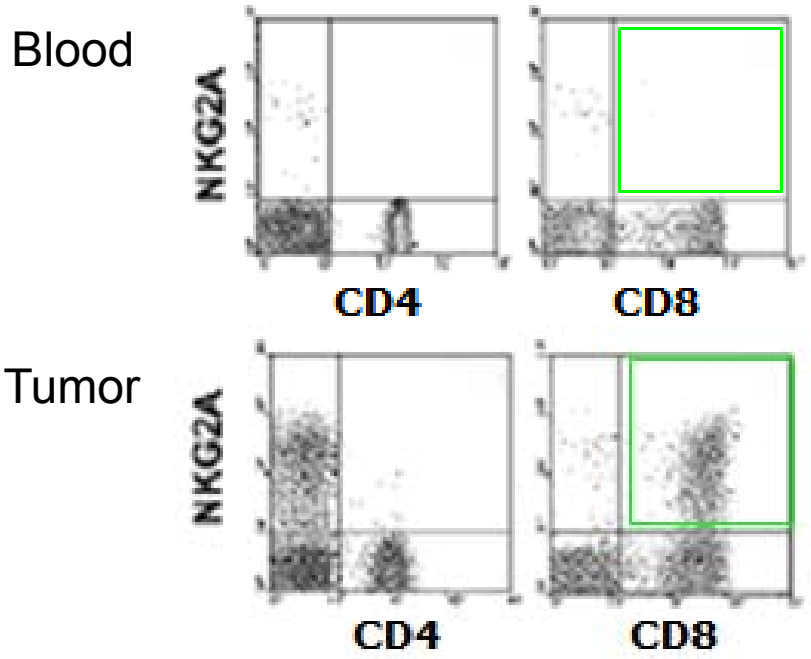
Lung carcinoma



Blood NK (HC) | Intratumoral NK

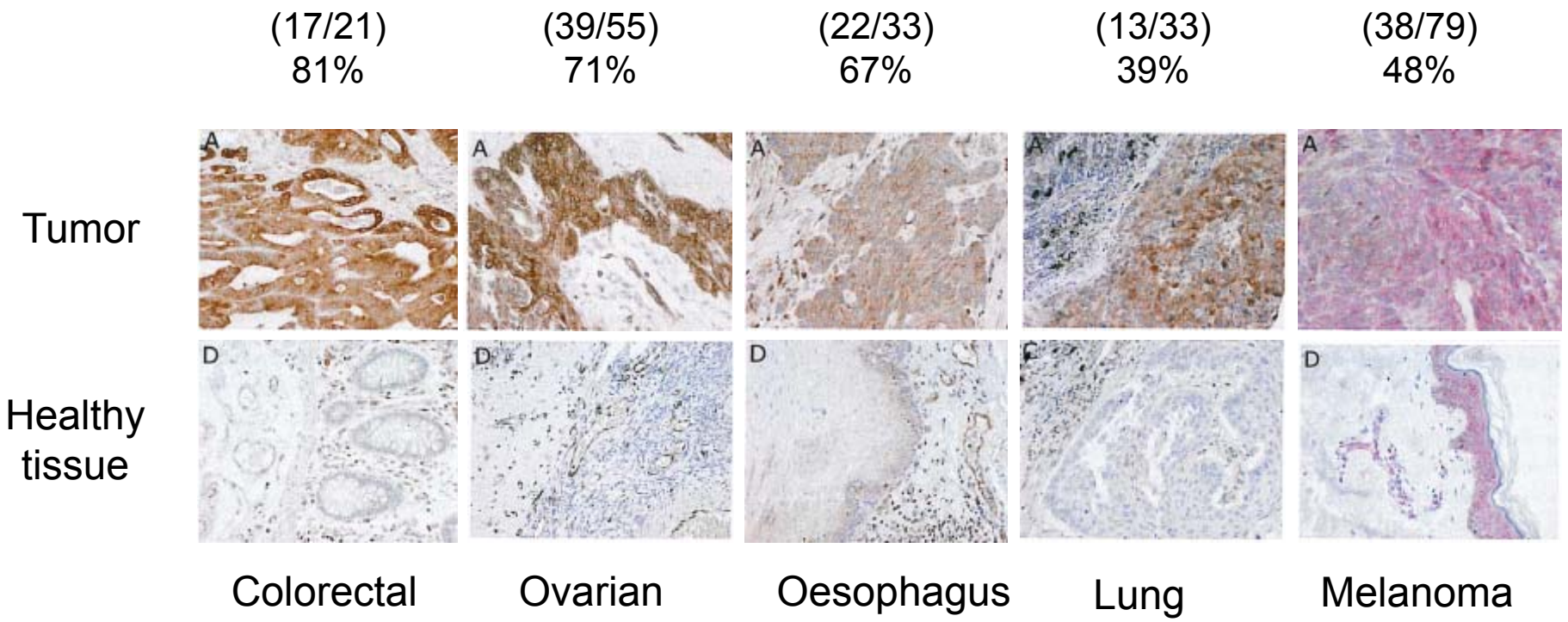
NKG2A on tumor infiltrating CD8⁺ T cells

Cervical cancer



From L to R : Platonova et al. 2011, Sheu et al. 2005

HLA-E over-expressed in diverse solid tumor types



HLA-E over-expressed in hematological cancers

HLA-E expression on 28/30 AML patients samples

HLA-E expression on 4/4 ALL and 7/7 CLL patient samples

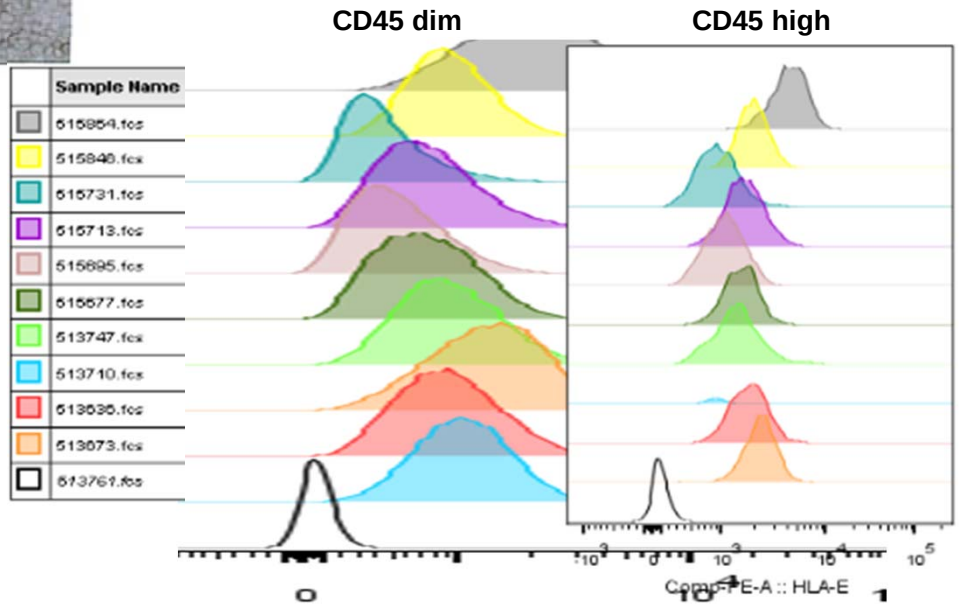
AT 178	CLL A	+++
AT 181	CLL A	+++
AT 208	CLL A	+++
AT 484	CLL	+++
AT 482	CLL	+++
AT 397	CLL	+++
AT 522	CLL	+++

AT181 CLL

IHC staining with MEM-E/02

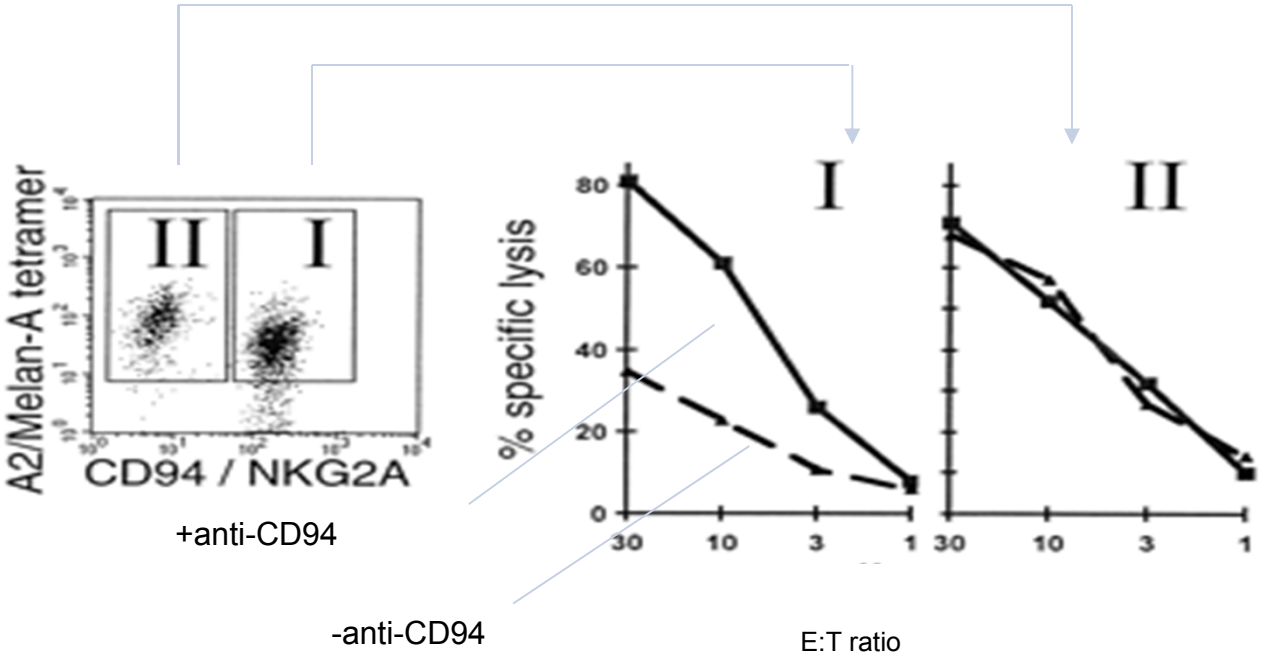
Flow cytometry: staining CLL with anti-HLA-E mAb

$CD19^{+}$ $CD3^{-}$ Blasts (n=8)



