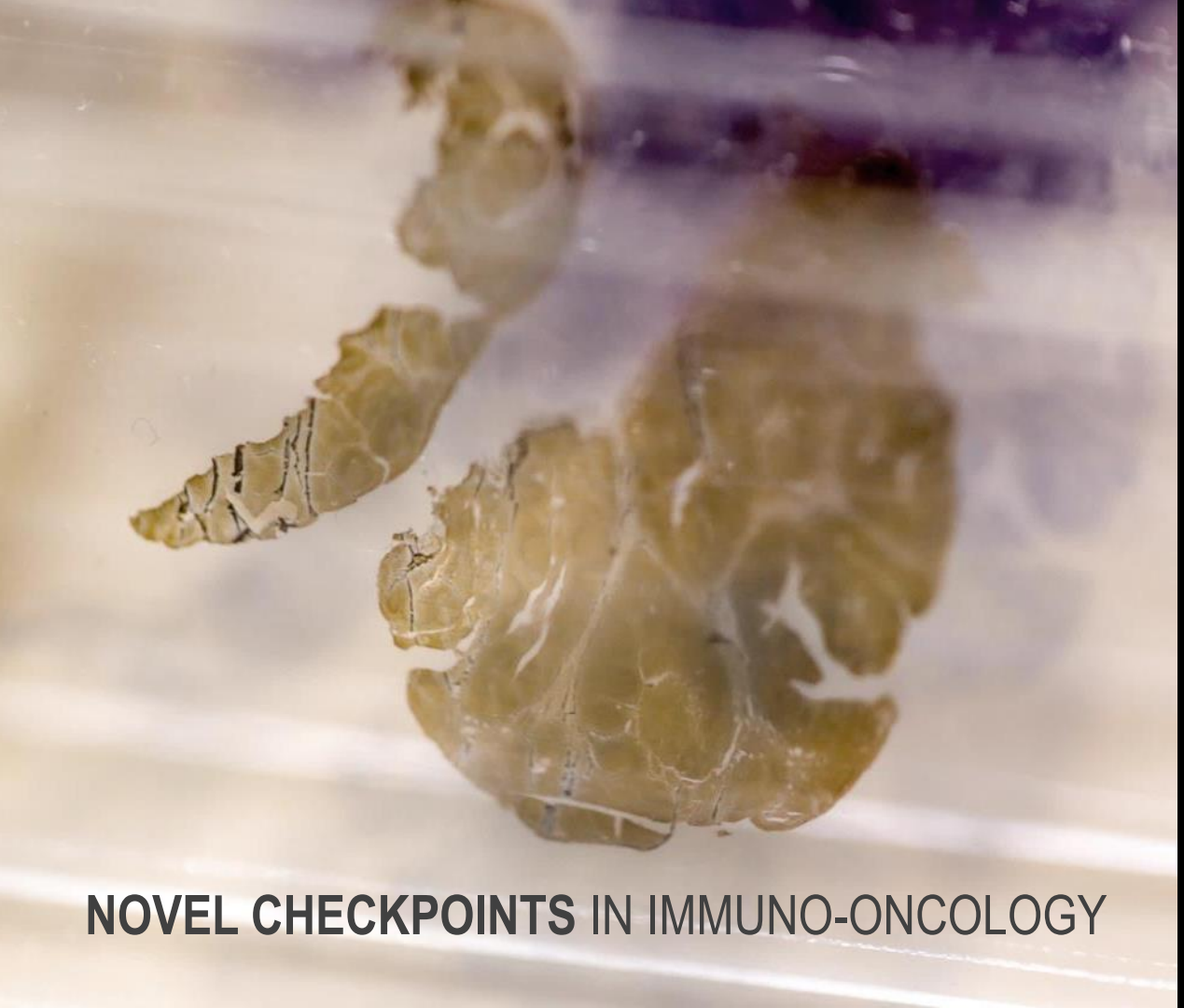

NOVEL CHECKPOINTS IN IMMUNO-ONCOLOGY

innate pharma

ANTI-C5AR ACQUISITION

JUNE 2017

IPH LISTED EURONEXT

afaq ISO 9001 Qualité AFNOR CERTIFICATION

FORWARD LOOKING STATEMENT

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This document contains forward-looking statements. Although the Company believes its expectations are based on reasonable assumptions, these forward-looking statements are subject to various risks and uncertainties, which could cause the Company's actual results or financial condition to differ materially from those anticipated. Please refer to the risk factors outlined from time to time in the Company's regulatory filings or publications.

