Enhancing Checkpoint Inhibitors for the Treatment of Solid Cancers

KOL Call July 2020

BioInvent

FORWARD-LOOKING STATEMENT

This presentation does not constitute or form part of any offer or invitation to purchase or subscribe for, or any offer to underwrite or otherwise acquire, any shares or any other securities in BioInvent International AB ("**BioInvent**"). Neither shall the presentation or any part of it, nor the fact of its distribution or communication, form the basis of, or be relied on in connection with, any contract, commitment or investment decision in relation thereto.

This presentation contains forward-looking statements, which are subject to risks and uncertainties because they relate to expectations, beliefs, projections, future plans and strategies, anticipated events or trends and similar expressions concerning matters that are not historical facts. All statements other than statements of historical fact included in this presentation are forward-looking statements. Forward-looking statements give BioInvent's current expectations and projections relating to its financial condition, results of operations, plans, objectives, future performance and business. These statements may include, without limitation, any statements preceded by, followed by or including words such as "target," "believe," "expect," "aim," "intend," "may," "anticipate," "estimate," "plan," "project," "will," "can have," "likely," "should," "would," "could" and other words and terms of similar meaning or the negative thereof. Such forward-looking statements involve known and unknown risks, uncertainties and other factors, which may cause the actual results, performance or achievements of BioInvent or the industry in which it operates, to be materially different than any future results, performance or achievements expressed or implied by such forward-looking statements. Given these risks, uncertainties and other factors, recipients of this presentation are cautioned not to place undue reliance on these forward-looking statements. The forward-looking statements referred to above speak only as at the date of the presentation. BioInvent will not undertake any obligation to release publicly any revisions or updates to these forward-looking statements to reflect future events, circumstances, anticipated events, new information or otherwise except as required by law or by any appropriate regulatory authority.

The information included in this presentation may be subject to updating, completion, revision and amendment and such information may change materially. No person, including BioInvent and its advisors, is under any obligation to update or keep current the information contained in this presentation and any opinions expressed in relation thereto are subject to change without notice. Neither BioInvent nor any of its owners, affiliates, advisors or representatives (jointly the "**Disclosers**") make any guarantee, representation or warranty, express or implied, as to the accuracy, completeness or fairness of the information and opinions contained in this presentation, and no reliance should be placed on such information. None of the Disclosers accept any responsibility or liability whatsoever for any loss howsoever arising from any use of this presentation or its contents or otherwise arising in connection therewith.

By attending this presentation or by accepting any copy of this document, you agree to be bound by the foregoing limitations.

COMPANY SNAPSHOT

LEADING IMMUNO-ONCOLOGY ANTIBODY PLATFORM

- Advancing Cancer Immunotherapy by overcoming tumor resistance
- Differentiated platform for functional screening to identify new relevant tumor targets and antibodies
- Highly advanced antibody discovery platform with in-house GMP manufacturing facilities

ROBUST PIPELINE FUELED BY WORLD CLASS, FULLY INTEGRATED RESEARCH ENGINE

- Lead product, BI-1206, currently in Phase I/II for relapsed or refractory indolent Non-Hodgkin Lymphoma (iNHL) patients and for relapsed or refractory patients with solid cancers
- Growing portfolio: 2 proprietary programs in the clinic – 4 programs in the clinic by YE'2020

TECHNOLOGY PLATFORM VALIDATED BY DEAL WITH PFIZER

- Discovery of new anti-tumor associated myeloid (anti-TAM) targets and antibodies
- Upfront technology access fee from Pfizer with potential milestones payments of up to >$500 million
- BioInvent maintains participation in future commercial upside with up to double digit royalties

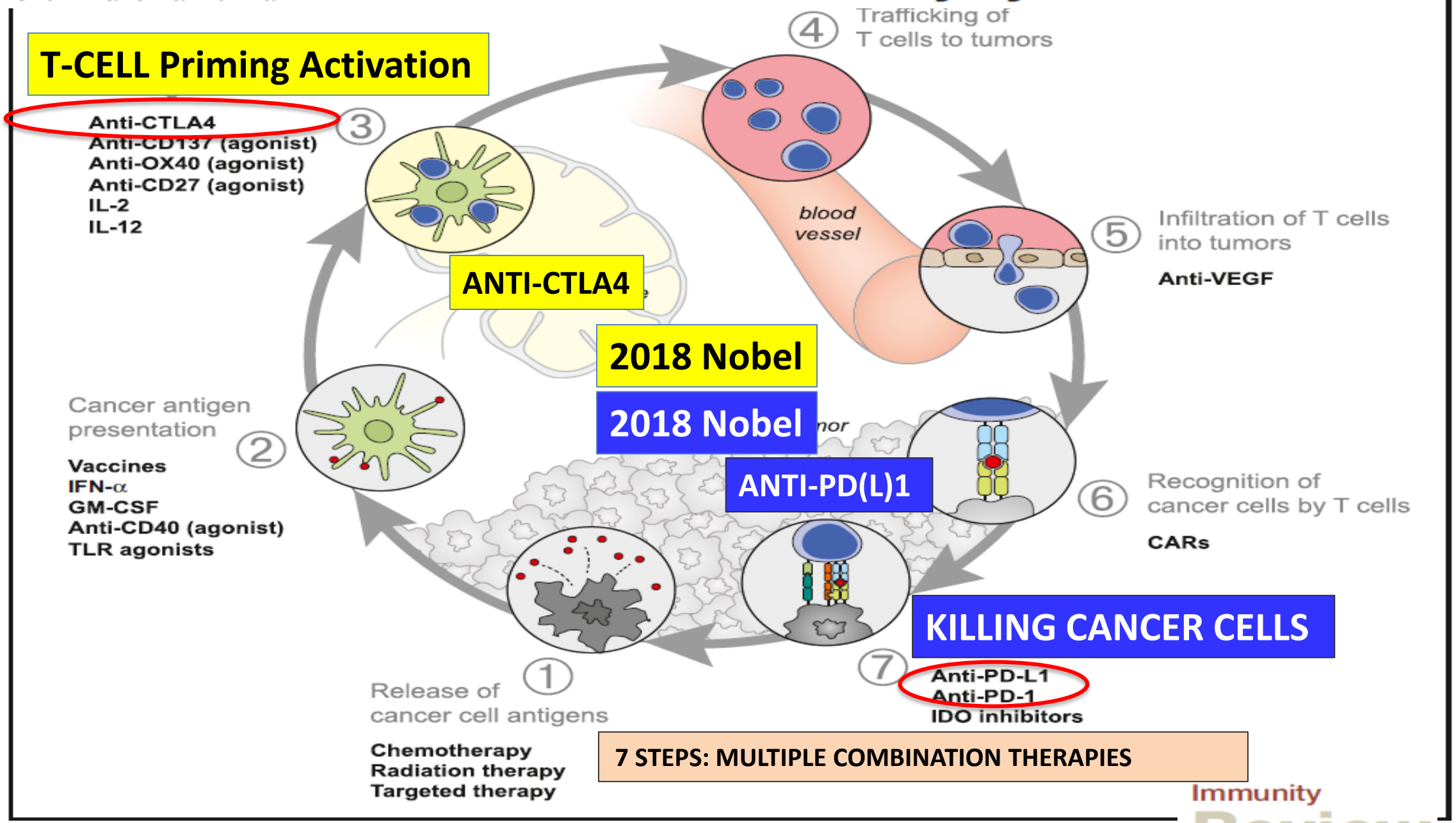
EXPERIENCED MANAGEMENT TEAM AND STRONG INSTITUTIONAL SHAREHOLDER BASE

- Significant senior executive experience with deep scientific and clinical expertise, strong focus on dealmaking
- Shareholders include Van Herk Investments, Omega, HBM, Robur, AP4, Invus, Pfizer, Handelsbanken
- Listed on NASDAQ Stockholm since 2001 (https://www.bioinvent.com/investors/#shareprice)

BI-1206 (anti-FcγRIIB) overcoming rituximab resistance in NHL and enhancing on anti-PD-1 in solid cancer

Daniel S. Chen[1,3] and Ira Mellman[2,3,]

The Cancer-Immunity Cycle



Immunity Review

Figure 3. Therapies that Might Affect the Cancer-Immunity Cycle

IMMUNE SYSTEM BLOCKED AT MULTIPLE LEVELS

- 1) **CTL PRIMING**
  - e.g. CTLA4 ……. **Unblock: anti-CTLA4**
- 2) **CTL EFFECTOR Function**
  - e.g. PD-1 / PDL-1…… **Unblock: anti-PD1/anti-PDL1**
- 3) **MACROPHAGES in Tumor Infiltrate (TAM)**
  - **e.g. Macrophages; MDSC** **Unblock: - anti-FcγR**
    - **Fcγ-R modulation: optimize ICI / overcome resistance**
    - M2-M1 repolarization agents (CCR5; CCR5/CCR2)
- 4) **Various Immune Escape Mechanims**
  - e.g.:
    - JAK1/2 mutations and loss Gamma-IFN pathways
    - B2M mutations, Loss MHC Class I molecules, Loss Recognition
    - B-actenin pathway activation : immune exclusion
    - TOX and T-cell exhaustion
    - Fcγ-Receptor modulation: optimize ICI effect / overcome resistance

JOURNAL OF CLINICAL ONCOLOGY ORIGINAL REPORT

Phase I Keynote-001 :
3 yr 52% and 5 yr 41% survival
Pembrolizumab in advanced melanoma

Pooled Analysis of Long-Term Survival Data From Phase II and Phase III Trials of Ipilimumab in Unresectable or Metastatic Melanoma

Dirk Schadendorf, F. Stephen Hodi, Caroline Robert, Jeffrey S. Weber, Kim Margolin, Omid Hamid, Debra Patt, Tai-Tsang Chen, David M. Berman, and Jedd D. Wolchok

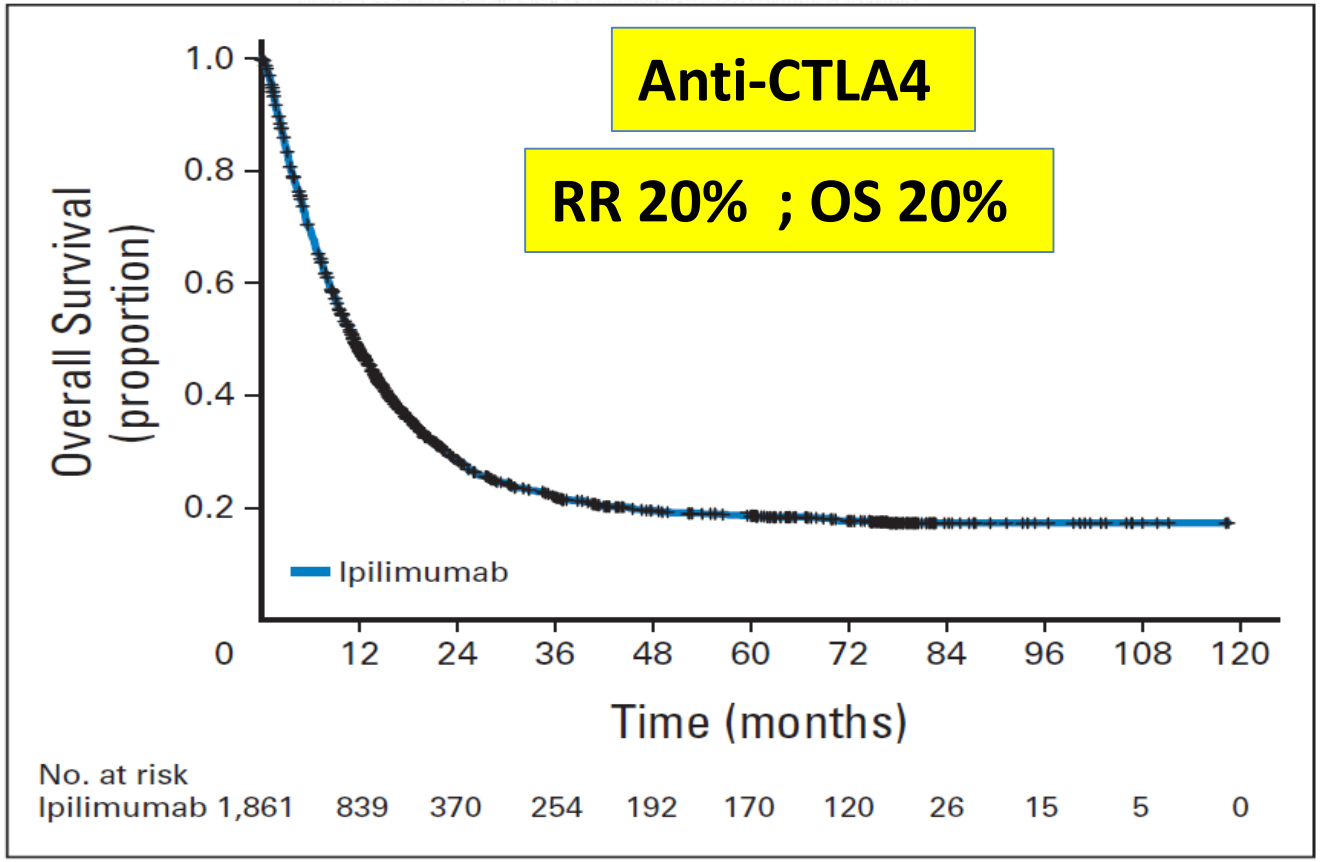
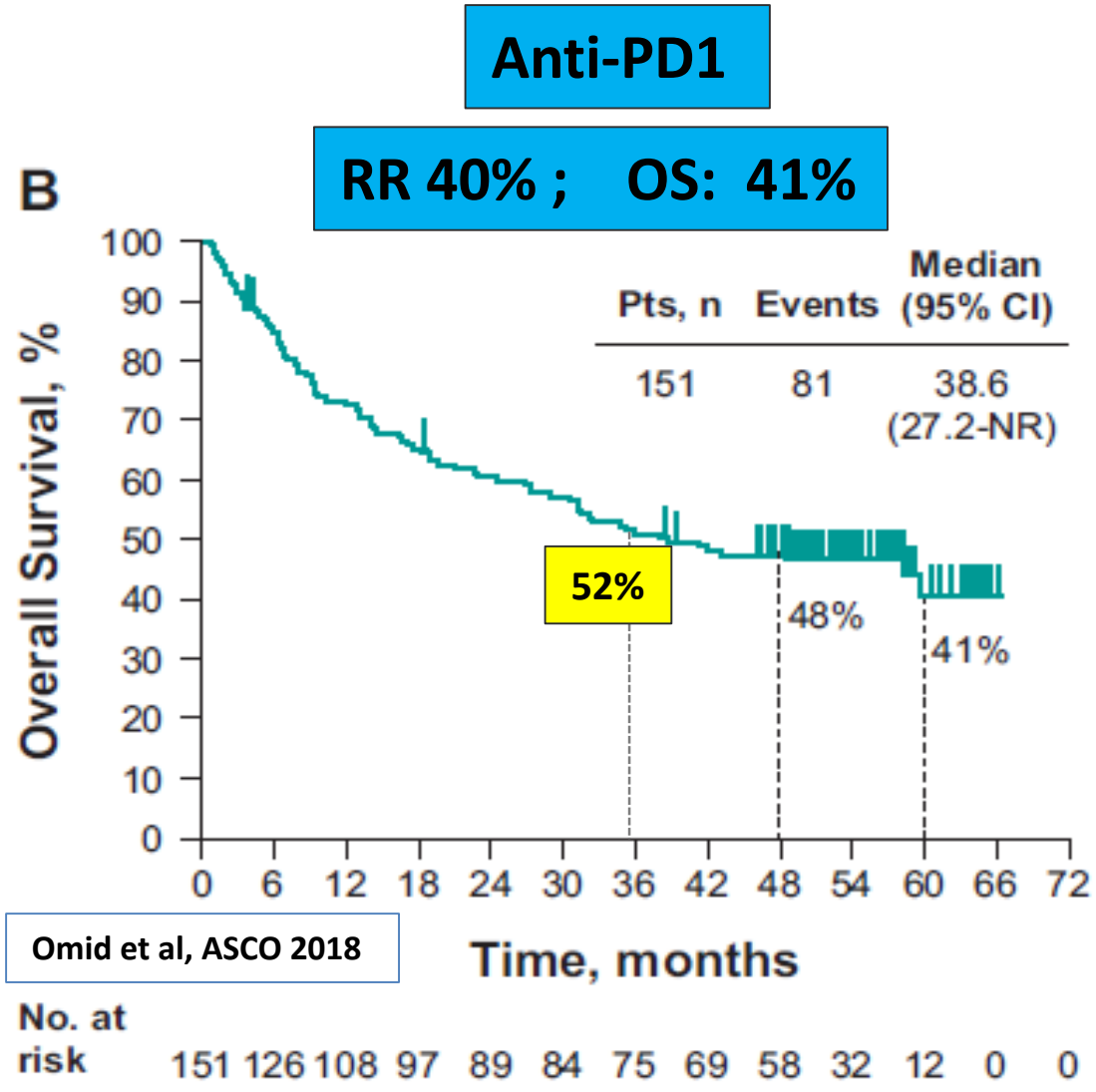