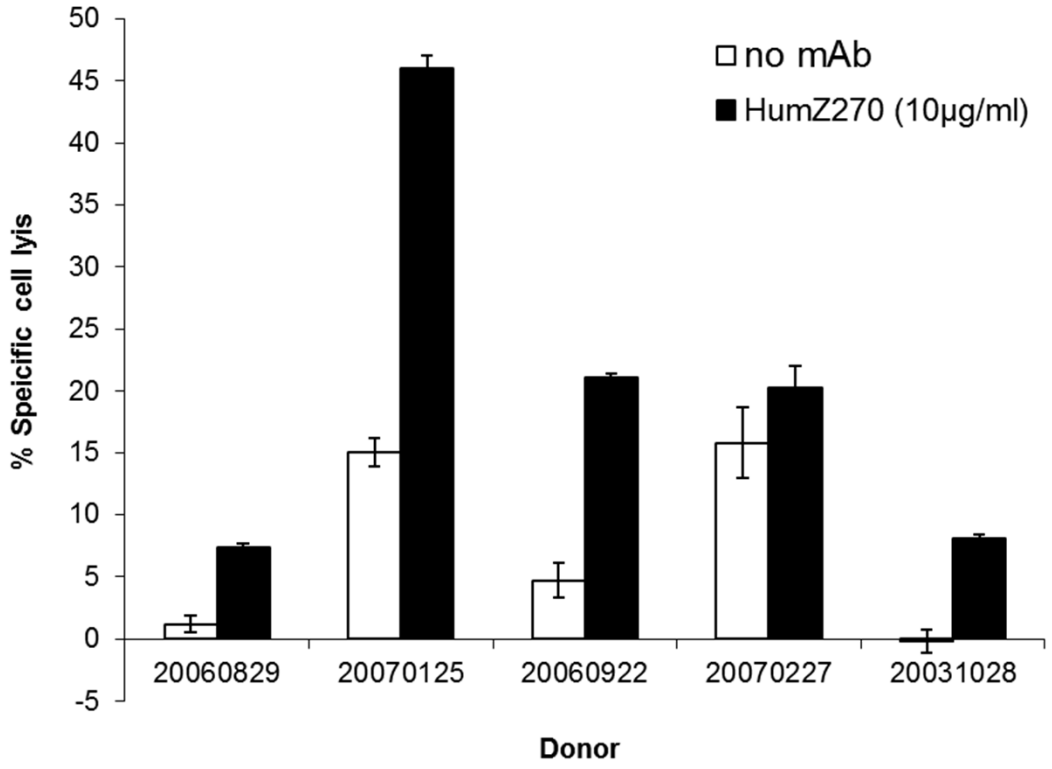
Internal data

Ex vivo, NKG2A blockade enhances T cell killing of melanoma cells

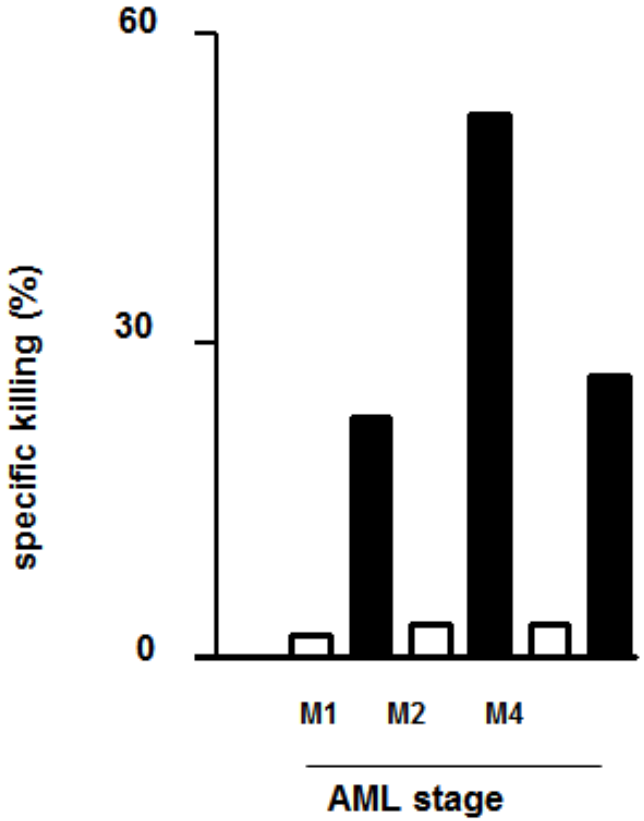


Speiser et al., JEM 1999

IPH2201: First-in-class humanized anti-NKG2A mAb potentiates NK killing of primary B cell blasts



IPH2201 stimulates NK killing of primary AML blasts



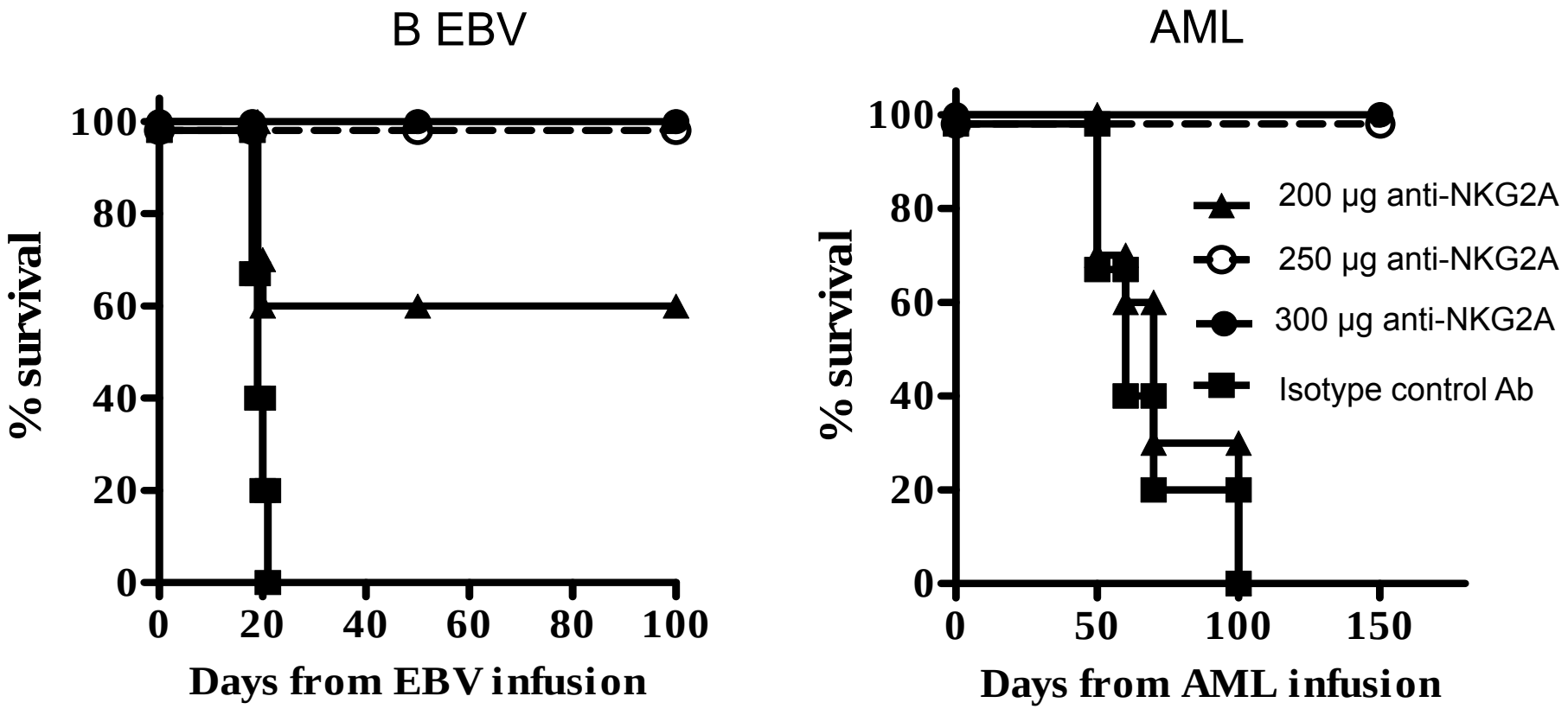
Effectors: IL2 act NKG2A+/KIR- NK cells

Targets: AML blasts (M1, M2, M4 according to FAB classification)

E/T ratio: 15:1

Ruggeri & Velardi, unpublished

In vivo anti-tumor activity of IPH2201 in established malignancies in humanized NSG mice