This document contains data pertaining to the Company's potential markets and the industry and environment in which it operates. Some of this data comes from external sources that are recognized in the field or from Company's estimates based on such sources.

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Please refer to the Document de Référence filed with the Autorité des Marchés Financiers ("AMF") on March 31st, 2017, available on the AMF's website (www.amf-france.org) and on the Company's website (www.innate-pharma.com). Such documents may not be necessarily up to date.

This document and the information contained herein do not constitute an offer to sell or a solicitation of an offer to buy or subscribe to shares of the Company in any country.

INNATE PHARMA ACQUIRES FIRST-IN-CLASS ANTI-C5AR ANTIBODY

ANTI-C5AR, A NOVEL THERAPEUTIC APPROACH IN IMMUNO-ONCOLOGY (IO)

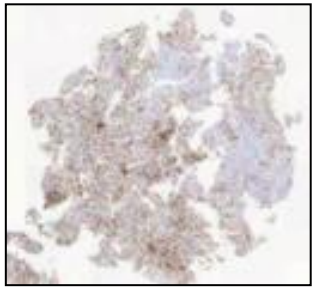
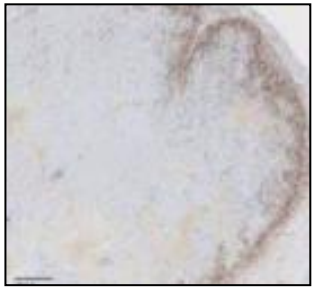
- Anti-C5aR (IPH5401) is a first-in-class therapeutic mAb targeting myeloid-derived suppressor cells (MDSC) and neutrophils in the tumor microenvironment
  - > These cells suppress anti-tumor immune-responses
  - > They are associated with resistance to checkpoint blockers

- New proprietary clinical-stage asset, with strategic fit to
  - > Innate Pharma's pipeline of mAbs designed to stimulate anti-tumor activities of NK and other immune cells
  - > IPH's pipeline of mAbs targeting the tumor microenvironment, boosted with clinical asset

- Trial initiation with IPH5401 in oncology expected in 2018
  - > Favorable safety profile in single- and multi-dose Phase I trials in arthritis patients (Novo Nordisk A/S)

STRENGTHENING OF INNATE PHARMA'S PIPELINE OF FIRST-IN-CLASS MABS IN IO

Inflamed — **Non inflamed**

Checkpoint-inhibited T and NK cells	T and NK cells suppressed or excluded	No T or NK cells in tumor
		
NK cell checkpoint lirilumab	**MDSC** IPH5401	**Bispecific antibodies**
NK and T cell checkpoint monalizumab	**ADCC mAb** IPH4301	
ADCC mAb IPH4102	**Adenosine pathway** IPH52 and IPH53	

Adapted from Hedge et al., Clin Cancer Res 2016

SUMMARY OF THE TRANSACTION

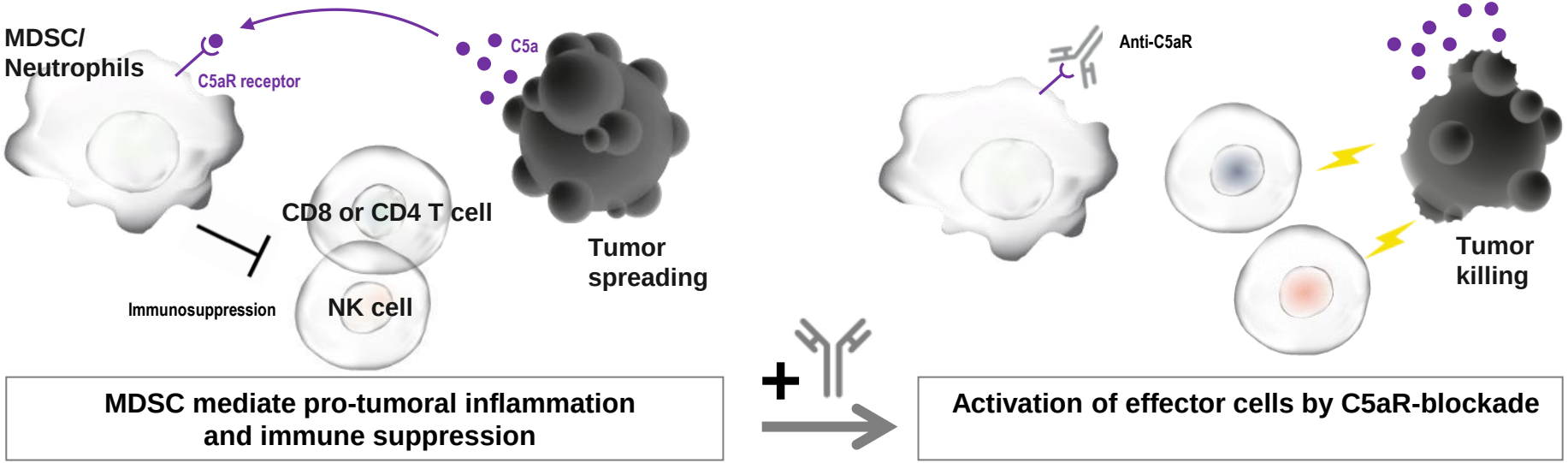
- Agreement granting IPH full worldwide exclusive rights to develop and commercialize a first-in-class clinical-stage anti-C5aR antibody
- Upfront payment of €40M
  - > €37.2M in new Innate shares and €2.8M in cash
  - > The number of shares to be issued will be based on the 10 days VWAP* preceding closing, subject of a minimum of 2,700,000 shares and to a maximum of 3,500,000 shares
  - > After the closing of the transaction, Novo Nordisk A/S's stake in Innate Pharma will increase from 10.3% currently to between 14.6% to 15.8%, depending on this average trading price
  - > Closing expected by July 12, 2017
- Development, regulatory and sales milestones payments up to €370M
- Double-digit royalties on net sales