Fig 1. Primary analysis of pooled overall survival (OS) data. Individual patient data were pooled from 10 prospective trials and two retrospective, observational studies of ipilimumab in metastatic melanoma (n = 1,861). Median OS was 11.4 months (95% CI, 10.7 to 12.1 months) with a 3-year survival rate of 22% (95% CI, 20% to 24%). Crosses indicate censored patients.

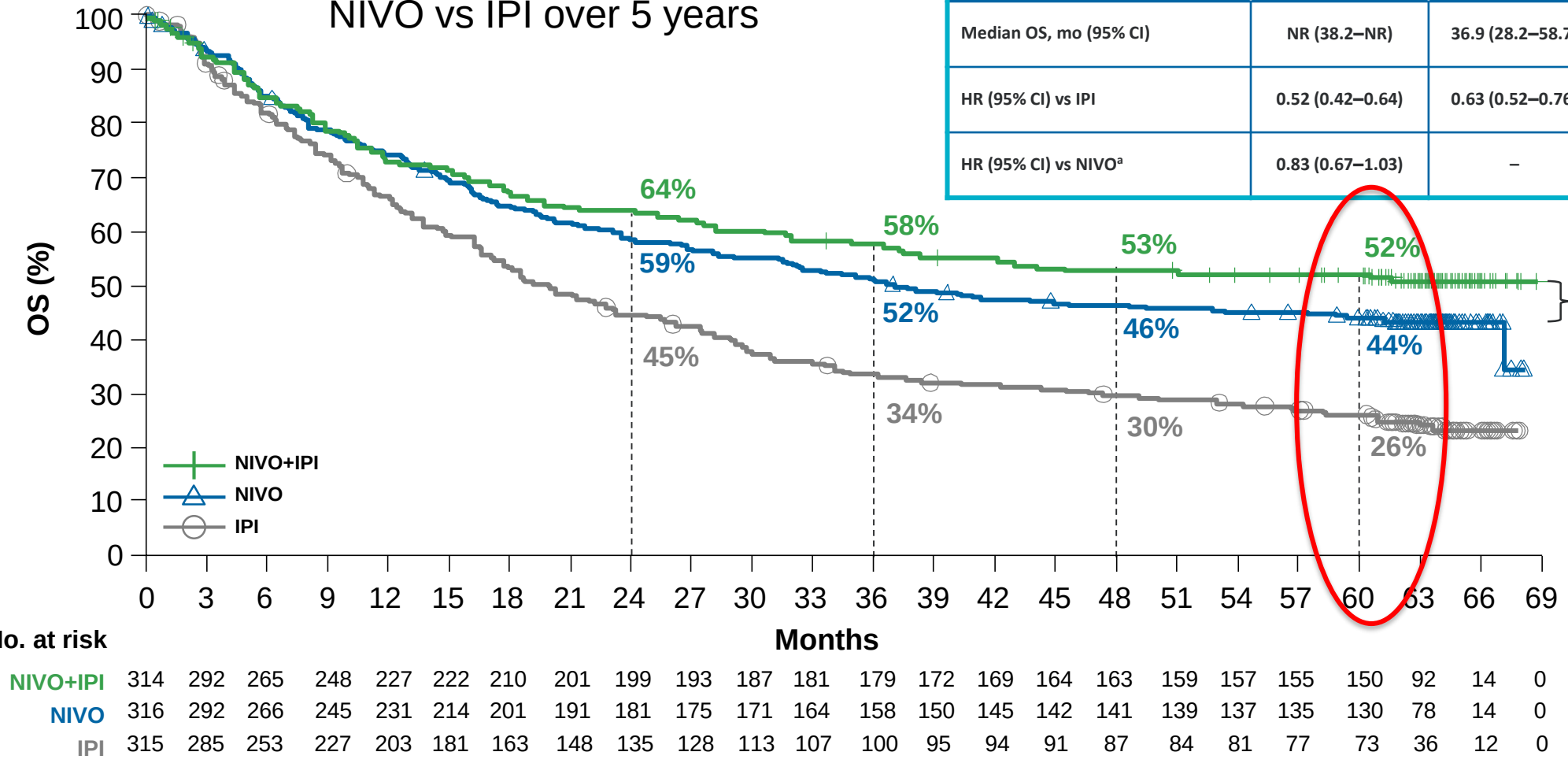


Omid et al, ASCO 2018

NIVO + IPI: 5 Year Overall Survival

- Improved OS with NIVO+IPI and NIVO vs IPI over 5 years

	NIVO+IPI (n = 314)	NIVO (n = 316)	IPI (n = 315)
Median OS, mo (95% CI)	NR (38.2–NR)	36.9 (28.2–58.7)	19.9 (16.8–24.6)
HR (95% CI) vs IPI	0.52 (0.42–0.64)	0.63 (0.52–0.76)	–
HR (95% CI) vs NIVO[a]	0.83 (0.67–1.03)	–	–



No. at risk

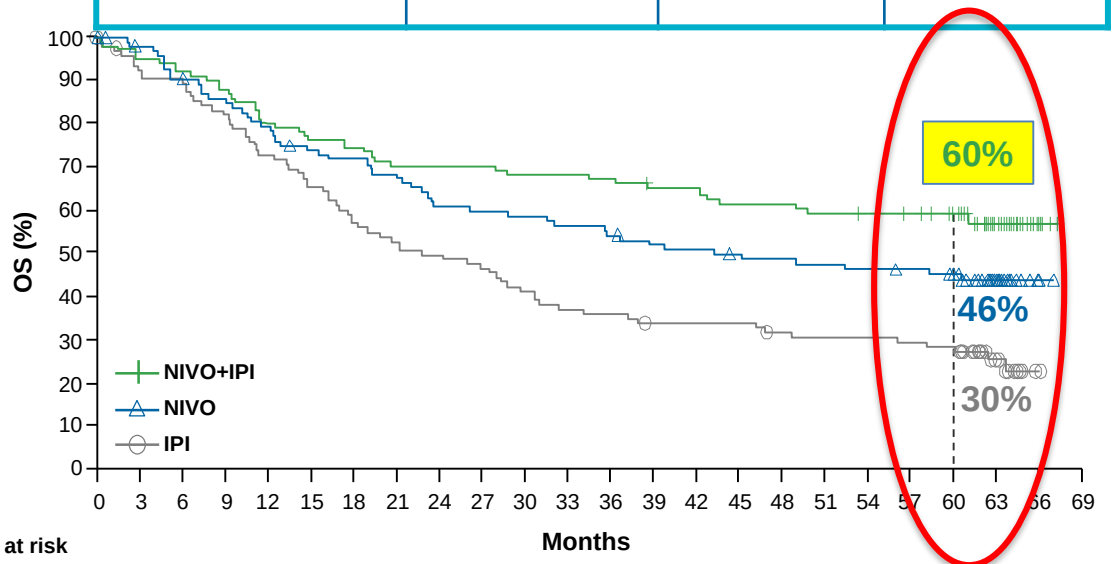
NIVO+IPI	314	292	265	248	227	222	210	201	199	193	187	181	179	172	169	164	163	159	157	155	150	92	14	0
NIVO	316	292	266	245	231	214	201	191	181	175	171	164	158	150	145	142	141	139	137	135	130	78	14	0
IPI	315	285	253	227	203	181	163	148	135	128	113	107	100	95	94	91	87	84	81	77	73	36	12	0

[a]Descriptive analysis. 1. Larkin J, et al. Oral presentation at the AACR Annual Meeting; April 1–5, 2017; Washington DC, USA. Abstract CT075; 2. Wolchok JD, et al. *N Engl J Med* 2017;377:1345–1356; 2. Hodi FS, et al. *Lancet Oncol* 2018;19:1480–1492.

OS in Patients With *BRAF*-Mutant and Wild-Type Tumors

BRAF Mutant

	NIVO+IPI (n = 103)	NIVO (n = 98)	IPI (n = 100)
Median, mo (95% CI)	NR (50.7–NR)	45.5 (26.4–NR)	24.6 (17.9–31.0)
HR (95% CI) vs IPI	0.44 (0.30–0.64)	0.63 (0.44–0.90)	–
HR (95% CI) vs NIVO[a]	0.70 (0.46–1.05)	–	–



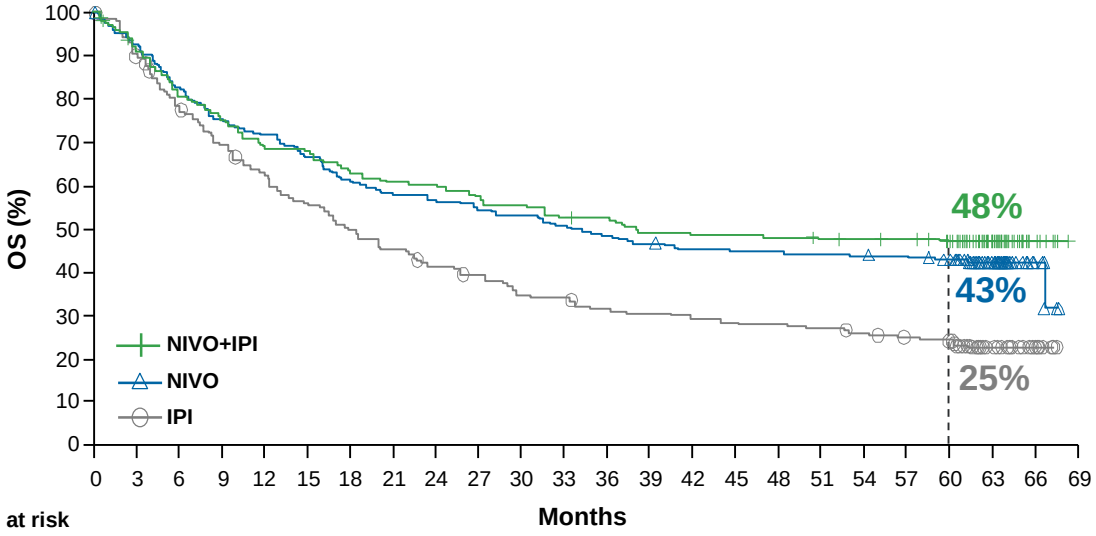
No at risk

NIVO+IPI	103	99	96	91	83	80	77	74	73	73	71	71	70	69	67	63	63	61	60	59	57	37	7	0
NIVO	98	93	86	81	75	69	67	64	57	56	55	53	52	48	47	45	44	43	42	41	40	27	4	0
IPI	100	91	88	81	71	64	58	53	49	47	41	37	36	33	33	33	30	29	29	28	27	13	2	0

- 5-year PFS rates of 38% (NIVO+IPI), 22% (NIVO), and 11% (IPI)

BRAF Wild-Type

	NIVO+IPI (n = 211)	NIVO (n = 218)	IPI (n = 215)
Median, mo (95% CI)	39.1 (27.5–NR)	34.4 (24.1–59.2)	18.5 (14.1–22.7)
HR (95% CI) vs IPI	0.57 (0.45–0.73)	0.64 (0.50–0.81)	–
HR (95% CI) vs NIVO[a]	0.89 (0.69–1.15)	–	–