Ruggeri & Velardi, unpublished

Summary

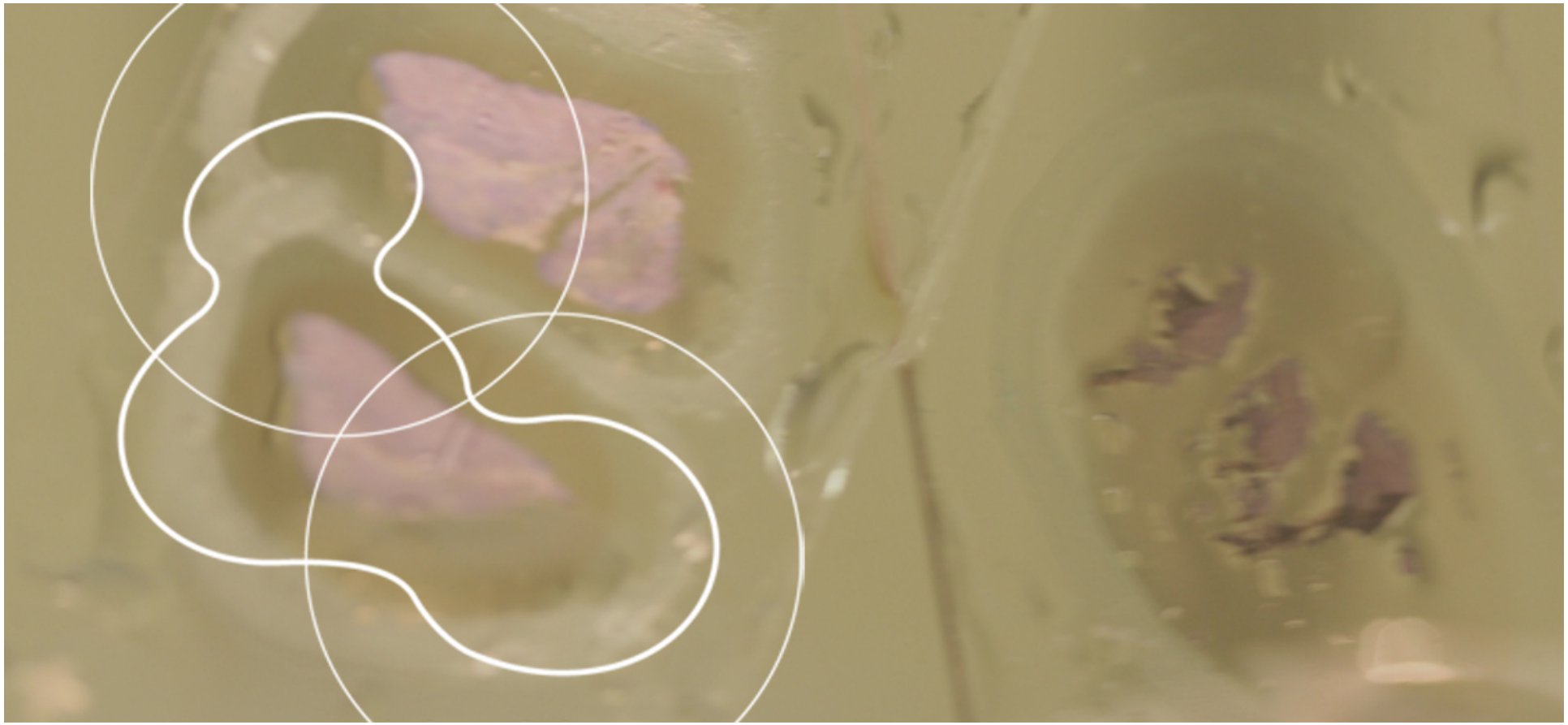
- NKG2A is a novel checkpoint receptor inhibitor, targeting subsets of cytotoxic NK and T lymphocytes
- Tumor infiltrating NK and T cells are often NKG2A^{+}
- NKG2A's ligand, HLA-E, is upregulated on a variety of tumor types
  - Can correlate with poor prognosis
  - May be a frequent tumor immuno-escape mechanism
- IPH2201 is a phase II-ready checkpoint inhibitor stimulating killing of HLA-E^{+} tumor cells
- HLA-E expression may provide biomarker opportunity

Novel targets and cell types

Compound	Ab type	MOA
Anti-KIR2DL1/2/3	IgG4	Activate NK cells
Anti-NKG2A	IgG4	Activate NK and CD8+ T cells
Anti-KIR3DL2	IgG1	ADCC, target tumor antigen Rare disease
Anti-TLR3	IgG4	Blocking inflammation
Anti-MICA	TBD	ADCC, target tumor antigen Modulating NK cells
Other targets ADC technology		Beyond I-O Beyond Cancer Beyond NK cells

Our pipeline – IP and competition

Coumpound	Exclusivity (US)	Competitors ahead
Anti-KIR2DL1/2/3	2026+	First in Class
Anti-NKG2A	2028+	First in Class
Anti-KIR3DL2	2033+	First in Class
Anti-TLR3	2033+	J&J
Anti-MICA	2033+	First in Class



Clinical development update

Marcel Rozencweig, CMO

R&D Update, NY, April 10, 2014

Agenda

I. Lirilumab: Overview

II. IPH2201 : Clinical development avenues

1. Summary of Phase 1 safety trial
2. Early clinical development strategy
   a. Squamous cell carcinoma of the head and neck
   b. Chronic lymphocytic leukemia
   c. Ovarian cancer
3. Expected 2014-2017 outcomes

Lirilumab

Overview

Lirilumab: Partnered with Bristol-Myers Squibb
Overview of clinical development

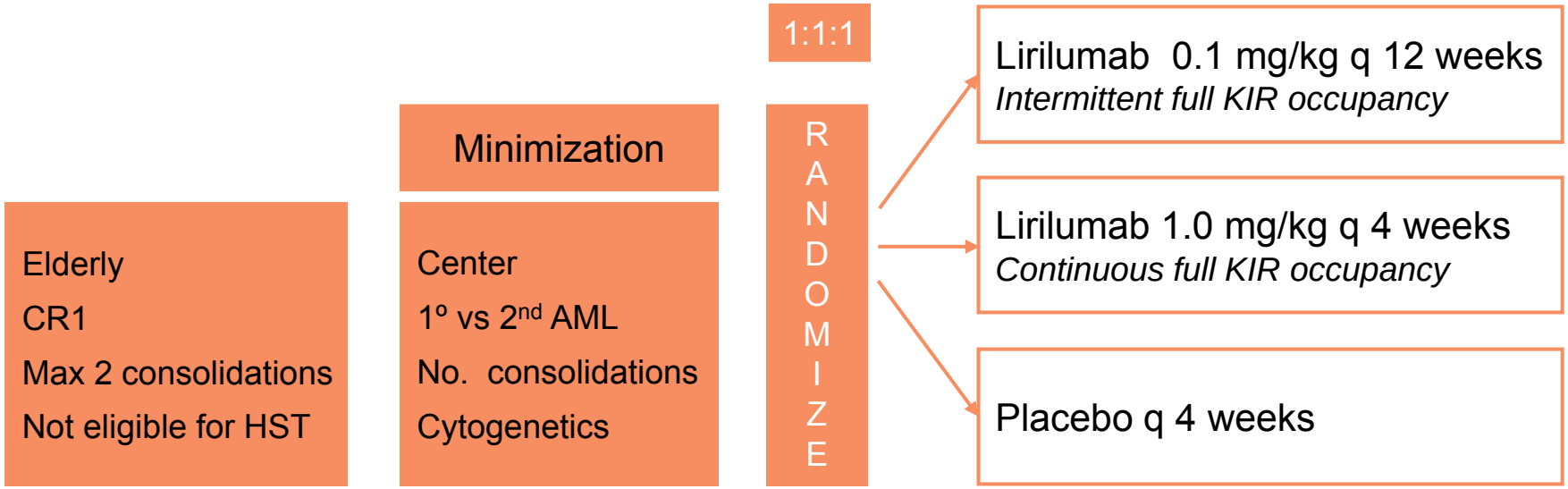
Acute Myeloid Leukemia Single-agent	Phase 2 EffiKIR	150 patients *Executed by Innate Pharma per agreement with BMS*	2012/10 - 2016/06
Solid tumors Combination with ipilimumab	Phase 1	125 patients	2013/01 - 2015/07
Solid tumors Combination with nivolumab	Phase 1	162 patients	2012/10 - 2015/09

Source: clinicaltrials.gov

Intergroup ALFA/Goelams EFFIKIR Phase 2 trial

Double-blind placebo-controlled randomized trial of lirilumab in AML

- First randomized Phase 2 of lirilumab
- Sponsored by Innate



Treatment for 2 years

Primary endpoint: Leukemia-Free Survival (Independent Review Committee)

N=50 per arm (100 events) for overall α at 0.05 one-sided and power of 0.80, assuming median LFS of 12 months in the control group vs. 20 months in the treatment groups

Recruitment period: 15-18 months; Maximum follow-up period: 24 months after last patient entry

Phase 1 study of lirilumab in combination with nivolumab

Patients with advanced solid tumors

- Dose escalation completed

- Cohort expansion in various solid tumors:
  - > Nivolumab 3 mg/kg, every other week + lirilumab 3 mg/kg, every four weeks
  - > Patients dosed for up to two years

- Participating centers (USA):
  - > Memorial Sloan-Kettering Cancer Center, New York, NY
  - > Dana-Farber Cancer Institute, Boston, MA
  - > University of Chicago Comprehensive Cancer Center, Chicago, IL
  - > Johns Hopkins Comprehensive Cancer Center, Baltimore, MD
  - > Providence Portland Medical Center, Portland, OR

- Estimated enrollment: 162

Phase 1 study of lirilumab in combination with ipilimumab

Patients with advanced solid tumors

- Regimen schedule: concurrent administration q 3 weeks x 4 then q 12 weeks x 4
- Dose escalation: observation period of 9 weeks after first cycle

Dosage during dose escalation

Dose level number	Total number of patients	Lirilumab, mg/kg	Ipilimumab, mg/kg
1	3-9	0.1	3
2	3-9	0.3	3
3	3-9	1	3
4	3-9	3	3
5	3-9	3	10
Total	**15-45**		

- Cohort expansion:
  - > Treat at the MTD or maximum administered dose
- Indications:
  - > Non-small cell lung cancer
  - > Castrate resistant prostate cancer
  - > Melanoma

- Participating centers (USA)
  - > Memorial Sloan-Kettering Cancer Center, New York, NY
  - > Dana-Farber Cancer Institute, Boston, MA
  - > Ohio State University, Columbus, OH
  - > Sarah Cannon Research Institute/ Tennessee Oncology, Nashville, TN
  - > Moffitt Cancer Center, Tampa, FL
  - > Masonic Cancer Center, University of Minnesota, MN

IPH2201

Anti-NKG2A Antibody

Clinical Development Avenues

IPH2201

Phase 1 Trial of IPH2201

Study design

- Randomized, double-blind, placebo-controlled, dose-escalation trial
- 92 patients enrolled in 3 separate cohorts
  - > Single dose i.v. up to 10 mg/kg
  - > Single dose s.c. up to 4 mg/kg
  - > Multiple doses s.c. with q 2 weeks x 4 schedule up to 4 mg/kg
  - > 4-7 Patients per dose level
- Single center trial (Charité CRO, Berlin, Germany)

Patient selection

- M or F, age 18 to 75 yrs
- Active RA (ACR 1987 classification)
  - > DAS28 (CRP) ≥ 3.2
  - > Diagnosis ≥ 3 months duration prior to randomization
- Methotrexate at stable doses (7.5 to 25 mg/week) for ≥ 4 weeks
- Patients on stable doses of oral corticosteroids (prednisolone or equivalent ≤10 mg/day), NSAIDs, acetaminophen a/o opioids acceptable

Interim results

- At least 12 week safety data available for all patients
  - > MTD not reached, no SUSAR
  - > No drug-related serious adverse events
  - > Nasopharyngitis and headache two most frequently reported AEs
- PK similar to other mAbs binding a membrane receptor
- LPLV in March 2014
- Trial completion expected mid-2014

Recommendations for Phase 2 trials with IPH 2201

Objective:
To maintain full NKG2A receptor occupancy during a whole cycle

- Not only in peripheral blood (PB) but also in tumor micro-environment
- Considering that mAb levels would likely be markedly lower in profound tissues