* VWAP: volume weighted average price

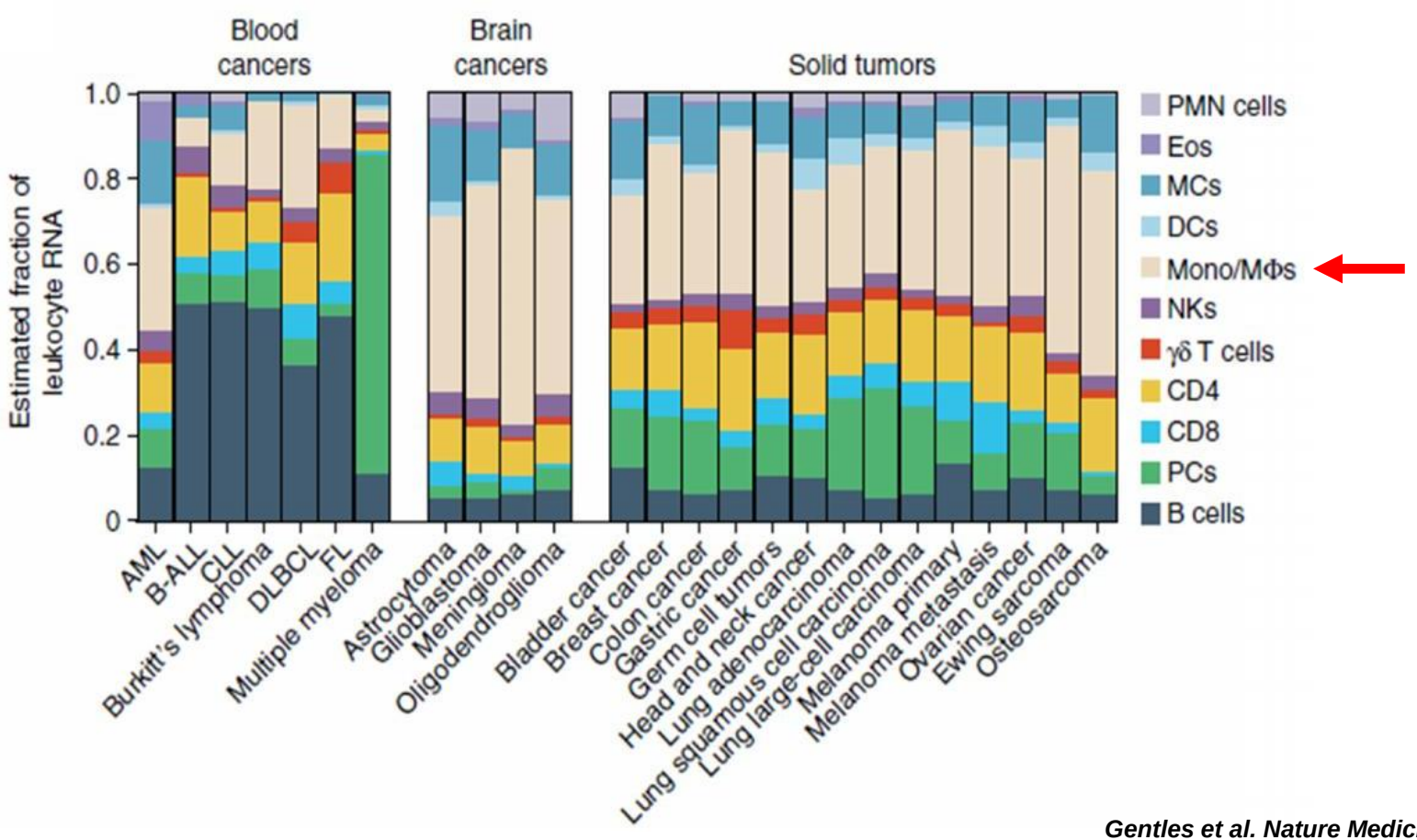
RATIONALE FOR TARGETING MDSC AND C5AR IN ONCOLOGY

IPH5401, A FIRST-IN-CLASS MAB
MECHANISM OF ACTION

- IPH5401 is a fully human anti-C5aR blocking antibody
  - > High affinity and high potency; no Fc-mediated depletion or agonist activity