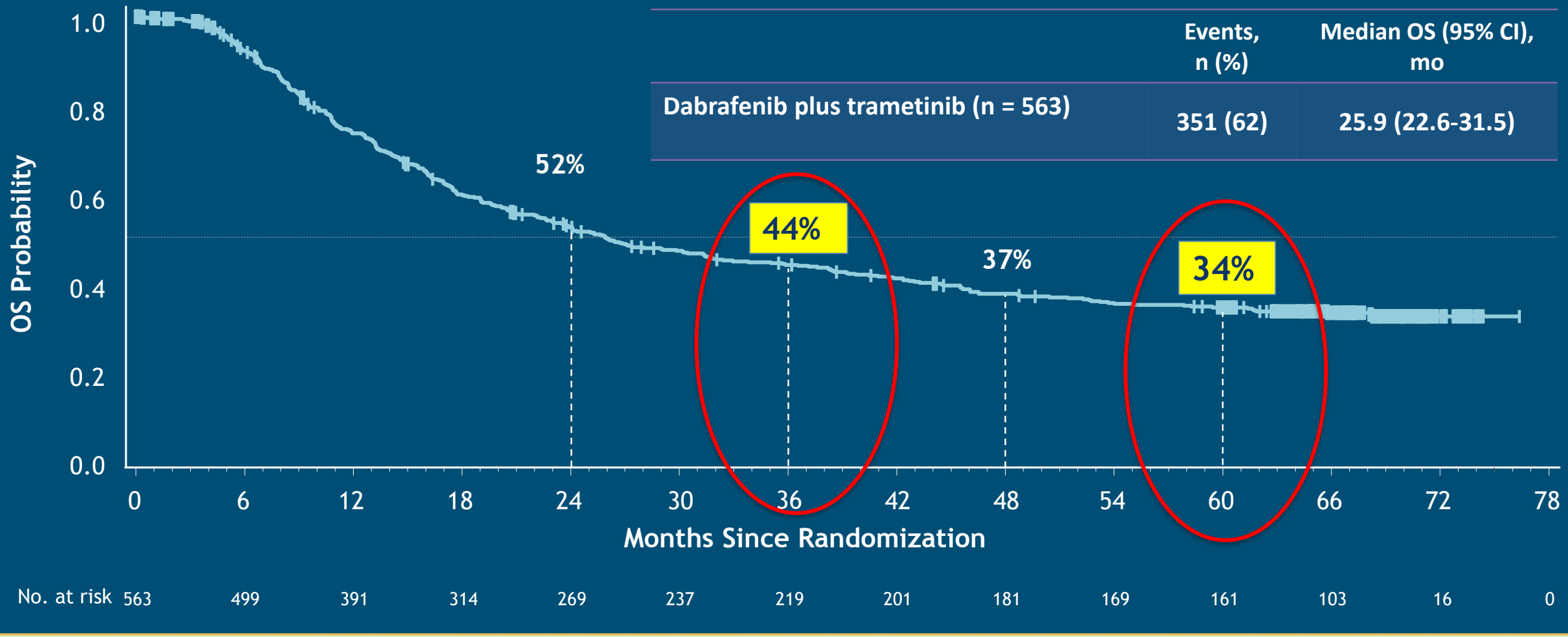
No. at risk

NIVO+IPI	211	193	169	157	144	142	133	127	126	120	116	110	109	103	102	101	100	98	97	96	93	55	7	0
NIVO	218	199	180	164	156	145	134	127	124	119	116	111	106	102	98	97	97	96	95	94	90	51	10	0
IPI	215	194	165	146	132	117	105	95	86	81	72	70	64	62	61	58	57	55	52	49	46	23	10	0

- 5-year PFS rates of 35% (NIVO+IPI), 32% (NIVO), and 7% (IPI)

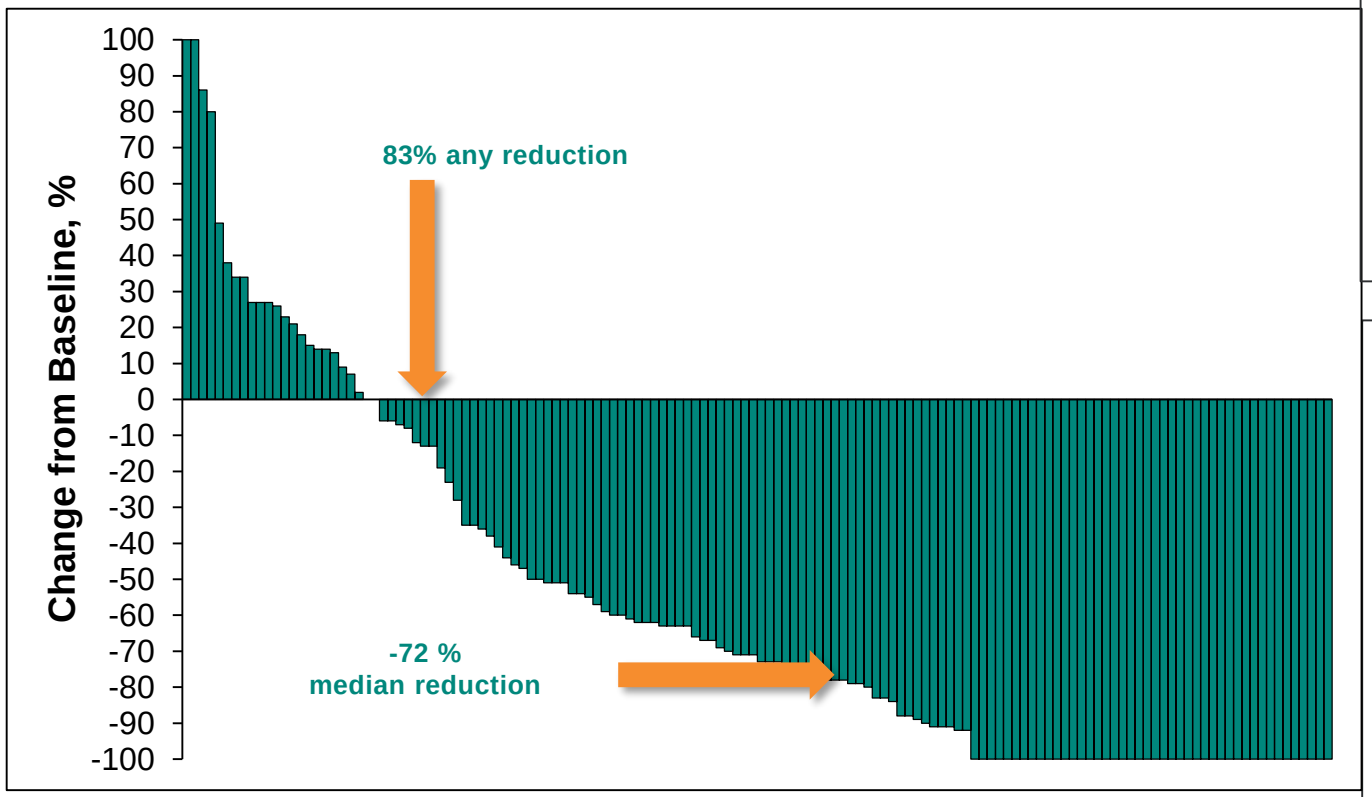
[a]Descriptive analysis.

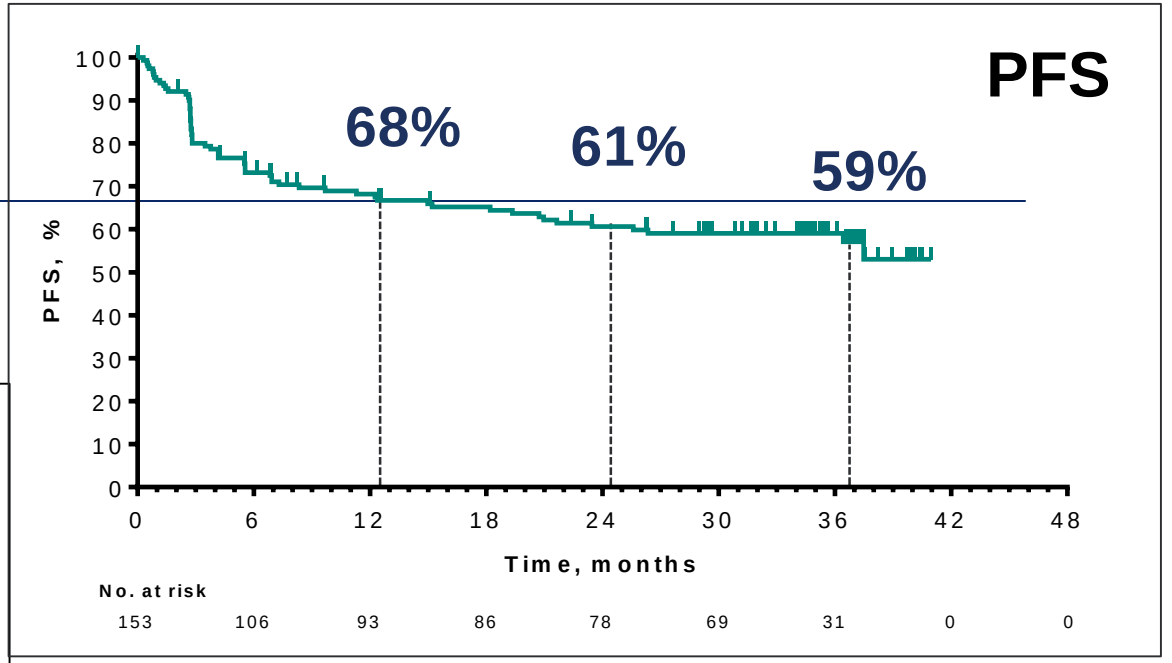
Dabrafenib Plus Trametinib: 3Yr 44% and 5-Yr 34% OS

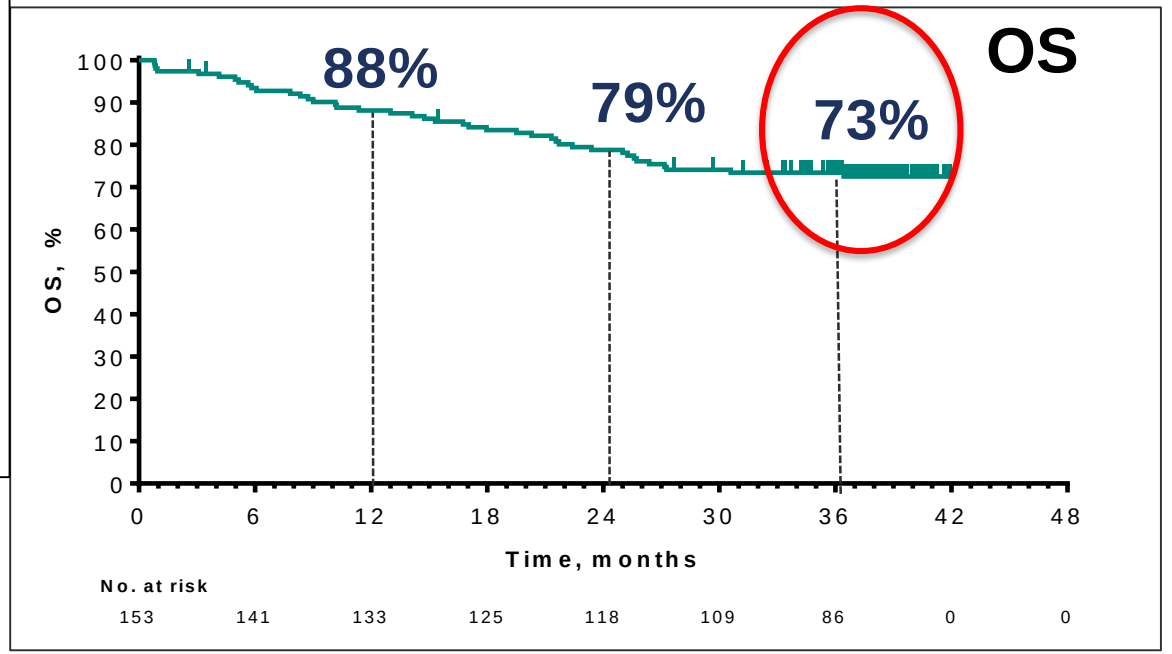


	Events, n (%)	Median OS (95% CI), mo
Dabrafenib plus trametinib (n = 563)	351 (62)	25.9 (22.6-31.5)