2 doses and schedules could be tested in Phase 2

- i.v. 10 mg/kg q4w
- s.c. 4 mg/kg q2w

Treatment to disease progression

IPH2201

Early Clinical Development Strategy

Summary of clinical development plan of IPH2201

Five targeted Phase 2 trials

Heme and solid tumors

Single agent and combinations

Rapid clinical proof of principle

Criteria for development strategy and selection of indications

- Speed to generation of activity data
  - > Endpoint of clinical trial: Tumor shrinkage vs. duration of tumor control
  - > Available patient population
  - > Support of large cancer centers and KOL
- Scientific rationale
  - > Expression of NKG2A ligand HLA-E on tumor in vast majority of patients
  - > Prognostic correlate of HLA-E expression with clinical outcome
  - > Evidence of activity of NKG2A+ immune effectors
- Unmet medical need
- Defined path to registration from efficacy signal

Selecting patients based on HLA-E expression

- Tumor types where vast majority of patients known to express HLA-E on surface of tumor cells
  - > Reduced need for restricting patient selection
- Need for extensive prospective biomarker studies
  - > Tumor tissue availability for biomarker studies

Indications

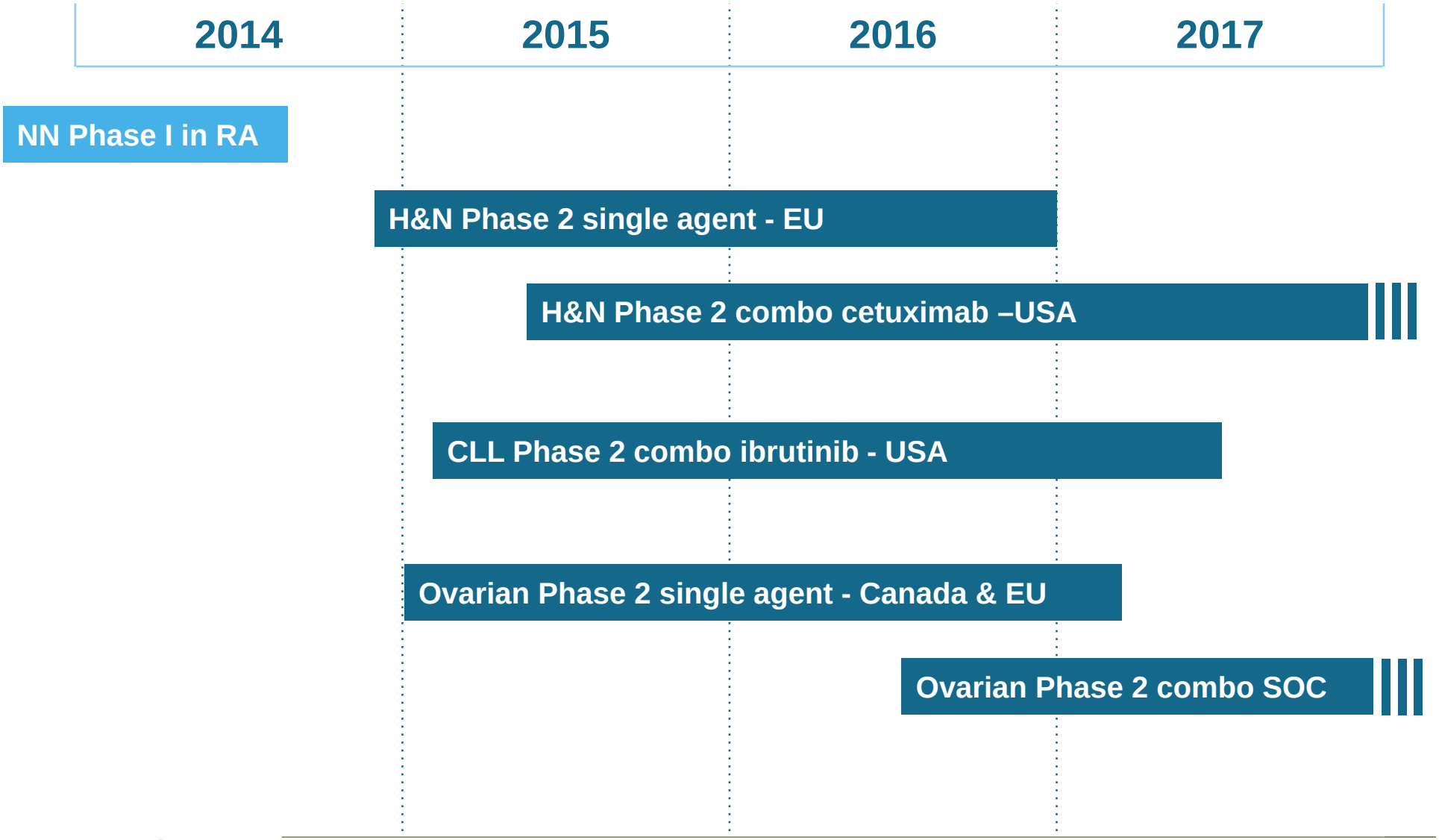
Preliminary prioritization:

- Head and neck cancer
- CLL
- Ovarian cancer

Additional indications to be considered:

- Melanoma
- AML
- Colorectal
- NSCLC
- Endometrium
- Cervix
- Renal
- Bladder
- Others (ongoing investigations)

IPH22 Development highlights



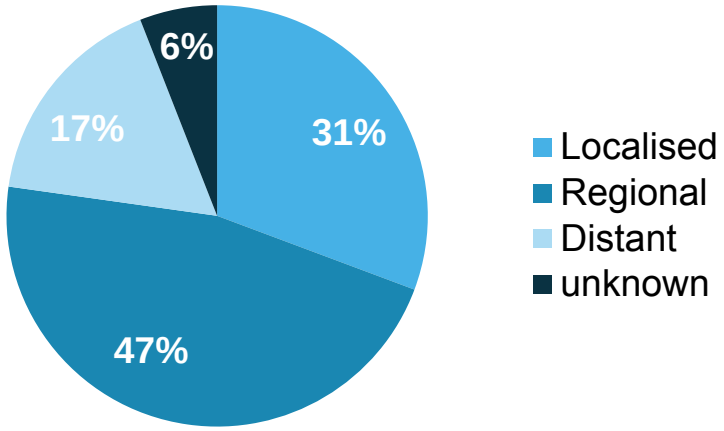
IPH2201

Squamous Cell Carcinoma of the Head and Neck (SCCHN)

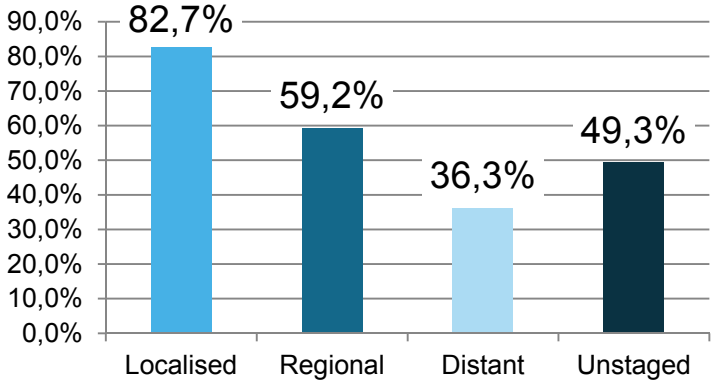
Incidence and prognosis of SCCHN cancer

- Sixth most common cancer worldwide[1] (estimate)
  - > 650,000 cases and 200,000 deaths /yr worldwide, most common cancer in Asia[1]
    - US: about 42,440 new cases and 8,390 deaths in 2014[2]
    - EU: 139,000 new cases / yr [3] and > 68000 death /yr [1]

Percent of cases by stage [4]



5-year relative survival rate [4]



1.Globocan Project Geneva: World Health Organisation, 2010; 2. Siegel R, CA Cancer J Clin 2014; 3.ESMO Guidelines; Gregoire V, Ann Oncol 2010; 4 .SEER Stat Fact sheets: oral cavity and pharynx cancer, 2003-2009

SCCHN
Risk factors

- Tobacco use, alcohol use and human papillomavirus (HPV) infection are important risk factors
- Better prognosis associated with HPV [5,6]

5. Fakhry C, JCO 2006; 6. Ritchie JM, Int J Cancer 2003

SCCHN
Drugs Approved

- Bleomycin
- Cetuximab
- Cisplatin
- Docetaxel
- Fluorouracil
- Methotrexate

SCCHN

Treatment strategy in locally advanced disease

- At diagnosis, 60% of patients have locally advanced disease
  - > SOC: Surgery and cisplatin-based chemo-radiation [3,7]
- The majority of these patients develop local and/or regional recurrences and distant metastases occur in 20%–30% of patients[8]
- Recurrent or metastatic SCCHN have a poor prognosis with a median overall survival ranging from 6 to 9 months[9]

3.ESMO Guidelines; Gregoire V, Ann Oncol 2010;
7. NCCN. Clinical practice guidelines in oncology: head and neck cancers. v.2, 2013;
8. Murar S, Mayo Clin Proc 2008; 9. Specenier PM, Expert Rev Anticancer Ther 2008

SCCHN

Standard of care in recurrent/metastatic disease

- Active cytotoxic agents
  - > Cisplatin, carboplatin, 5-FU, taxanes, methotrexate, ifosfamide, gemcitabine (for NPC), bleomycin, others
  - > Methotrexate is FDA approved for use with HNSCC but no longer commonly used in the US
- First-line therapy [3,7]
  - > For patients with good PS: historically platinum-based doublet (eg, cisplatin/5-FU or carboplatin/paclitaxel)
    - ORR: 30% to 40%; median OS: 6-9 mos regardless of specific drugs
    - Cetuximab commonly added to current treatment regimens
  - > For patients with poor PS: use single agent or cetuximab
- Second-line therapy: taxanes [3,7], methotrexate[7], cetuximab[3,7] vinorelbine [3]

3.ESMO Guidelines; Gregoire V, Ann Oncol 2010;
7. NCCN. Clinical practice guidelines in oncology: head and neck cancers. v.2, 2013

Cetuximab in SSCHN

- EGFR is overexpressed in about 80% of SCCHN and is associated with a poor prognosis[1]
- Cetuximab: IgG1 monoclonal antibody that inhibits ligand binding to the EGFR and stimulates ADCC
- Cetuximab prolongs OS when added to a first-line platinum - fluorouracil regimen for recurrent or metastatic disease and is approved for that indication[2]
- Cetuximab also approved as a single agent for 2nd line systemic treatment in H&N: ORR:13%, TTP: 70 days, OS: 178 days[3]. Still a SOC in that setting
- K-ras mutations much less frequent than in colorectal cancer; resistance related to frequent H-ras mutations?

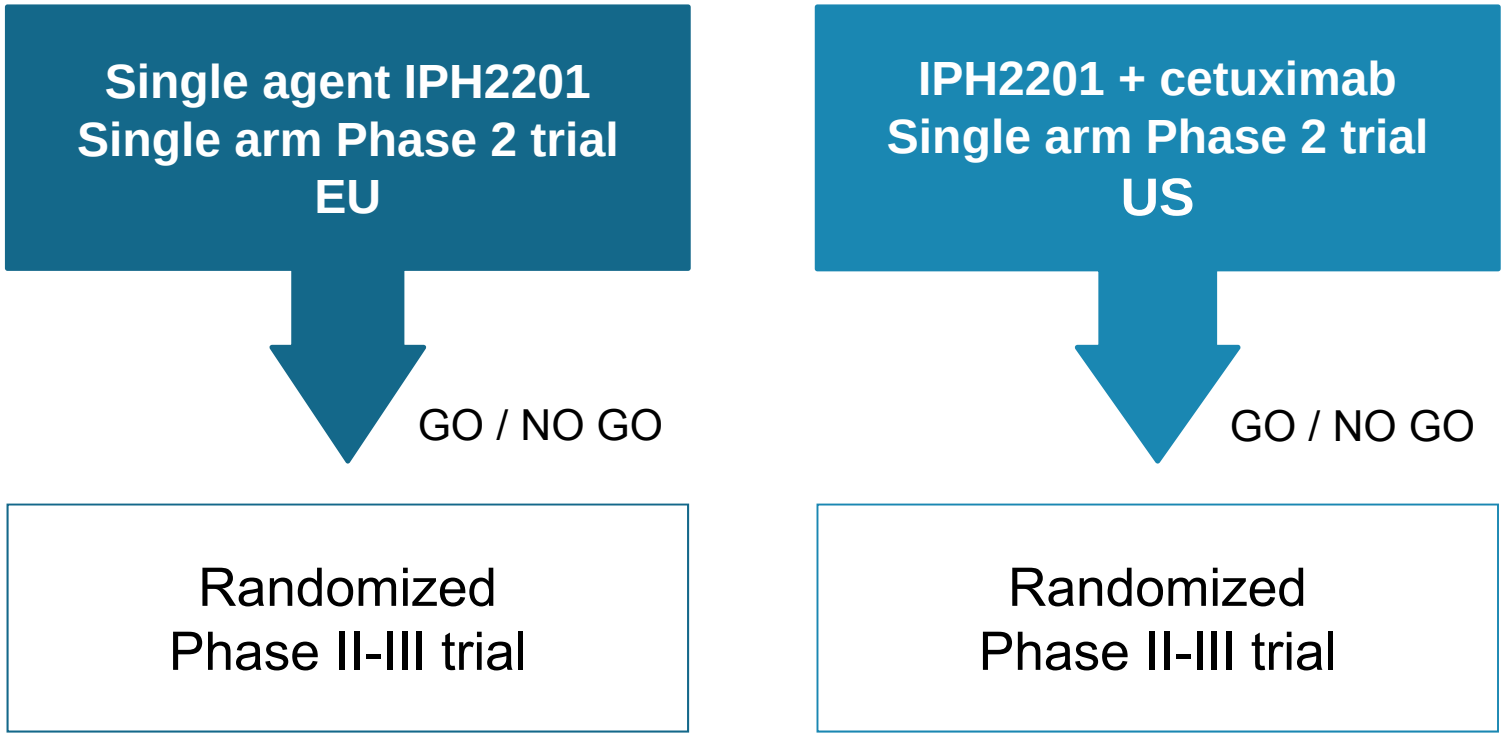
1.Temam S, JCO 2007; 2. Vermorken J, N Engl J Med 2008; 3. Vermorken J, JCO 2007

Rationale for testing IPH2201 in SCCHN

- HLA-E expression
  - > NN Esophagus SCC (n=33): 69%
  - > Tonsillar SCC (n=150): >80% in HPV + and 30/31 HPV neg ($p<0.05$) [1]
- HLA-E expression associated with histopath grade of laryngeal tumors[2]
- Intratumoral preponderance of NKG2A NK cells in the tongue[3]
- Cetuximab-mediated cellular cytotoxicity inhibited by HLA-E membrane expression in colon cancer cell lines[4]
- High unmet need
- Clear path to registration

1.Nasman A, Int J Cancer 2013; 2.Silva TG, Histol Histopathol 2011; 3.Katou F, Cancer Res 2007; 4.Levy EM, Innate Immunity 2009

Early development of IPH2201 + cetuximab in platinum refractory SCCHN



Phase 2 trial of IPH2201 as a single agent in SCCHN

Patient selection:

- Stage III/IV SCCHN
- Metastatic or recurrent
- HPV + and HPV -
- Documented PD within 1 month of platinum-based chemotherapy
- ≤ 1 regimen for recurrent or metastatic disease
- PS 0-1

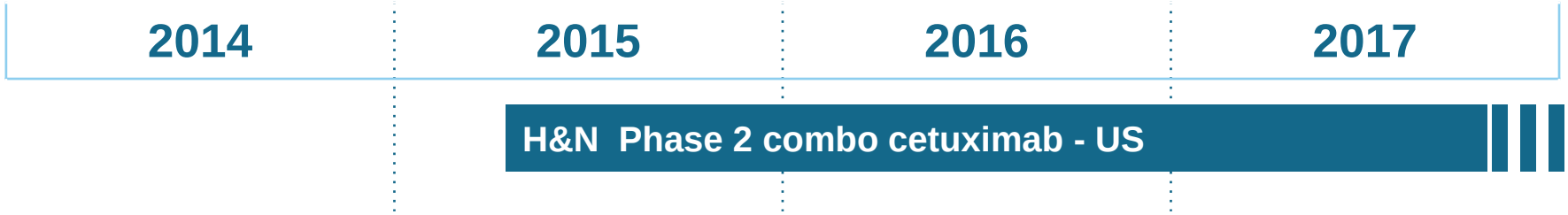
Optimum 2-stage Simon design

- 19 - 39 evaluable patients
- Primary efficacy endpoint: PFS at 4 months

Regimen:

- IPH2201: 10 mg/kg IV q4 weeks

Phase 2 trial of IPH2201 + cetuximab in patients with platinum-refractory SCCHN



Patient selection:

- Stage III/IV SCCHN
- Metastatic or recurrent
- Platinum Refractory : documented PD within 1 month of platinum –based chemotherapy
- No prior cetuximab or other EGFR therapy
- PS 0-1

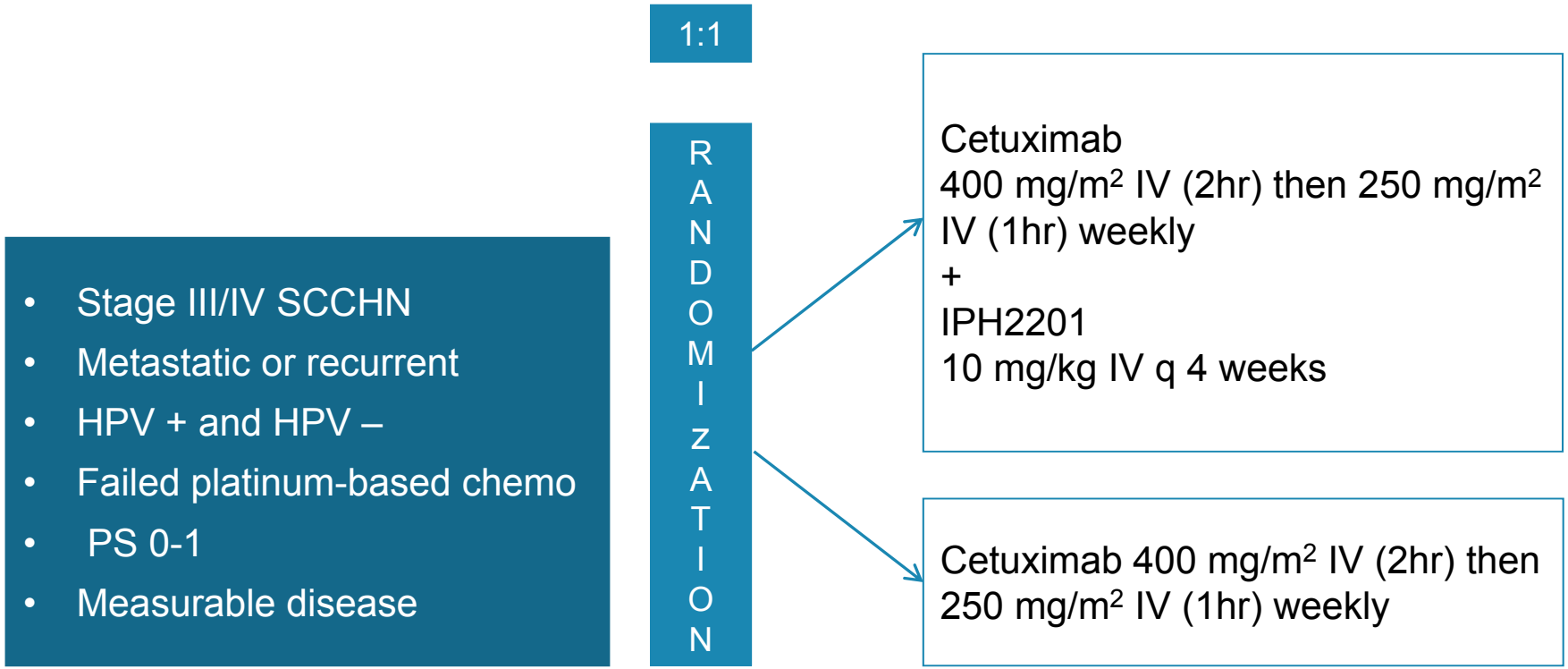
Regimen:

- Cetuximab: 400mg/m2 IV then 250 mg/m2 IV weekly + IPH2201: 10 mg/kg IV q 4weeks
- Until PD or untoward toxicity

Optimum 2-stage Simon design

- 19 - 34 evaluable patients
- Primary endpoint: RECIST Overall RR

Option for registration trial of IPH2201 in recurrent or metastatic SCCHN



Primary endpoint OS. With n=238, the probability is 0.80 that the study will detect a treatment difference at a 2 sided 0.05 significance level, if the true HR is 1.5, based on the assumption that the accrual period is 12 months, the follow-up period 12 months and the median survival in the control group is 6 months (190 events)