KEYNOTE-029 3YR DATA
Pembro + Ipilimumab 1mg

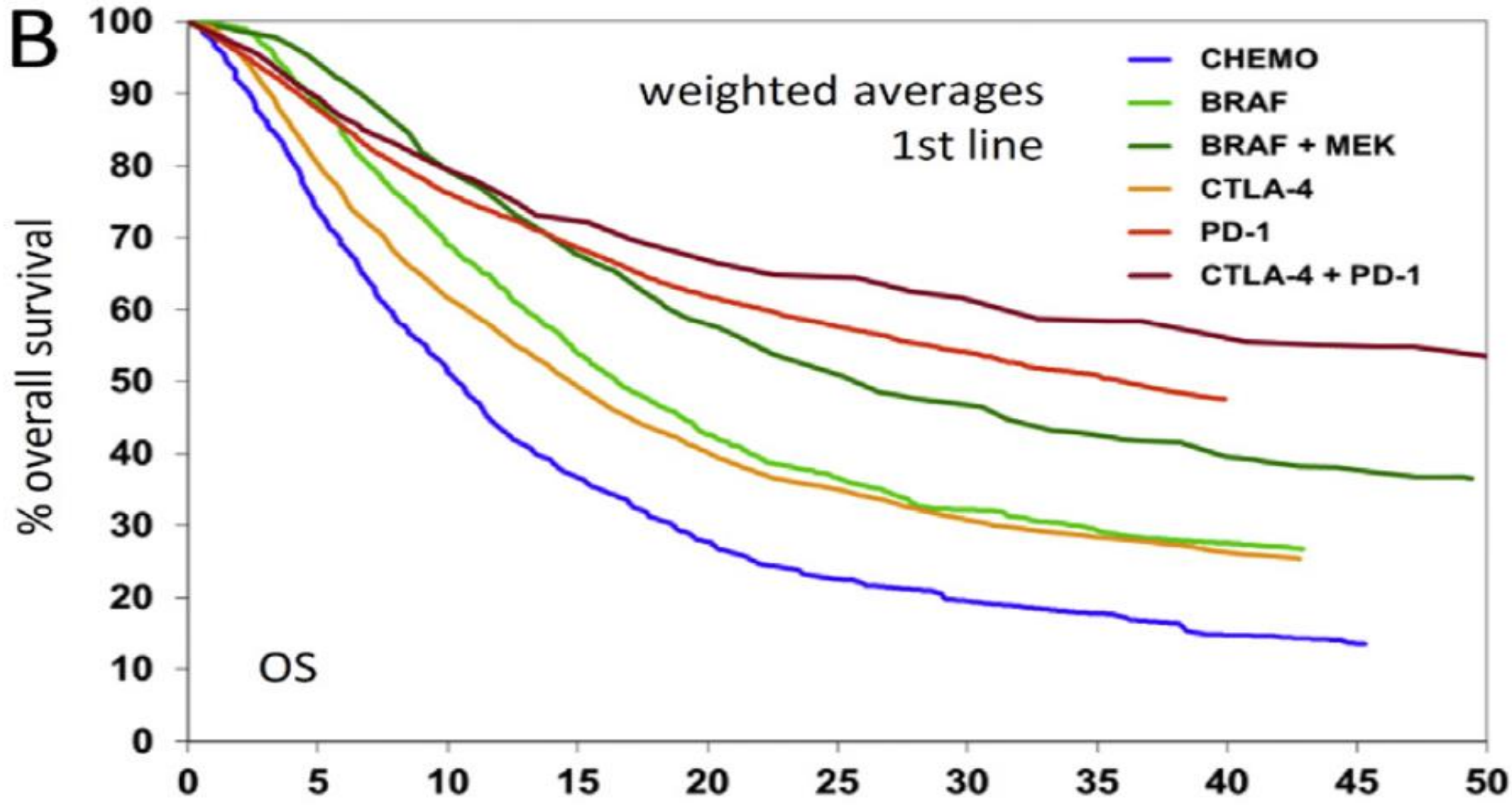






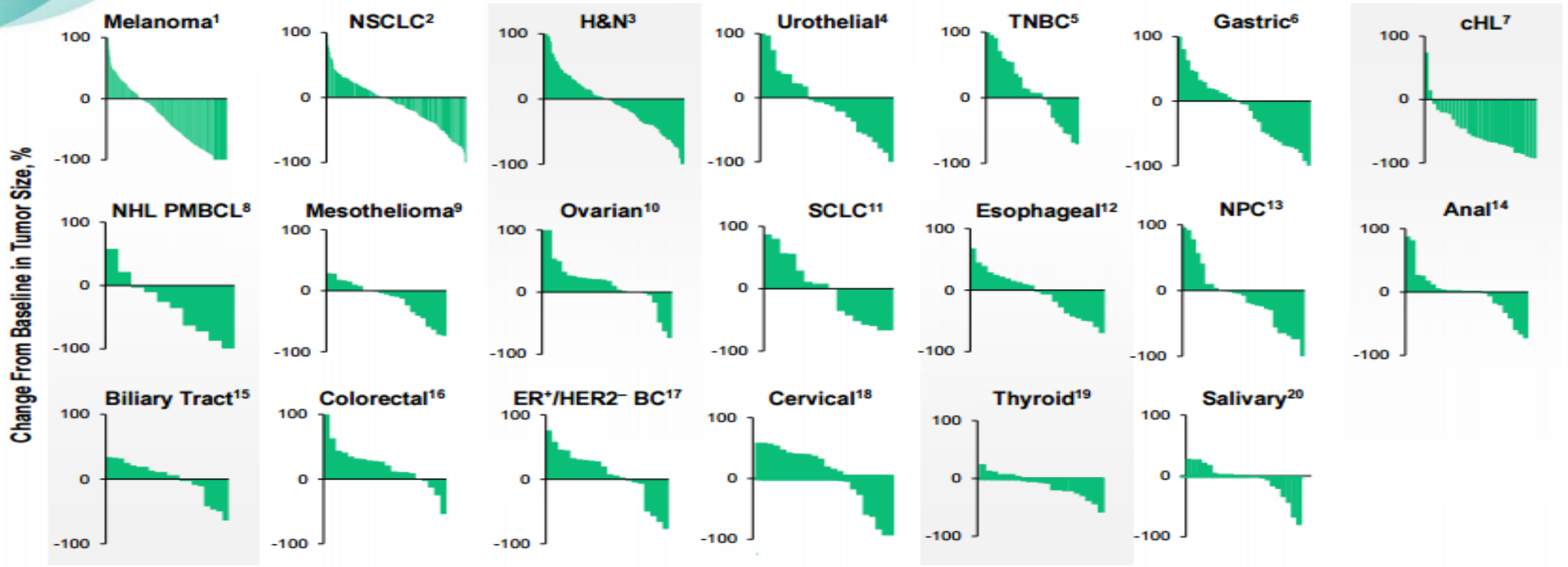
Long GV et al ESMO 2018..

Melanoma: Superiority Immunotherapy 1st line Overall Survival



S. Ugurel et al. / European Journal of Cancer 130 (2020) 126–138

Anti-PD1 demonstrates broad antitumor activity



1. Daud A et al. ASCO 2015; 2. Garon EB et al. ESMO 2014; 3. Seiwert T et al. ASCO 2015; 4. Plimack E et al. ASCO 2015; 5. Bang YJ et al. ASCO 2015; 6. Nanda R et al. SABCS 2014; 7. Moskowitz C et al. ASH Annual Meeting 2014; 8. Alley EA et al. AACR 2015; 9. Varga A et al. ASCO 2015; 10. Ott PA et al. ASCO 2015; 11. Doi T et al. ASCO 2015.

COMBINATION TRIALS WITH Anti-PD(L)-1

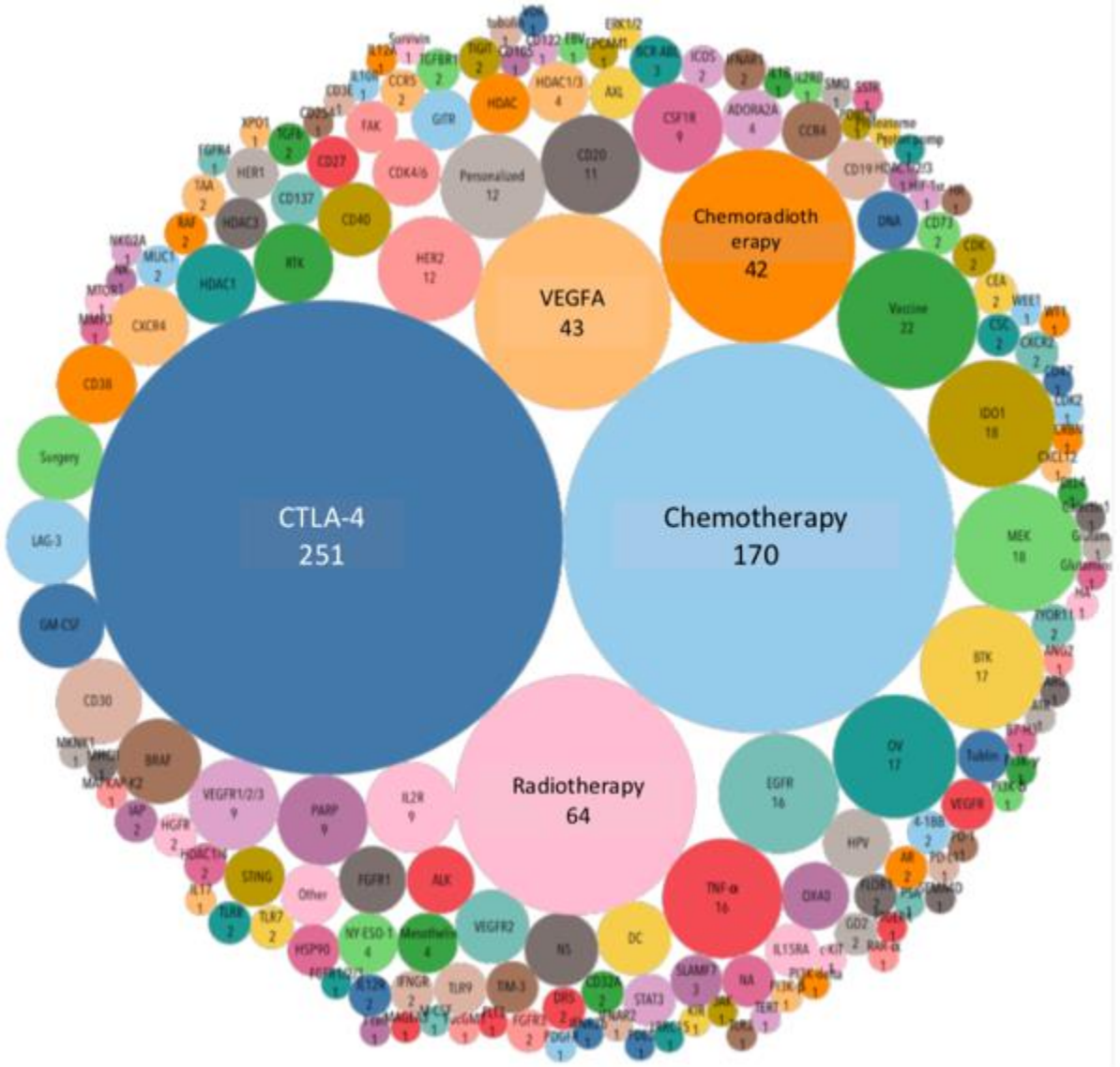
PD-1/L1 COMBO PARTNER ANALYSIS

165 DIFFERENT TARGETS ARE BEING COMBINED

Numbers of Trials Using Common Combo

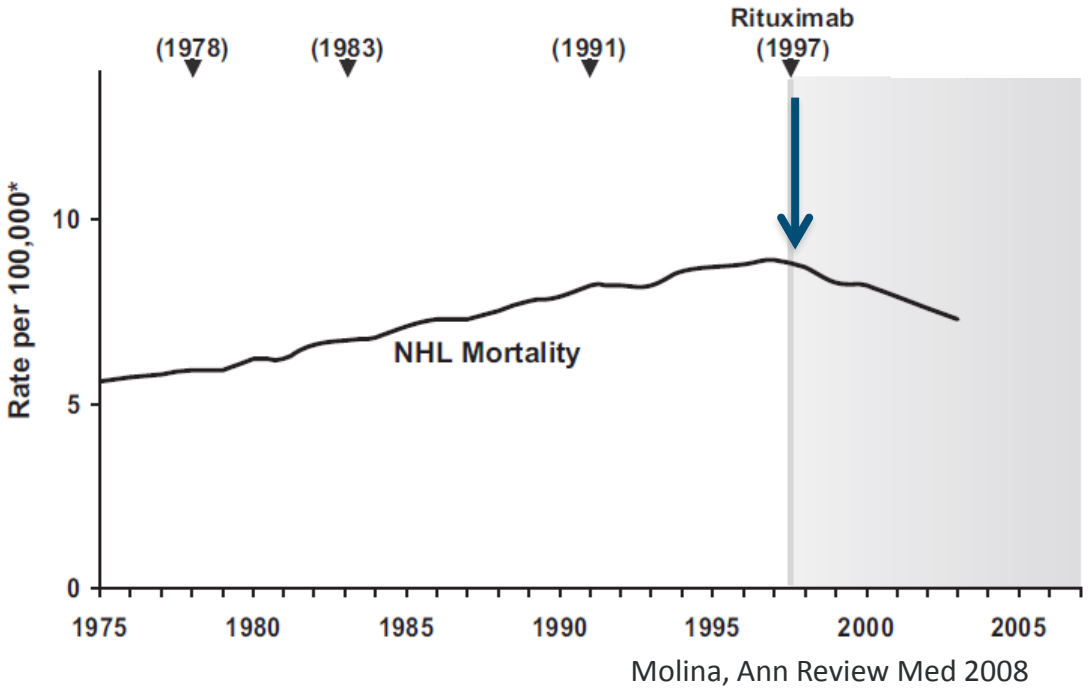
Strategies:

1. Anti-CTLA-4 agents: 251
2. Chemotherapies: 170
3. Radiotherapies: 64
4. Anti-VEGFA agents: 43
5. Chemoradiotherapy combos: 42

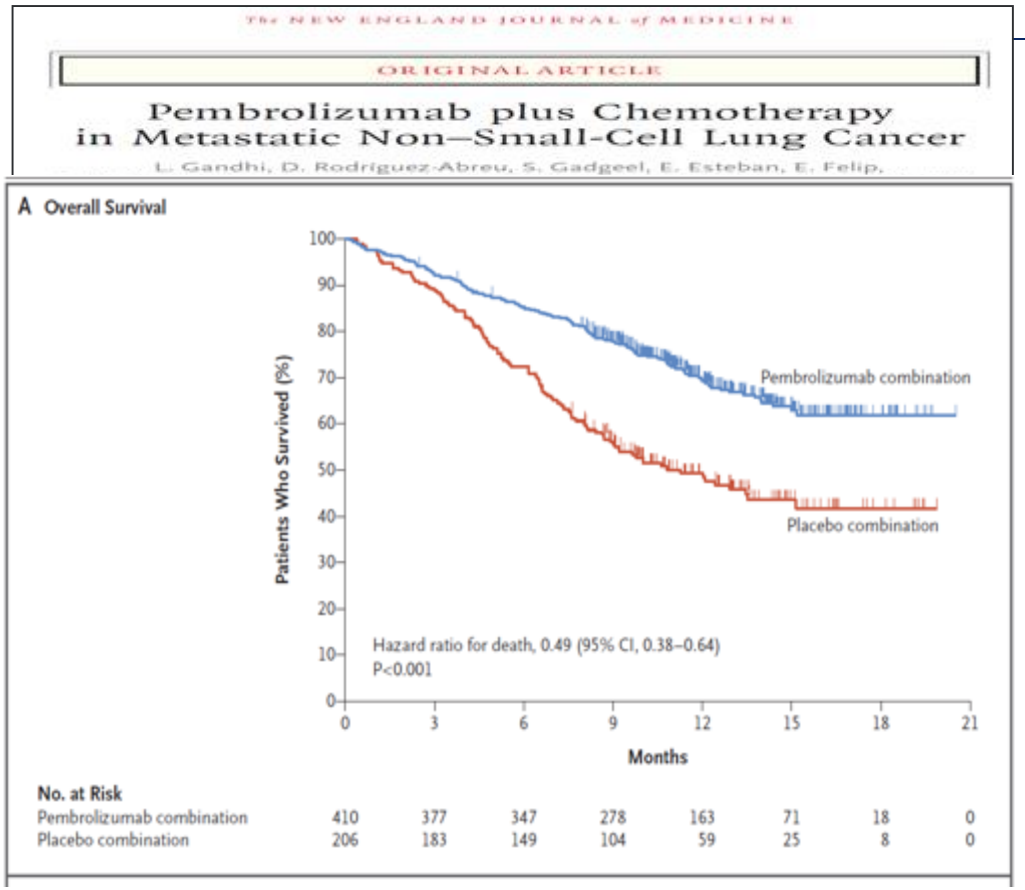


ANTIBODIES HAVE TRANSFORMED CANCER SURVIVAL

Tumor-targeting mAb



Molina, Ann Review Med 2008



- Despite improved OS the majority of cancer patients do not respond or develop resistance to mAb therapy
- Can mechanisms of mAb resistance be identified and overcome?