New drugs in development in SCCHN

Second-line Phase 2 randomized trials *vs*. cetuximab

Cetuximab ± X where X=	Start/End dates	Location	Primary endpoint	No. of patients	No. of sites
EMD1201081 TLR9 agonist (Serono)	2009/ 03-2012	US + EU	PFS	105	30
PX866 PI3K inhibitor (Oncothyreon)	2010/ 01-2014	US Canada	na	178 (H&N and CRC)	40
Tivantinib c-met inhibitor (NCI)	2012/ 12-2015	US	ORR	76	24

New drugs in development in SCCHN

Phase 3 randomized trials in platinum failures

Design	Date initiated/ Stop	Location	Primary endpoint	No. of patients	No. of sites
Afatinib* *vs* Methotrexate (LUX-H&N 1)	2011/ 03-2014	US + EU South America	PFS	474	98
Afatinib* *vs* Methotrexate (LUX-H&N 3)	2013	Asia +Africa	PFS	300	56

* Afatinib: an irreversible EGFR/erbB2/erbB4 blocker (pan-HER blockade)

Open label randomized Phase 2 trial of afatinib vs. cetuximab in platinum-refractory SCCHN (N=124)

	Afatinib	Cetuximab
Dose schedule	50 mg/d	400 mg/m2 then 250 mg/m2 weekly IV
ORR	8.1	9.7
PFS	13 wks	15 wks
Cross-over	N= 32	N= 36
Duration of disease control	17.3	16.6 wks
AEs		
• Diarrhea	78.8%	20.2%
• Rash/Acne	78.7%	76.6%
• Leading to discontinuation	37.7%	16.7%

Challenges

- Competition for patient resources
- Competitive trials
- Prior usage of EGFR blockade expected to slow down development of IPH2201 in combination with cetuximab

IPH2201

Chronic Lymphocytic Leukemia

CLL incidence and prognosis

- Clonal expansion of morphologically mature, immunologically less mature $CD5^+$ and $CD23^+$ B lymphocytes
- Most common adult leukemia in western world[1]
  - > Incidence 4.2/100,000/year
  - > >15,500 new cases and >4,500 deaths in US estimated in 2013
- Disease of elderly
- Heterogeneous disease[2]
  - > Variable prognosis
    - From never requiring treatment to 18 month median survival
  - > Problems include cytopenias, infections, constitutional symptoms
- Incurable with current standard therapies
- Natural history of active disease may be changing with new therapies[3]

1.National Cancer Institute. SEER stat fact sheets: chronic lymphocytic leukemia;
2. Mertens D, JCO 2014; 3. Hallek M, Blood 2013

Changing landscape of CLL

- Rapidly evolving therapeutic landscape with recent approval of new agents active in poor-risk patients [6,7]
  - > High response rate and prolonged responses, but mostly PRs, few CRS
- Redefining unmet needs
  - > High response rate (>20%?)
  - > Durable responses
  - > Safety
  - > High-risk patients

6. Byrd JC, N Engl J Med 2013; 7.Goede V, N Engl J Med 2014;

Approved agents for CLL

- Cyclophosphamide
- Chlorambucil
- Fludarabine
- Alemtuzumab 2007
- Bendamustine 2008
- Ofatumumab 2009 AA
- Rituximab 2010
- Obinutuzumab 2013
- Ibrutinib 2014 AA
- Idelalisib 2014 (submitted)?

Idelalisib combined with rituximab in poor-risk patients with relapsed CLL[14]

- Randomized double-blind placebo controlled trial in 220 patients with decreased renal function, prior chemo-induced myelosuppression or co-morbidities
- Prior rituximab in 187 patients
- Ide + R vs. Placebo + R
  - > ORR: 81% vs.13% (p<0.001), no CR
  - > Med PFS: not reached vs. 5.5 months (p<0.001)
  - > 1-yr survival rate: 92% vs 80% (P<0.02)

14.Furman R, N Engl J Med 2014;
Breakthrough Therapy designation, NDA submitted 13/12/06

Accelerated approval in relapsed/refractory CLL

	N	CR	CR+PR	Response duration	Ref
Ofatuzumab	59	0%	42%	6.5 mos	FDA
Ibrutinib	48	0%	58.3%	5.6-24.2 mos	FDA
ABT-199 (Phase I)?	67	23%	84%		Seymour,[15] ASH 2013

15.Seymour JF, ASH 2013 abstract # 872

Salvage regimens for CLL

- Ibrutinib [6] (ORR 71%, at 26 mo PFS 75% and OS 83%)
- FCR (ORR 70%, CR 24%, PFS 30.4 mos) [8]
- Bendamustine + rituximab (ORR 59%, CR 9%, PFS 14 mos) [9]
- Lenalidomide ± rituximab (ORR 66%, CR 12%, PFS 17 mos) [10]
- Ofatumumab: 50% response, PFS 6 mos, and does not work in bulky del(17p13.1) [11]
- High-dose methylprednisolone + rituximab : 30-50% response, PFS ≈12 mos, but very immunosuppressive [12]
- Alemtuzumab: ORR 33%, PFS ≈ 6-12 mos [13]

6. Byrd JC, N Engl J Med 2013; 8. Hallek M, Lancet 2010; 9. Fischer K, JCO 2012;
10. Badoux XC, JCO 2013; 11. Wlerda WG , JCO 2010;
12. Pleckyte R , Leuk Lymphoma 2011; 13. Flegl M, Ann Hematol 2014

Rationale for testing IPH2201 in CLL

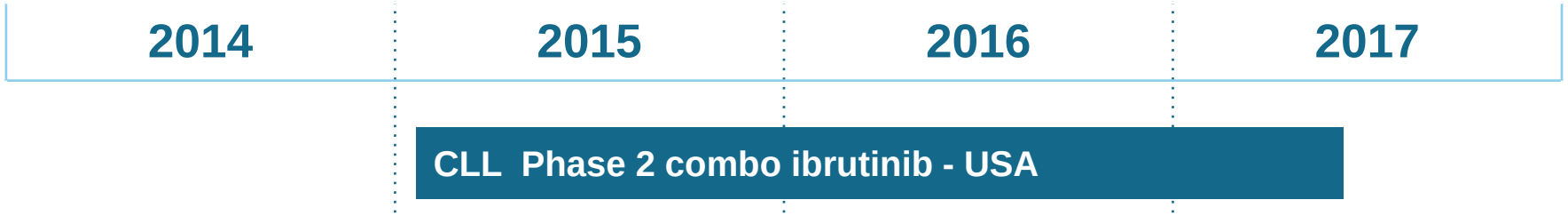
- Consistent and strong expression of HLA-E in CLL cells
  - > 7/7 (IPH data on file)
- NK cell count predictive of disease progression in newly diagnosed CLL[16]
- NK cells from B-CLL patients seem functionally and phenotypically competent, but weak cytolytic activity against primary B-cells without cytokine activation. NK cell activation results in greatly enhanced antitumor activity of rituximab in NSG mice[17]
- Immunomodulation of ibrutinib favors Th1 response and could lead to additive or synergistic effect with IPH2201[18]
- No influence of ITK inhibition on direct NK cell cytotoxicity or CD8 T-cells

16. Palmer S, Brit J Haematol 2008; 17. Veuillen C, J Clin Immunol 2012; 18. Dubovsky JA, Blood 2013

Development options for IPH2201 in CLL

- Single agent
  - > Refractory disease, poor-risk patients
- Combination regimens
  - > Ibrutinib (Causes Th1 polarization)
  - > Anti-CD20 antibody (obinutuzumab)
  - > Lenalidomide (activates NK-cells, T-cells)
- Others

Phase 2 trial of IPH2201+ ibrutinib in relapsed CLL



Patient selection:

- Relapsed refractory CLL
- ≥ 2 prior regimens
- PS 0-1
- Neutro ≥ 750, platelets ≥ 50,000

Regimen:

- Ibrutinib: 420 mg/d p.o. + IPH2201: 10 mg/kg IV q 4 weeks
- Until PD or untoward toxicity

Optimum 2-stage Simon design

- 10 – 29 evaluable patients

Primary endpoint

- CR (International workshop on CLL; bone marrow biopsy confirmation)

Goldman Sachs
March 2014 CLL Survey

- « *Imbruvika's long term potential to generate $5 Bn sales in CLL* »
- « *Imbruvika has best-in class profile to achieve 44% market share of the emerging oral therapies in the long run* »
- « *Physicians expect to have 38% of r/r CLL patients on Imbruvika 12 months post FDA approval* »

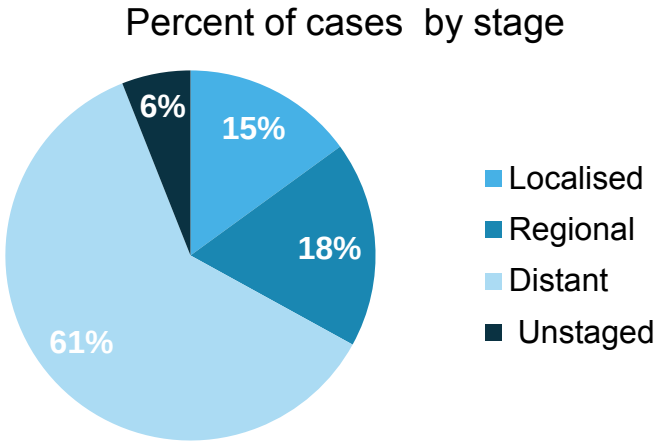
IPH2201

Ovarian Cancer

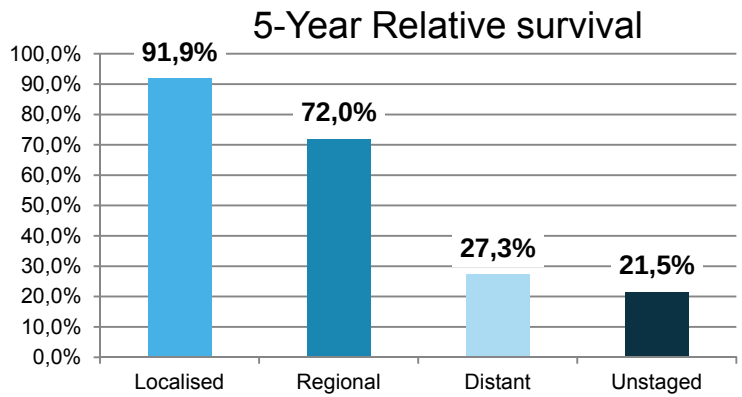
Incidence and prognosis of epithelial ovarian carcinoma (est)[1,2]