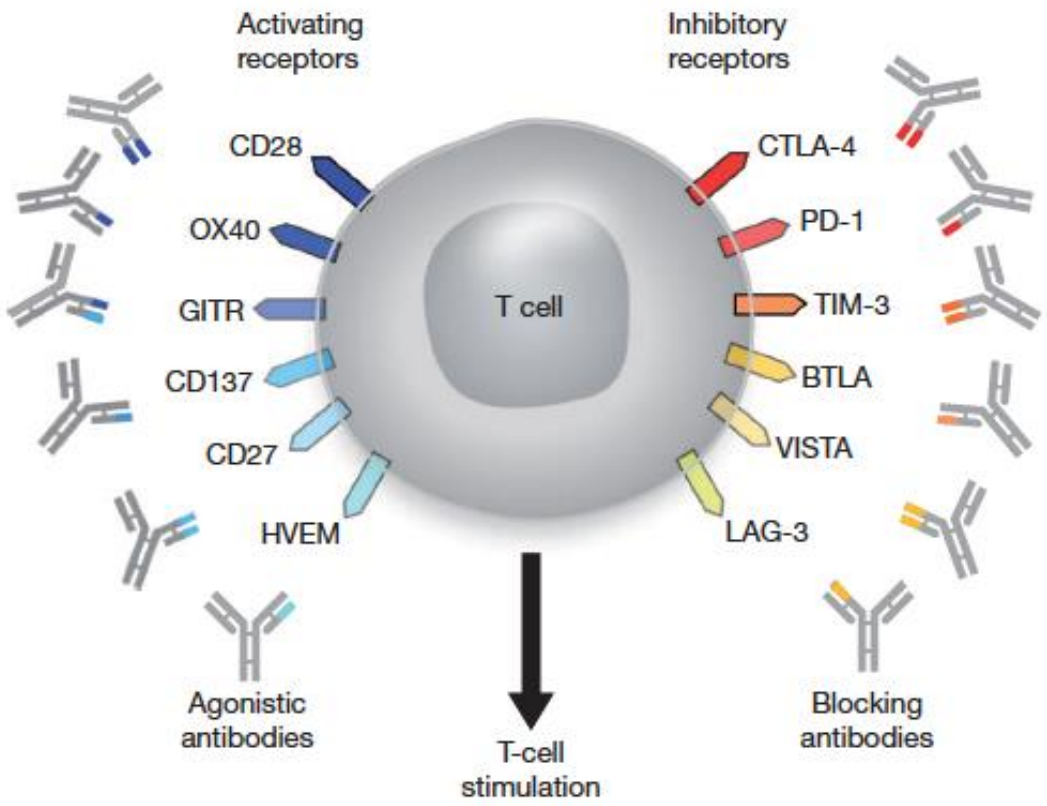
15

REVIEW

doi:10.1038/nature10673

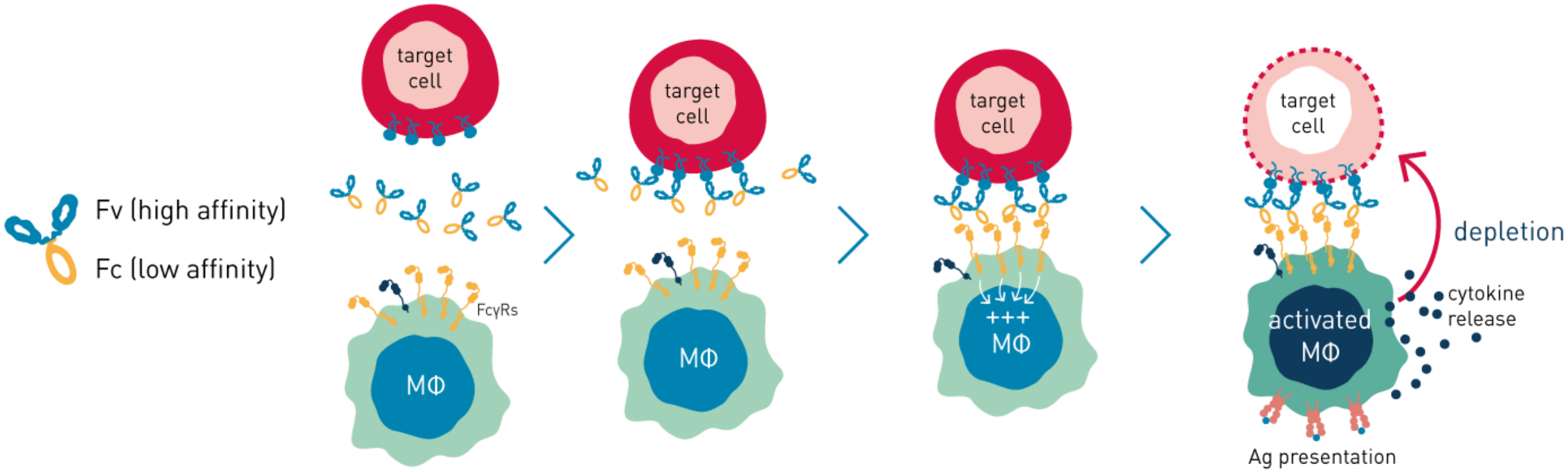
Cancer immunotherapy comes of age - a Role for FcγRs?

Ira Mellman[1], George Coukos[2] & Glenn Dranoff[3]



Mellman, Nature, 2011

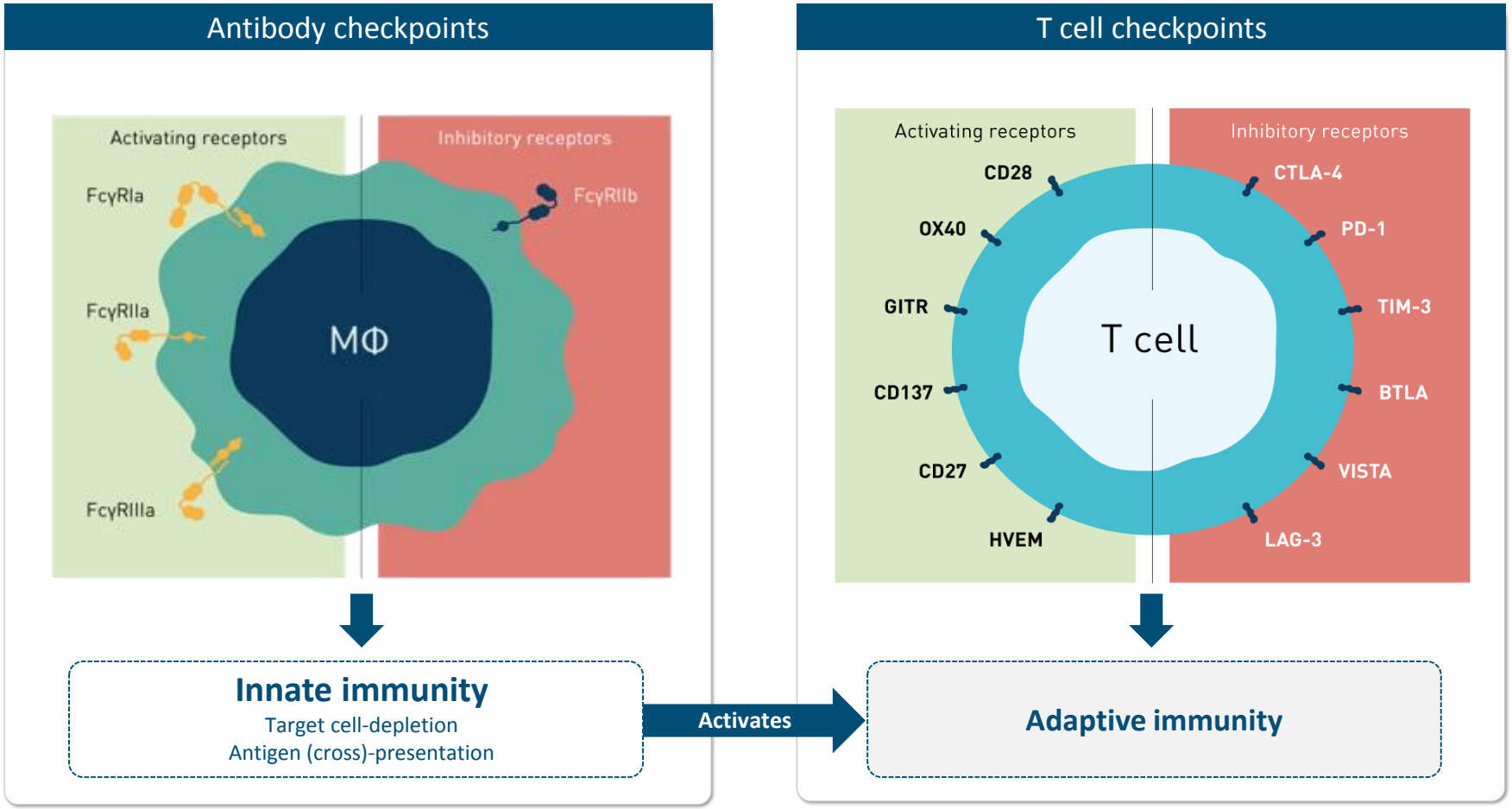
FcgRs MODULATE THE ACTIVITY OF IgG ANTIBODIES



- FcγRs modulate the functional activity of IgG antibodies
  - Antibodies to cell surface receptors
  - Antibodies to multivalent antigens