- Fifth most common site of cancer in women and fourth most common cause of death from cancer in women

Region	New cases	Deaths
US	20,400	14,400
Europe	65,538	42,704



- A majority of patients have advanced disease at presentation
- Overall 5-year survival is around 40%



1.SEER Fact Sheets : Ovarian Cancer 2003-2009 ;2.EUCAN Cancer Factsheets 2012: Ovary. http://eucancer.iarc.fr/EUCAN/CancerOne

Front-line chemotherapy SOC

Post-operative[3,4]

- Intermediate and high-risk Stage I disease
  - > Single agent carboplatin (3 cycles)
- FIGO Stage II-IV
  - > Carboplatin + paclitaxel combination (6 cycles)
    - Major impact of paclitaxel on survival
    - 70% of patients relapse within 3 years

3. NCCN guidelines ovarian cancer V 2, 2014; 4. ESMO Guidelines. Lederman JA, Ann Oncol 2013

Treatment strategy in recurrent ovarian cancer

Relapse	< 6 mos Platinum resistant[5]	>6 mos Platinum sensitive[6]
SOC	Single agent-sequential	Carboplatin combinations
Med PFS	3-7 mos	HR = 0.80 95% CI, 0.64-1.00 P = 0.05
Med OS	< 12 mos	HR = 0.68 95% CI, 0.57-0.81 P < 0.001
Objective	Symptoms control Quality of life	OS

PFS= Progression free survival;
SOC= Standard of Care;
CT= Chemotherapy

5. Gonzalez-Martin A, Ann Oncol 2013; 6. Raja FA, Ann Oncol 2013

Chemotherapy for platinum resistant disease (Relapse <6 months)

- Sequential single agent therapy
  - > Paclitaxel[11]
  - > Gemcitabine[12]
  - > Topotecan[13]
  - > Pegylated liposomal doxorubicin[14]
- Poor prognosis
  - > ORR <15%
  - > PFS = 3-4 months
  - > OS <12 months

11. McGuire WP, Ann Intern Med 1989; 12.Shapiro JD, Gynecol Oncol 1996; 13.Ten Bokel Huinink W, JCO 1997; 14. Gordon AN, JCO 2001

Bevacizumab in ovarian cancer
Approved in the EU but not in the US

Study	Arms	No. of patients	Median PFS (months)	HR	P-value	Survival advantage
GOG 218[7]	A. Carboplatin + paclitaxel + placebo	625	10.3			
	B. Carboplatin + paclitaxel + bevacizumab + placebo	623	11.2	0.91	0.16	No
	C. Same as in B with maintenance bevacizumab	625	14.1	0.72	<0.0001	Stage IV
ICON7[8]	A. Carboplatin + paclitaxel	764	17.3			
	B. Carboplatin + paclitaxel + bevacizumab with bevacizumab maintenance	764	19.0	0.81	0.004	Patients at high risk for progression'
AURELIA[9]	A. Single agent chemo*	182	3.4			
	B. Single agent chemo* + bevacizumab	179	6.7	0.48	0.001	No
OCEANS[10]	A. Carboplatin + gemcitabine + placebo	242	8.4			
	B. Carboplatin + gemcitabine + bevacizumab	242	12.4	0.48	<0.0001	No

* Weekly paclitaxel,topotecan or pegylated liposomal doxorubicin;

7. Burger R, N Engl J Med 2011; 8.Perren T, N Engl J Med 2011;
9. Pujade-Lauraine E, JCO 2014; 10. Aghajanian C, JCO 2012

New agents in randomized Phase 2 trials for recurrent ovarian cancer

- Sorafenib
- Pazopanib
- Volasertib
- Cabozantinib
- MLN8237
- LY2228020
- MK1775
- Olaparib
- Combretastatin
- Opsalin
- EP-100 (LH-RH)
- NGR-hTNF (CD13)
- Panitumumab (EGFR)
- MM121 (Her3)
- DNIB0600A (ADC)
- VTX2337 (TLR8 agonist)
- Reolysin
- Belotecan
- BNC105P
- SGI-110

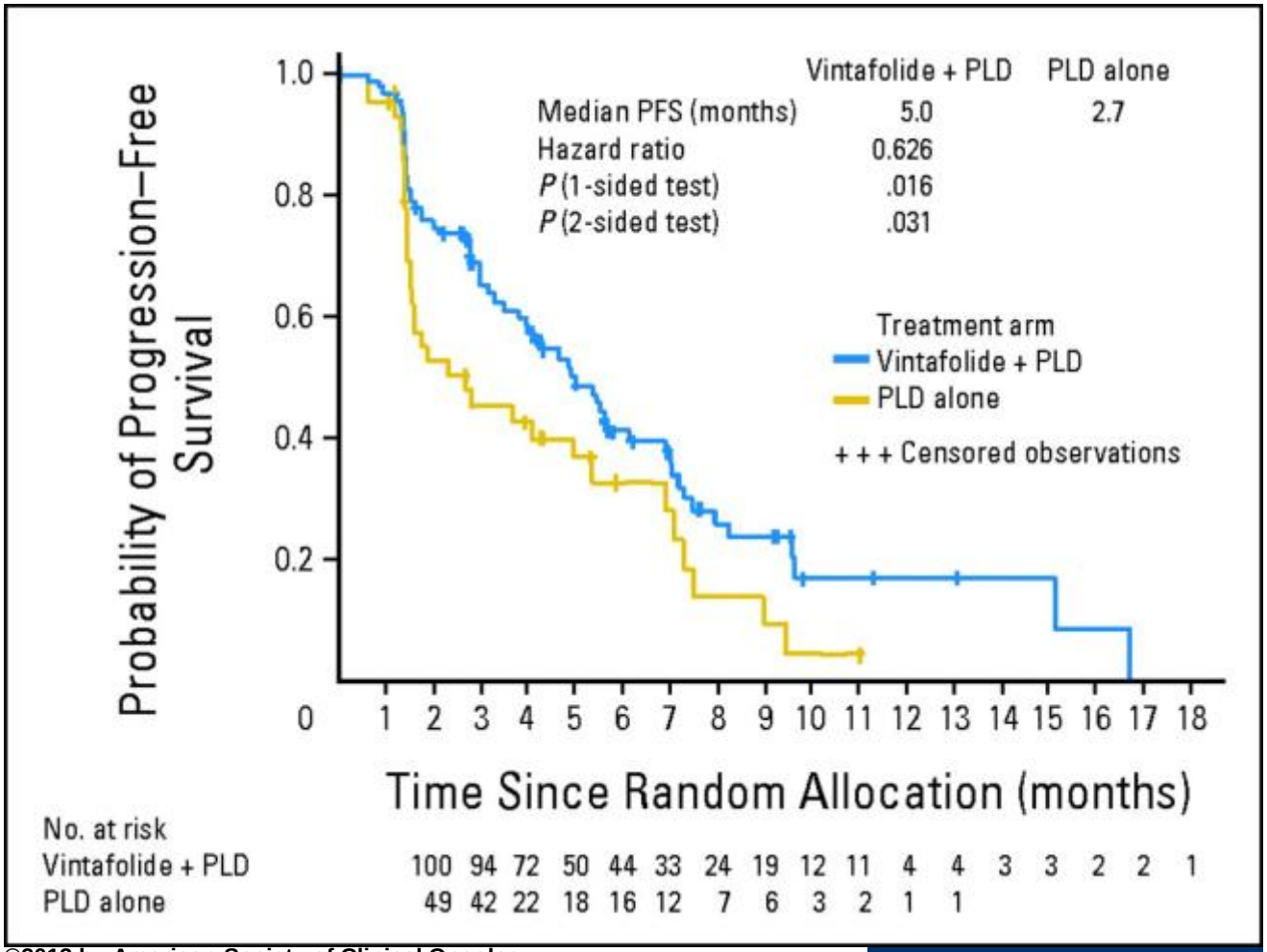
Ongoing randomized Phase 3 trials in recurrent ovarian cancer

Start/ End dates	Design	Co	No. of prior regimens	Months from platinum	Primary endpoint	No. of patients	No. of sites	Sites locations
2013/ 2018	D + Trabedectin *vs.* D	JNJ	≤2	> 6	OS	670	105	WW
2010/ 2017	P + Trebananib *vs.* P	AMG	≤3	1 - 12	PFS	919	246	WW
2011/ 2018	D + Trebananib *vs.* D	AMG	≤3	1 - 12	PFS	223	109	WW
2011/ 2016	D + Vintafolide* *vs.* D	MRK	≤2	< 6	PFS	640	171	WW
2014/ 2016	Rucaparib *vs.* Placebo	Clovis	1	> 6	PFS	540	95	WW

* Vintafolide: CHMP positive opinion on March 20 ,2014; recommended for conditionnal approval based on PFS superiority of the combination vs PDL alone;

D= Doxil/Caelyx; P= paclitaxel

Vintafolide+PDL is the 1st combination to show improvement of PFS over standard therapy in platinum resistant EOC