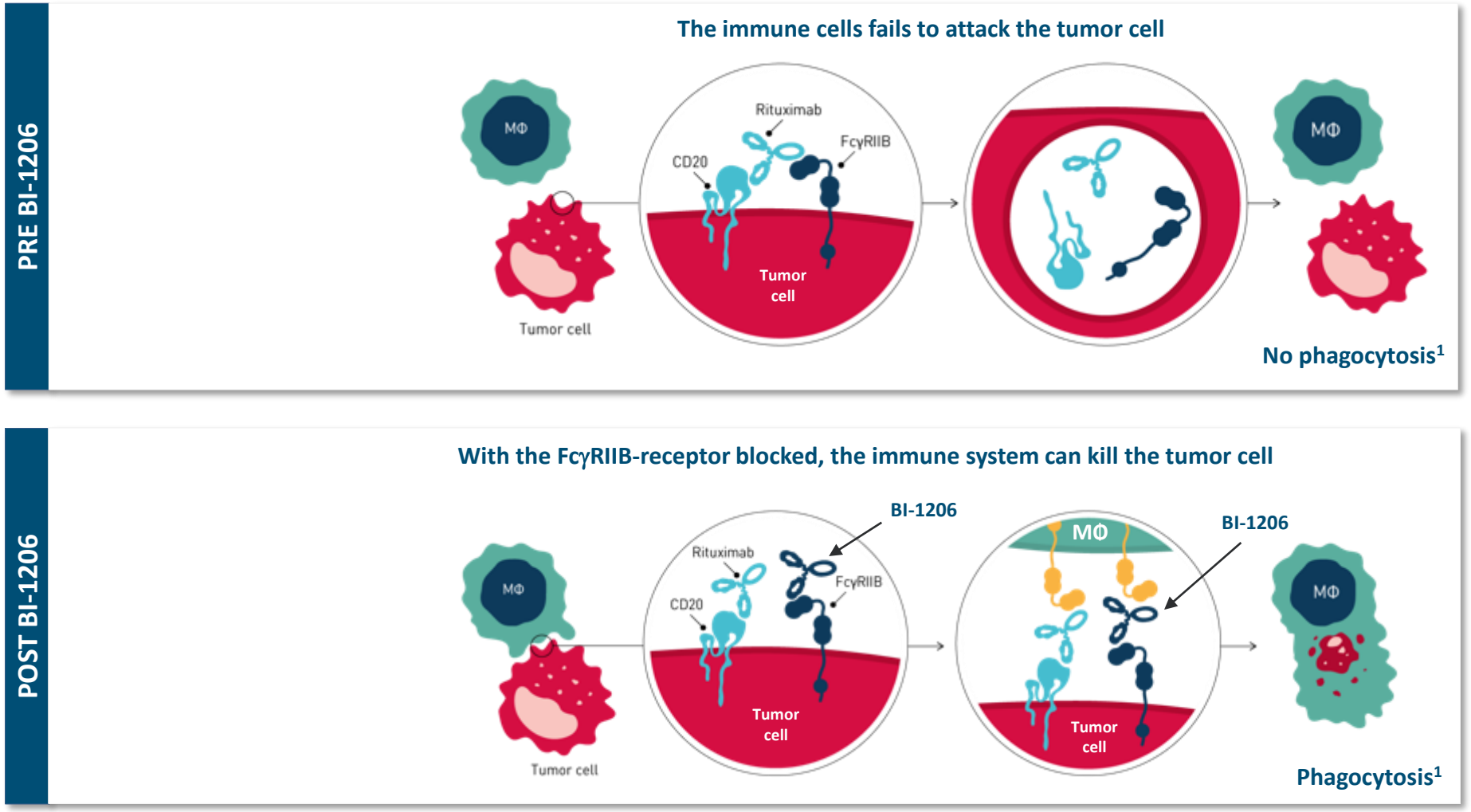
17

FcγRIIB – A SINGLE INHIBITORY ANTIBODY CHECKPOINT TO UNLOCK ANTI-CANCER IMMUNITY



Teige I. et al, Frontiers in Immunol, 2019

BI-1206 IN NON-HODGKIN LYMPHOMA OVERCOMING ANTI-CD20 RESISTANCE



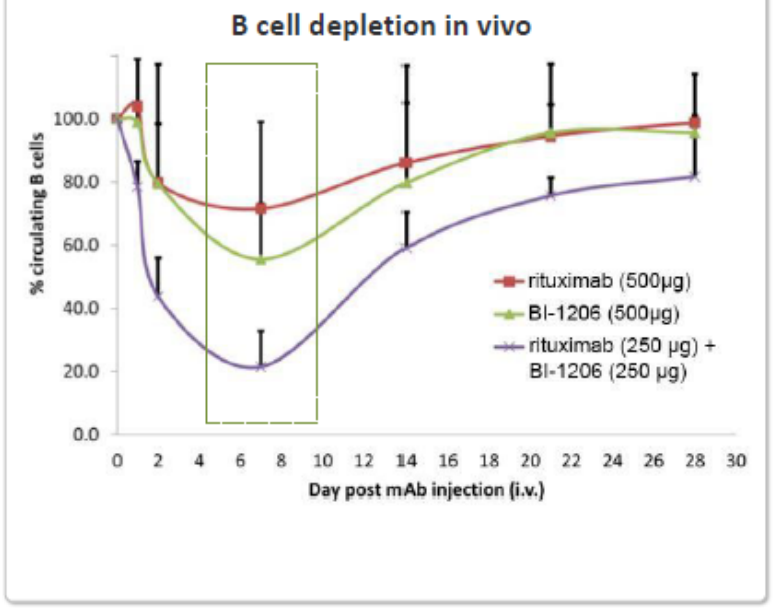
Teige et al, Front Immunol, 2019

BioInvent

BI-1206 BOOSTS EFFICACY AND OVERCOMES RITUXIMAB RESISTANCE

BI-1206 BLOCKS RITUXIMAB INTERNALIZATION AND IMPROVES ITS ANTI-TUMOR ACTIVITY

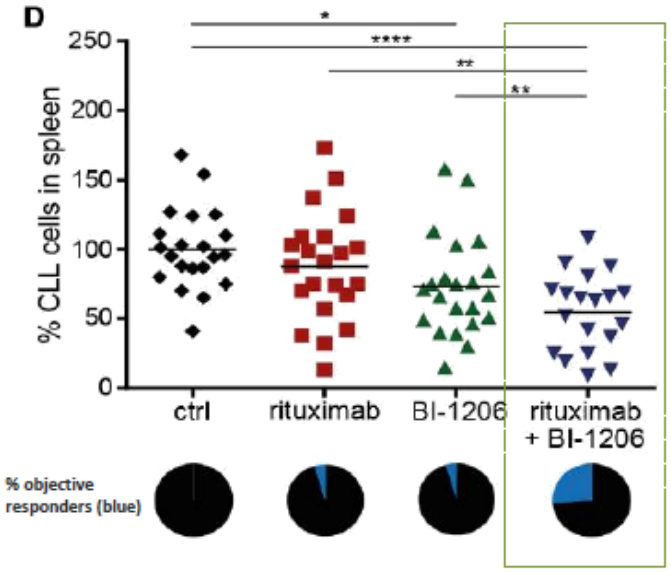
Human CD20 FcγRIIB double transgenic mice



BOOSTING RITUXIMAB'S EFFECT

OVERCOMING RESISTANCE

Humanized model of relapsed / refractory CLL[1]

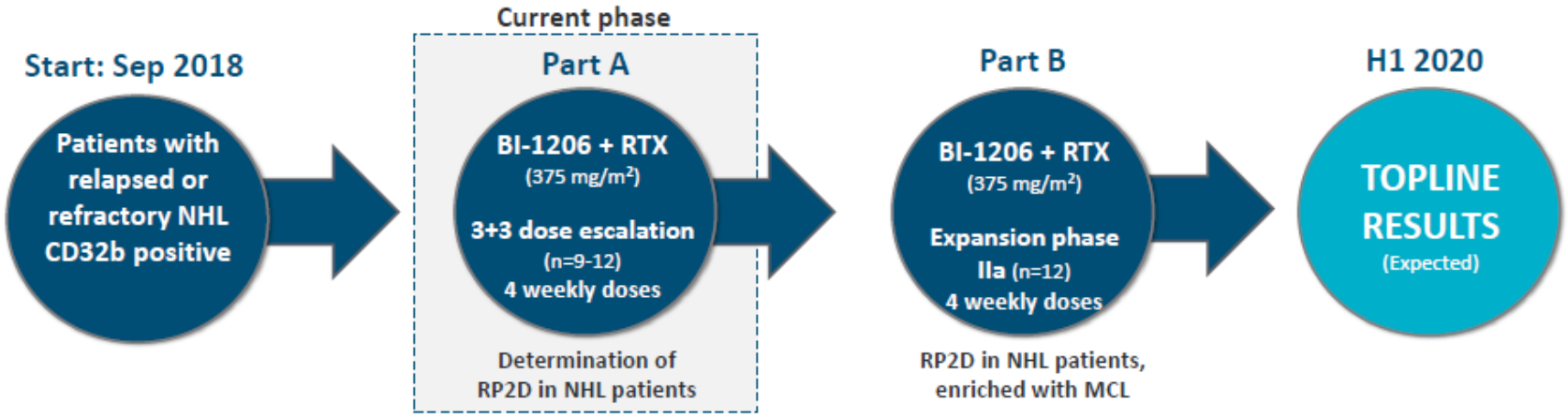


Roghanian, Cancer Cell, 2015

BI-1206-002 PHASE I/IIA STUDY (anti-FcγRIIB + anti-CD20)

STUDY OVERVIEW

- A multicenter, open label, Phase I/IIa study in relapsed or refractory indolent Non-Hodgkin Lymphoma (iNHL) patients enriched with Mantle Cell Lymphoma – approximately 24 patients across sites in US & EU
- High proportion of patients expressing FCγRIIB in enriched population
- High unmet medical need – despite the availability of targeted therapies



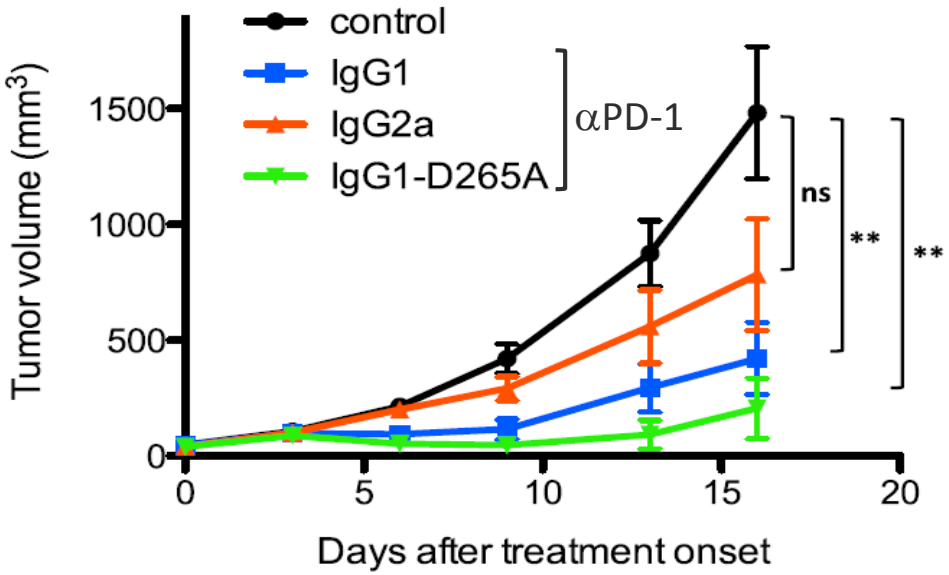
OBJECTIVES

- Safety & tolerability of BI-1206 in combination with rituximab
- PK/PD[1] of the antibody
- Recommended phase 2 dose (RP2D)
- Signs of efficacy of the combination treatment
- Biomarker exploration (B cell depletion, phosphorylation of FCγRIIB)
  - FCγRIIB overexpression is associated with a worse prognosis for the patient

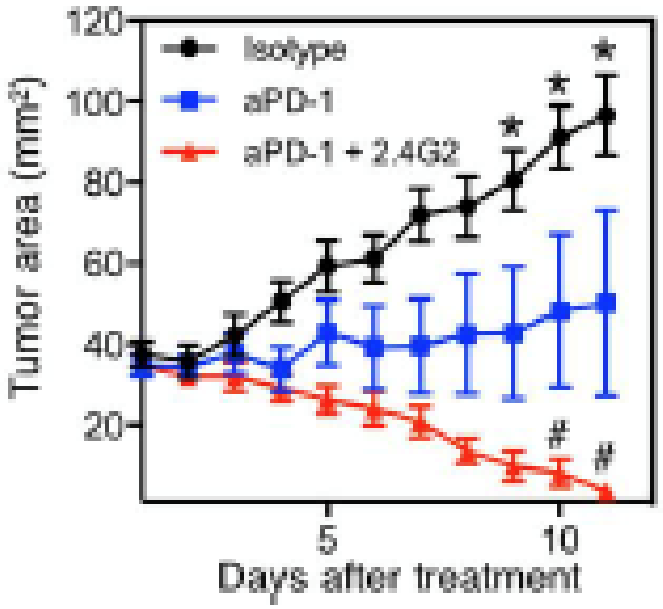
FcγR-INTERACTIONS MODULATE THE ANTI-TUMOR ACTIVITY OF ANTIBODIES TARGETING PD-1

Tumor growth

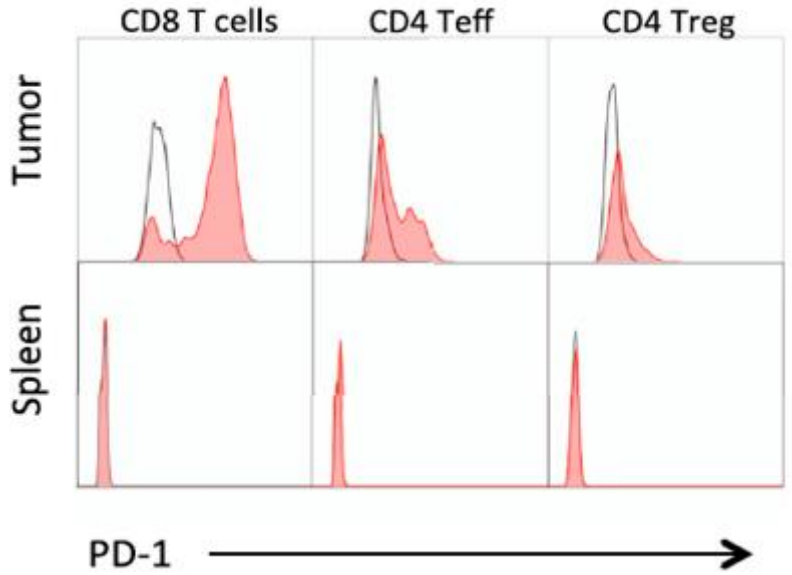
αPD-1 Fc-dependence



FcγR-block



PD-1 expression

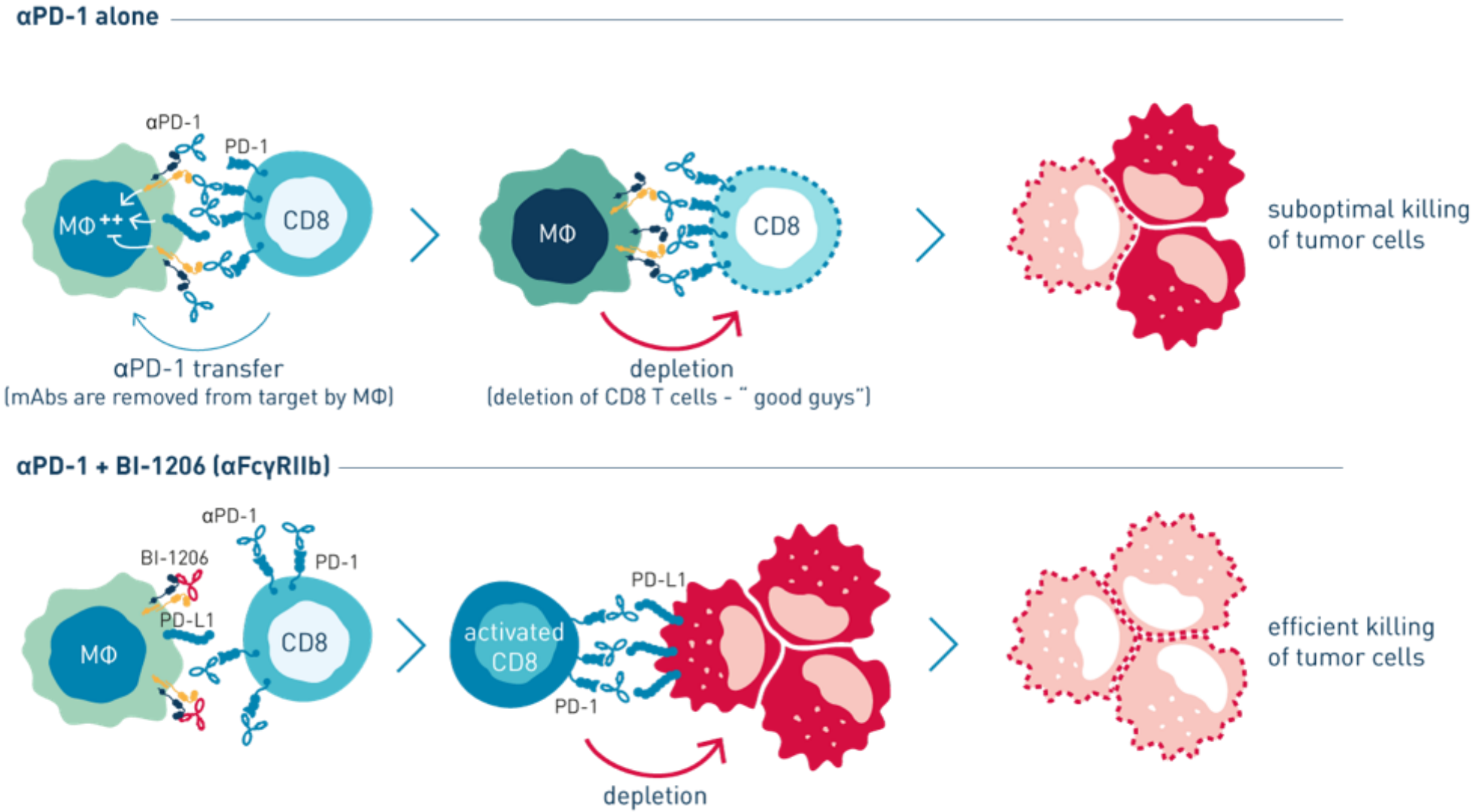


Dahan et al Cancer Cell 2015, Arlauckas Sci Transl Med 2017

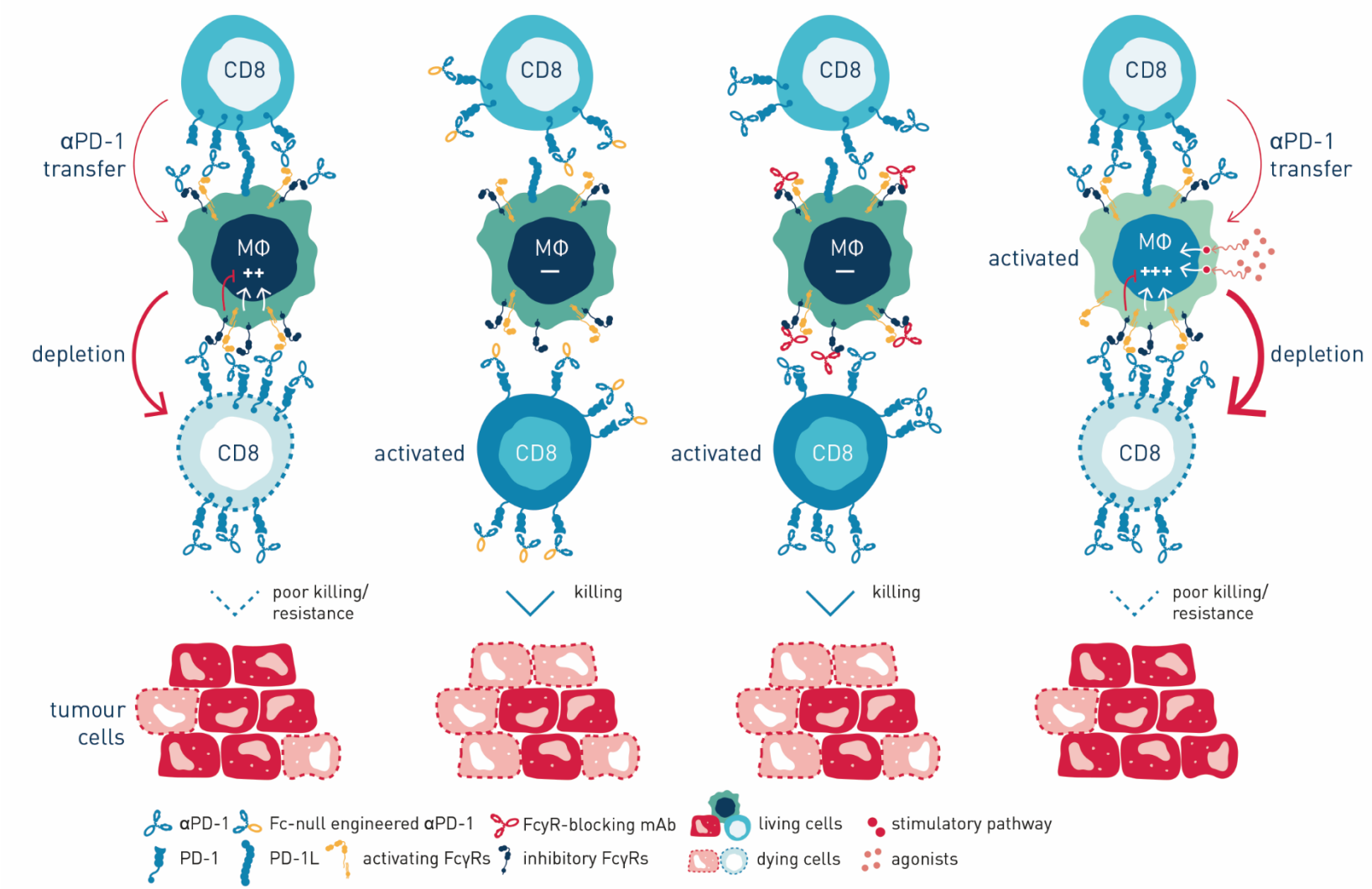
TWO CRUCIAL BENEFITS + ONE ADDITIONAL BENEFIT

- 1) Better / Prolonged anti-PD1 Blocking
    - Positioning BI-1206 in FIRST LINE in MULTIPLE Tumor Types
- 2) Overcome Resistance to anti-PD1
    - Positioning BI-1206 in SECOND LINE in PD1-Refractory settings in MULTIPLE Tumor Types
- 3) Dosing may be different in
    - 1st – 2nd line settings
    - in different tumor type settings: BI-1206 can de dosed as needed

BI-1206 POTENTIAL MECHANISMS OF ACTION - REDUCING aPD-1-MEDIATED CD8+ T CELL DEPLETION AND MAXIMIZING PD-1/PDL1 BLOCKADE



FcγRs may promote resistance to aPD-1 by several mechanisms



Beers and Frendéus, 2020, "IMMUNOTHERAPY IN RESISTANT CANCER: FROM THE LAB BENCH WORK TO ITS CLINICAL PERSPECTIVES" (Elsevier), *in press*

BI-1206 Clinical Update

July 2020

BioInvent

PIPELINE – MULTIPLE VALUE DRIVERS

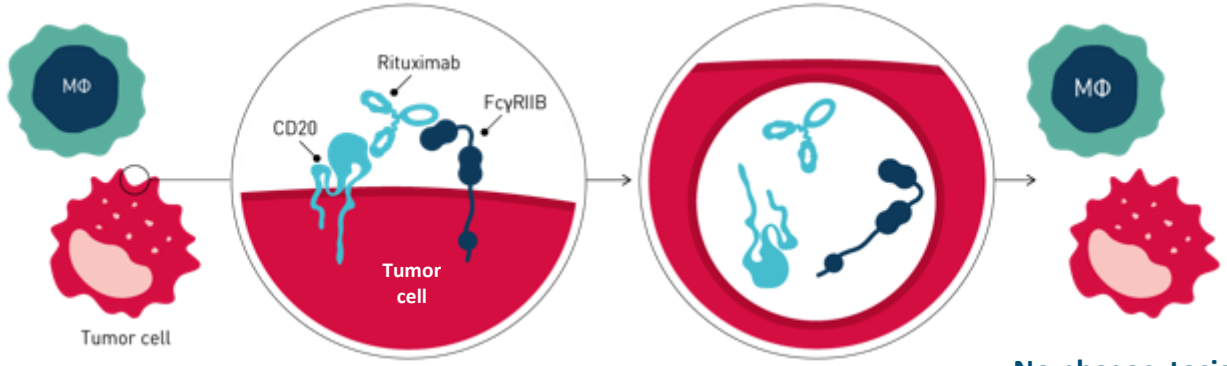
Indication	Program	Discovery	Preclinical	Phase I	Phase II	Partner
Target: FcγRIIB						
iNHL (MCL, MZL, iFL)	BI-1206/Rituximab					
Solid tumors	BI-1206/Pembrolizumab					MSD
Solid tumors	BI-1607			2021		
Target: Tumor associated regulatory T cells (Tregs)						
Solid tumors	BT-001 -α-CTLA4 Mab-VV			2020		transgene
Solid tumors	BI-1808 -α-TNFR2 MAb			2020		
Solid tumors	BI1910 - α-TNFR2 MAb					
Solid tumors	F.I.R.S.T.™ αTreg					
Target: Tumor associated myeloid cells (TAMs)						
Solid tumors	F.I.R.S.T. ™ αTAMs					Pfizer

BI-1206 IN NON-HODGKIN LYMPHOMA TURBOCHARGING ANTI-CD20

PRE BI-1206

- **Rituximab** (Roche's Rituxan® or Mabthera®) is a monoclonal antibody that kills malignant B cells by binding to **CD20** on the cell surface
- The **FcγRIIB**-receptor functions to remove rituximab from CD20, thus hampering its efficacy and protecting cancer cells from the immune system
- FcγRIIB overexpression is associated with a worse prognosis for the patient

The immune cells fail to attack the tumor cell

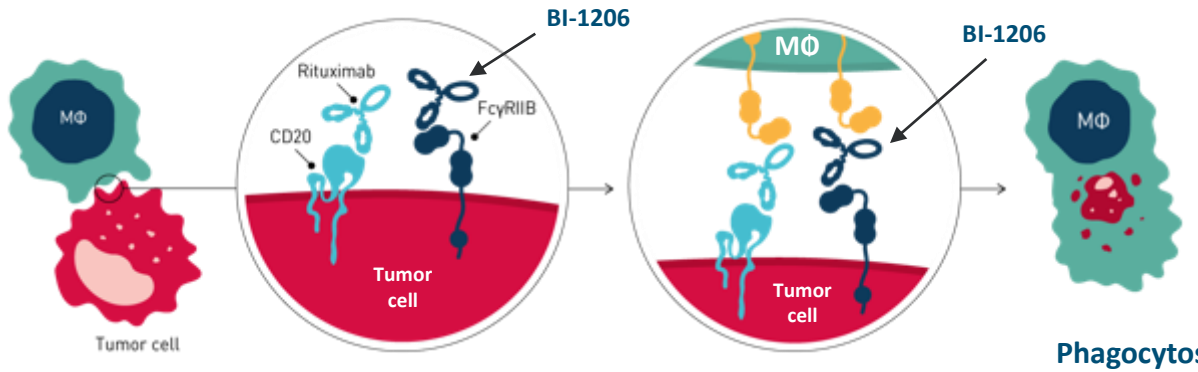


No phagocytosis[1]

POST BI-1206

- **BioInvent's BI-1206** blocks the **FcγRIIB** receptor, suppressing the tumor's protection. Its activity helps restore and enhance rituximab's effect
- With the **FcγRIIB**-receptor blocked, a better anti-tumor activity is engaged allowing the immune system to find and kill the tumor cell

With the FcγRIIB-receptor blocked, the immune system can kill the tumor cell

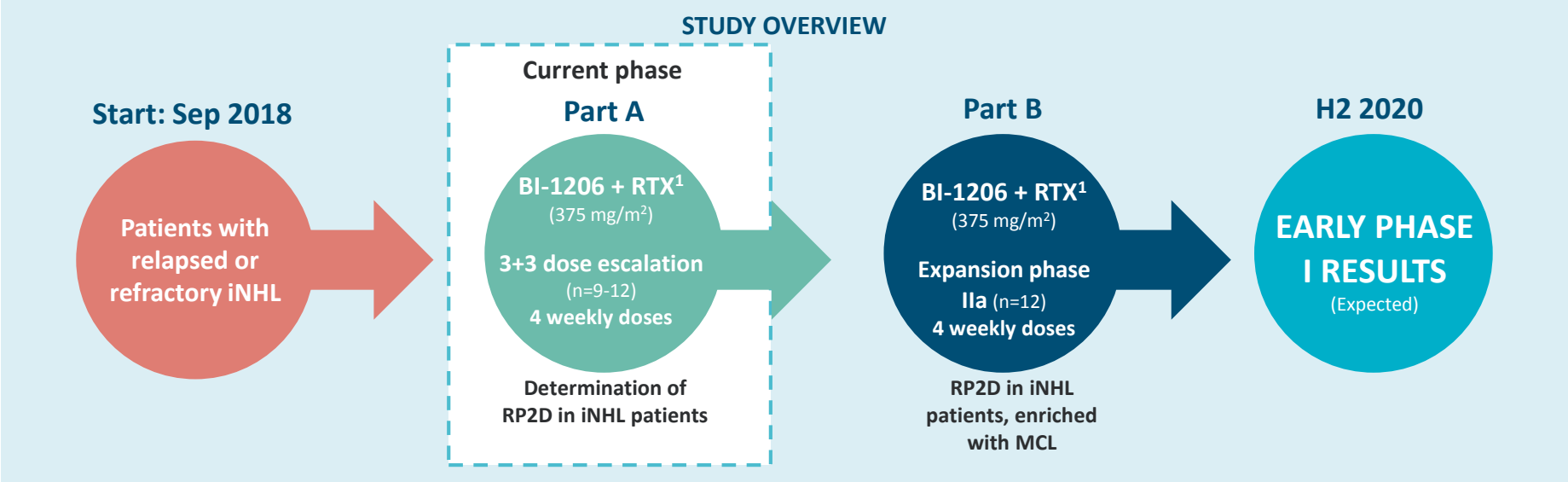


Phagocytosis[1]

Note:(**1**) Phagocytosis refers to the process by which cells (phagocytes) engulf foreign particles. This is a major mechanism for the immune system to remove pathogens and cell debris i.e. killing the tumor cell in this specific case.