JOURNAL OF CLINICAL ONCOLOGY ASCO

Naumann R W, JCO 2013

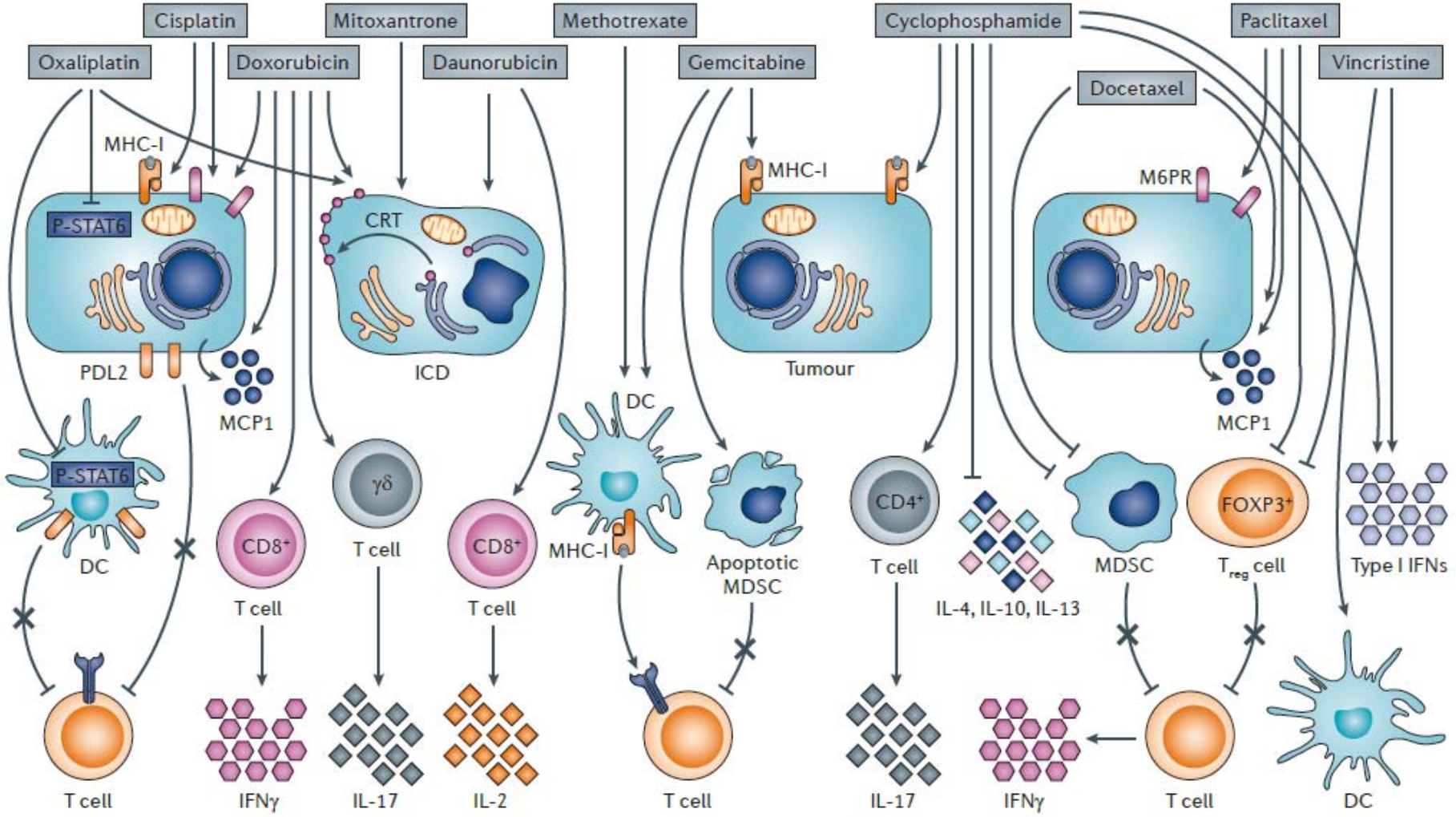
Rationale for testing IPH2201 in ovarian cancer

- HLA-E overexpression
  - > NN (n=57): 68%
  - > Gooden (n=270): 82% [15]
- The presence of TILs correlates with improved outcome[16], [16b] especially in those cancers with high HLA-E expression[17]
- CA125 (ovarian tumour marker) inhibits the cytotoxic responses of human natural killer (NK) cells and down-regulates CD16 (selective binding to $CD16^{+}$ $CD56^{dim}$ NK cells noted especially in ascites) [18]
- High unmet need
- Clear path to registration

Paclitaxel, Doxil/Caelyx, topotecan and gemcitabine appear to be good candidates for chemo- immunotherapy regimens

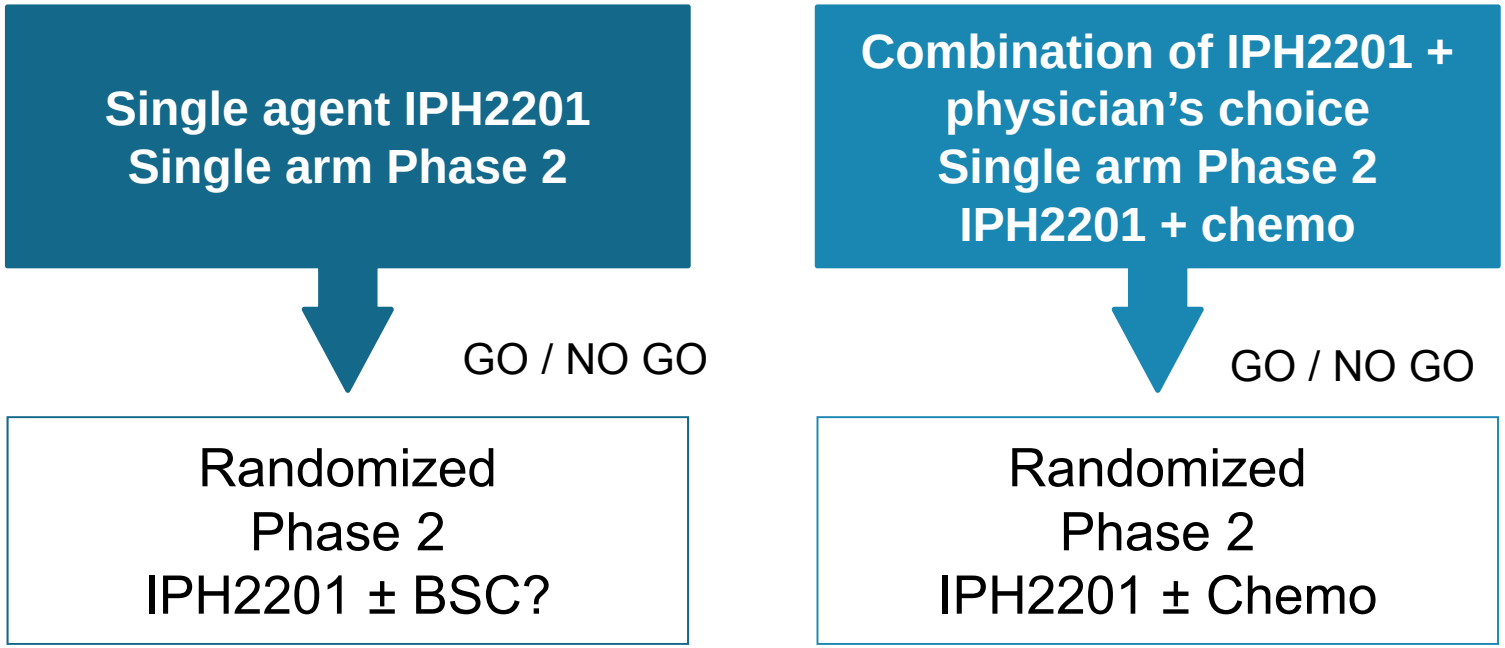
15. Gooden MJM, OncoImmunol 2012; 16. Zhang L, N Engl J Med 2003; 16b. Sato E, PNAS 2005; 17. Gooden, MJM PNAS 2011; 18 Belisle JA, Immunology 2007

Mechanisms through which targeted anticancer agents affect the immune system



Galluzzi L, Nat Rev Drug Disc 2012

Early development of IPH2201 in platinum-resistant ovarian cancer



Phase 2 trial of single agent IPH2201 in ovarian cancer

Patient selection:

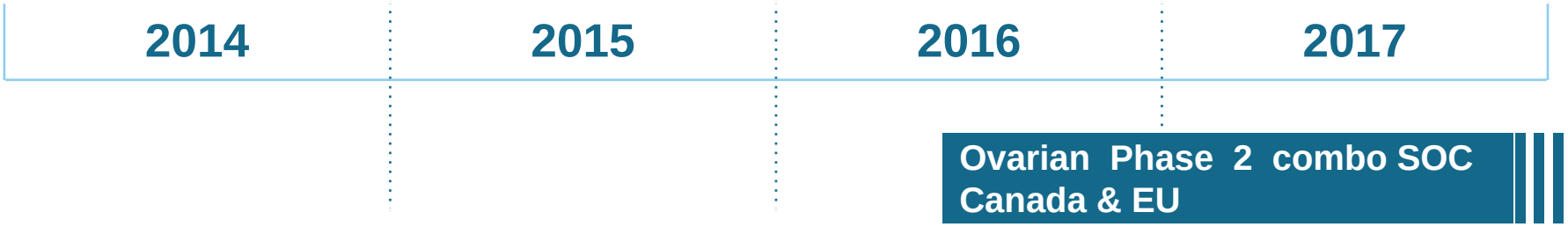
- Platinum resistant ovarian, peritoneal or tubal carcinoma with progression within 1-6 months of completing platinum-based chemotherapy
- ≤ 2 prior regimens for recurrent disease
- PS 0-2

Regimen:

- IPH2201, 10 mg/kg IV q4 weeks up to progression or untoward toxicity

- N = 25
- Primary endpoint: PFS at 4 months

Phase 2 trial of IPH2201 combined with chemotherapy in platinum-resistant ovarian cancer



Patient selection:

- Platinum-resistant disease (disease progression within <6 months of platinum therapy)
- ≤ 2 prior chemotherapy regimens
- ECOG performance status of 0-2

Optimum 2 –stage Simon design
- 15 – 35 evaluable patients
- Primary endpoint: Overall response rate

Regimen:

- Physician choice + IPH2201 10 mg/kg IV q4 weeks
- Physician choice: Gemcitabine, topotecan or Doxil/Caelyx (Pegylated liposomal doxorubicin,PLD)
- Until disease progression or untoward toxicity

Challenges

- Competition for patient resources
- Risk of poor-risk patient selection in early clinical trials
- Very compelling results needed for IPH2201 to move up the priority list of clinical trials
- Absence of harmonization of approval requirements complicates patient selection and study designs across geographic areas

Summary of clinical development plan of IPH2201

Five targeted phase 2 trials

Heme and solid tumors

Single agent and combinations

Rapid clinical proof of principle

Financial update

Catherine Moukheibir, EVP Finance

R&D Update, NY, April 10, 2014

Financial baseline at March 31, 2014

- Cash on hand: €37 million at March 31, 2014
- Guidance
  - > Maintain guidance of €12-€14 million of cash spend per year on like-for-like basis, during period 2014-2017 inclusive
  - > Spend on IPH4102 (KIR3DL2) clinical program in this timeframe
  - > Development milestones from BMS for lirilumab program or proceeds from additional partnering not included
- New elements
  - > Build-up of clinical and related development capabilities as Innate Pharma transitions to a multi-program development organization
  - > Spend requirements for IPH2201 (NKG2A) as described in previous session

Financial requirements for IPH2201 2014-2017 inclusive

- IPH2201 clinical development plan as presented
  - > Solid tumors and onco-hematology
  - > 5 Phase II trials to be started in 2014 and 2015
  - > About 200 patients
  - > In Europe and North America
  - > Expected to deliver results as of 2016 and 2017

- Total program costs during this timeframe: ~€40 million