BioInvent

BI-1206 IN NON-HODGKIN LYMPHOMA: PHASE I/IIA STUDY



STUDY OBJECTIVES	INCLUSION CRITERIA
▪ Explore safety & tolerability ▪ Illustrate pharmacokinetic and pharmacodynamic profile ▪ Establish recommended phase 2 dose (RP2D) ▪ Observe early signs of efficacy ▪ Biomarker exploration (B cell depletion, depletion of circulating tumoral cells, analysis of biomarkers predictive of response)	▪ Patients must have relapsed disease or disease (R/R) that is refractory to conventional treatment or for which no standard therapy exists. ▪ Lack of CR or PR during rituximab-containing treatment. ▪ Occurrence of progressive disease after completion of a regimen of rituximab-containing therapy.

Note(**1**): RTX = rituximab

BI-1206 IN NON-HODGKIN LYMPHOMA: VALUE PROPOSTION – KEY SEGMENTS & VALUE DRIVERS

BI-1206 value drivers

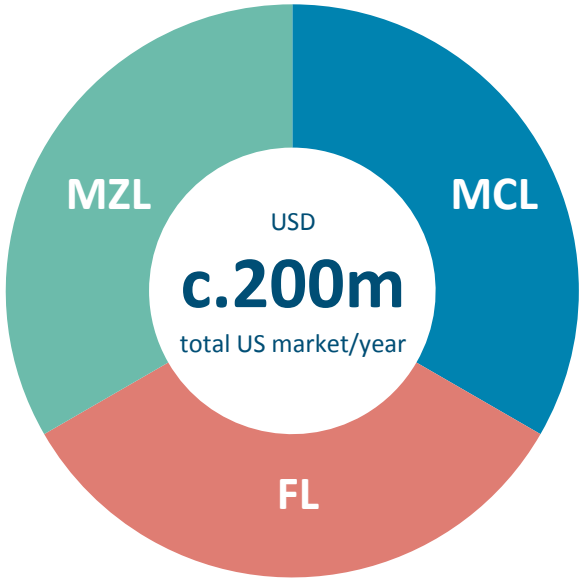
- Compelling scientific rationale in α-CD20 refractory B-cell lymphoma
- Chemo-free regimen
- Favorable safety profile
- Scalability of total addressable market

BI-1206 highlights

- First-in-class in hematology - no direct competitors
- High unmet need for chemotherapy-free, safer options in 2nd and 3rd lines
- Granted FDA Orphan Drug Designation for BI-1206 for MCL in January 2019

Possible label extension to all therapeutic areas where anti-CD20 mAbs are used (incl. autoimmune diseases)

KEY SUB-SEGMENTS OF NON-HODGKIN LYMPHOMA (NHL)



- **Mantle Cell Lymphoma (MCL[1])** may be slow growing (indolent) but can also be fast-growing (aggressive). Usually diagnosed in people in their early 60s. Resistance to ibrutinib results in a very aggressive disease with few treatment options
- **Follicular Lymphoma (FL[1])** is the most common form of slow-growing non-Hodgkin lymphoma
- **Marginal Zone Lymphoma (MZL[1])** is a slow growing type of B cell lymphoma with a median age of diagnosis of 65 years

Note (**1**): Cello Health BioConsulting, 2019

BI-1206 IN NON-HODGKIN LYMPHOMA: PROMISING PRELIMINARY DATA FROM PHASE I/IIA STUDY

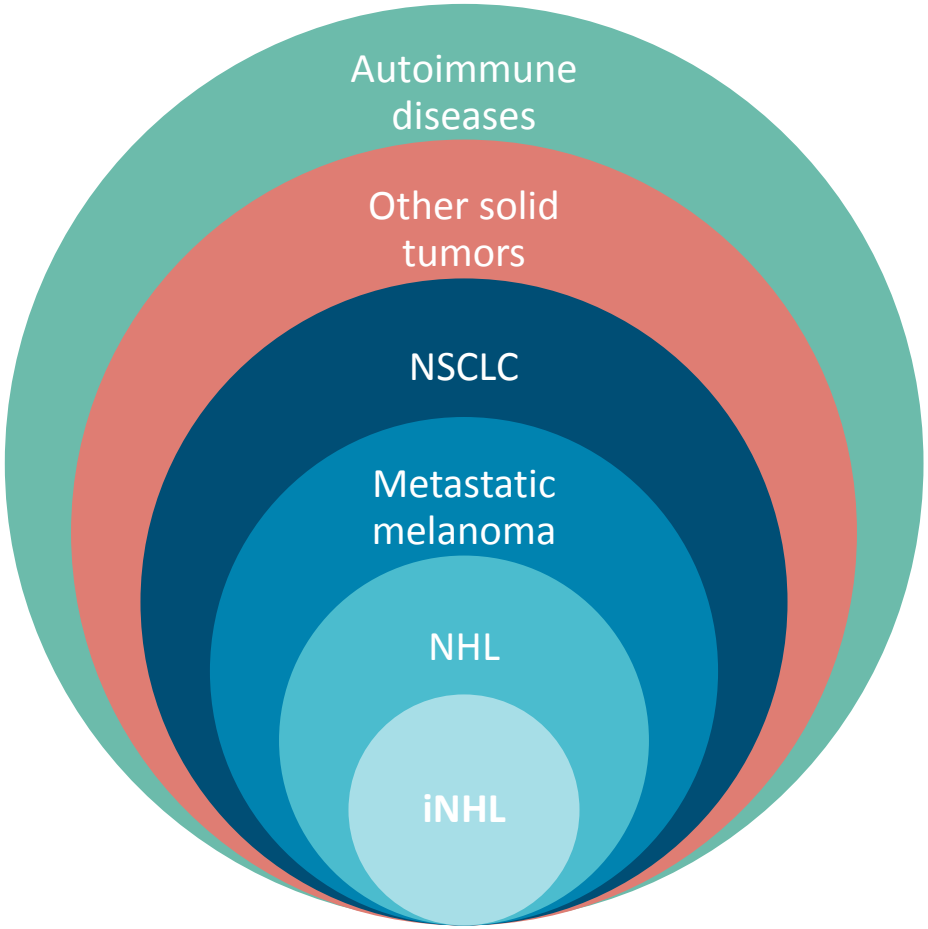
Preliminary data shows early signs of efficacy

- In the 30 mg cohort:
  - 1 patient with FL remained on treatment for the full maintenance period (1 year)
  - 1 patient with blastic Mantle Cell lymphoma, showed complete depletion of circulating MCL cells after BI-1206 infusion
- In the 70 mg cohort:
  - 1 FL patient has achieved a complete response
  - As described by the clinical investigator, the patient “has a very good general condition without toxicity”
- All responses observed thus far have been at dose levels that are below what is believed to be optimal
- The dose escalation continues as planned with additional data expected in H2’2020

Note: Data as of April 14, 2020

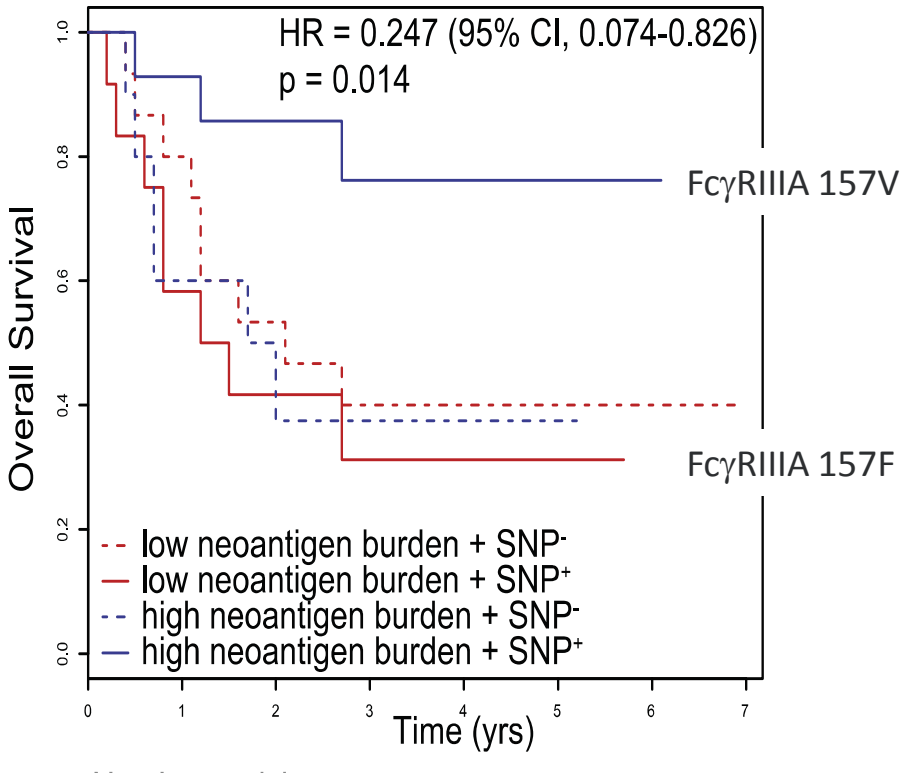
BI-1206 POSSESSES SUBSTANTIAL INDICATION GROWTH POTENTIAL

ESTABLISHING PROOF OF CONCEPT IN CERTAIN INDICATIONS CAN LEAD TO RAPID GROWTH IN TOTAL ADDRESSABLE MARKET



BioInvent

IPILIMUMAB HAS FcgR-DEPENDENT ANTITUMOR IMMUNITY

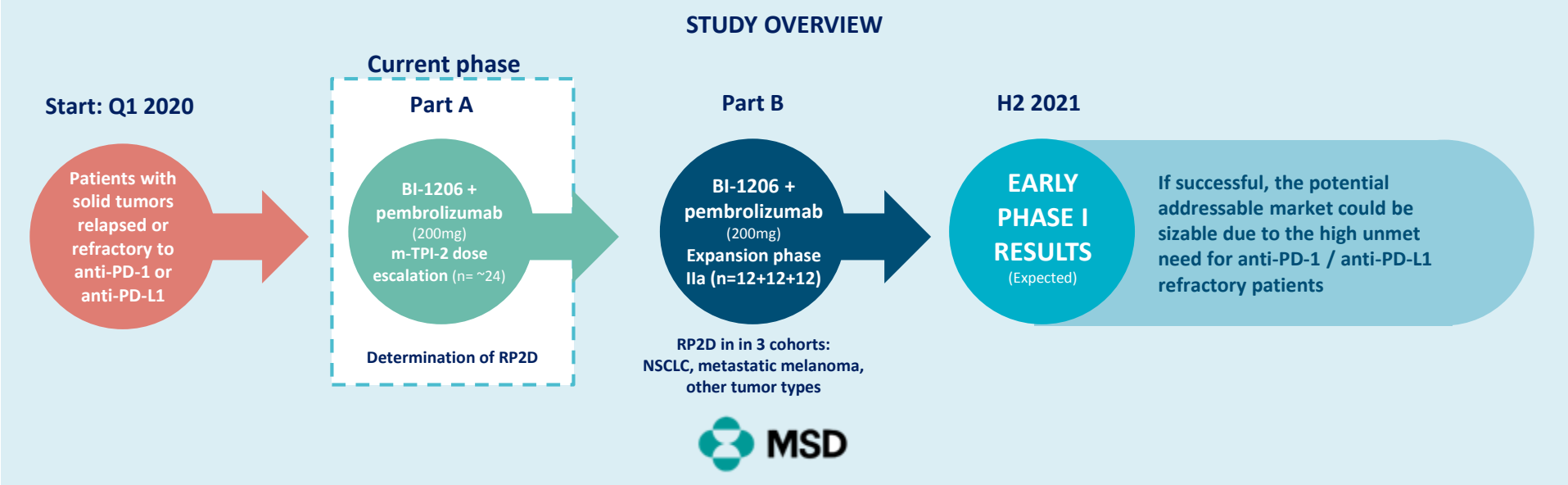


- Two cohorts of melanoma patients treated with ipilimumab
- Activating antibody checkpoint polymorphism (FcγRIIIa V157F) associated with survival
  - Only in inflamed (T cell infiltrated) tumors (solid lines)

Vargas et al Cancer Cell 2018

BioInvent

BI-1206 IN SOLID TUMORS: PHASE I/IIA STUDY WITH MERCK



STUDY OBJECTIVES

- Confirm strong rationale for combination, as FcγRs have been shown to modulate the activity of immune checkpoint inhibitors
- Explore overexpression of FcγRIIb that may determine resistance to anti-PD-1 therapy in metastatic melanoma, NSCLC and others
- Explore safety & tolerability and illustrate pharmacokinetic and pharmacodynamic profile of combination
- Determine recommended phase 2 dose (RP2D)
- Observe early signs of efficacy
- Biomarker exploration (B cell depletion, analysis of biomarkers predictive of response)

BI-1206 IN SOLID TUMORS: FPI IN THE PHASE I/IIA STUDY

- First patient has been treated
- Starting at higher, more relevant doses
- In collaboration with Merck (CTCSA) MSD
- Adaptive design
- Patients who are refractory or have progressed after treatment with anti-PD1/PDL1 targeting agents
- Potential immunological markers to predict clinical responses
  - In collaboration with SkylineDx

Note: Data as of April 14, 2020

UPCOMING NEWS FLOW

Q4 2019	✓ BI-1206 / pembrolizumab research and supply agreement with Merck (MSD) ✓ Pfizer selects second target for development from TAMs program collaboration ✓ BioInvent / Transgene announce promising preclinical data for BT-001 in solid tumors ✓ Promising preclinical data BI-1206 in mantle cell lymphoma presented at ASH 2019
2020	❑ Early results from Phase I open label study with BI-1206 / rituximab combination in indolent Non-Hodgkin Lymphoma (H2 2020) ❑ Potential additional milestones from collaborations ❑ Two new programs enter the clinic: BT-001 and BI-1808
2021	❑ Early results from Phase I open label study with BI-1206 / pembrolizumab combination in solid tumors (H2-2021) ❑ Potential additional milestones from collaborations ❑ One new program enters the clinic: BI-1607

Q&A

Enhancing Checkpoint Inhibitors for the Treatment of Solid Cancers

KOL Call July 2020

BioInvent