- Has potential to enhance anti-tumor immunity across a range of tumor types
  - > MDSC-targeting has proven effectiveness in a range of experimental models

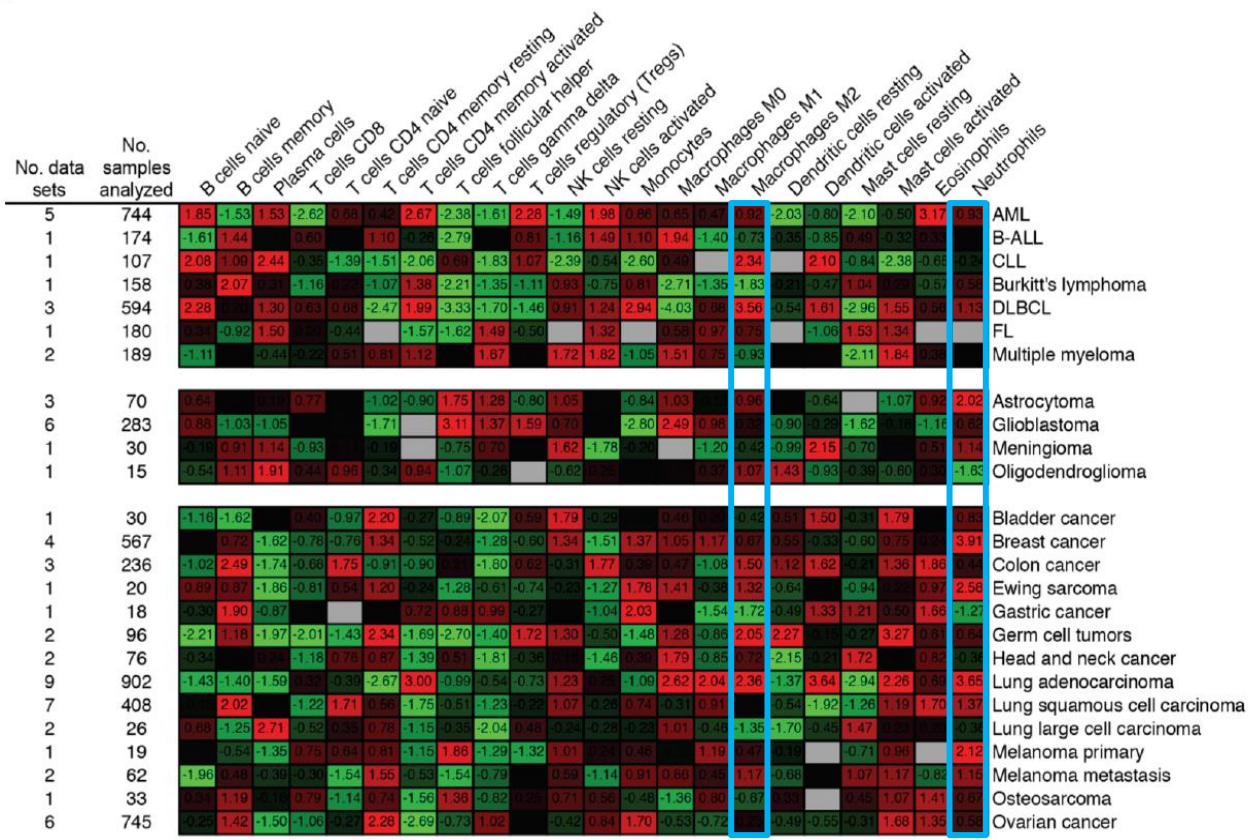


MDSC ARE ABUNDANT IN NUMEROUS CANCERS



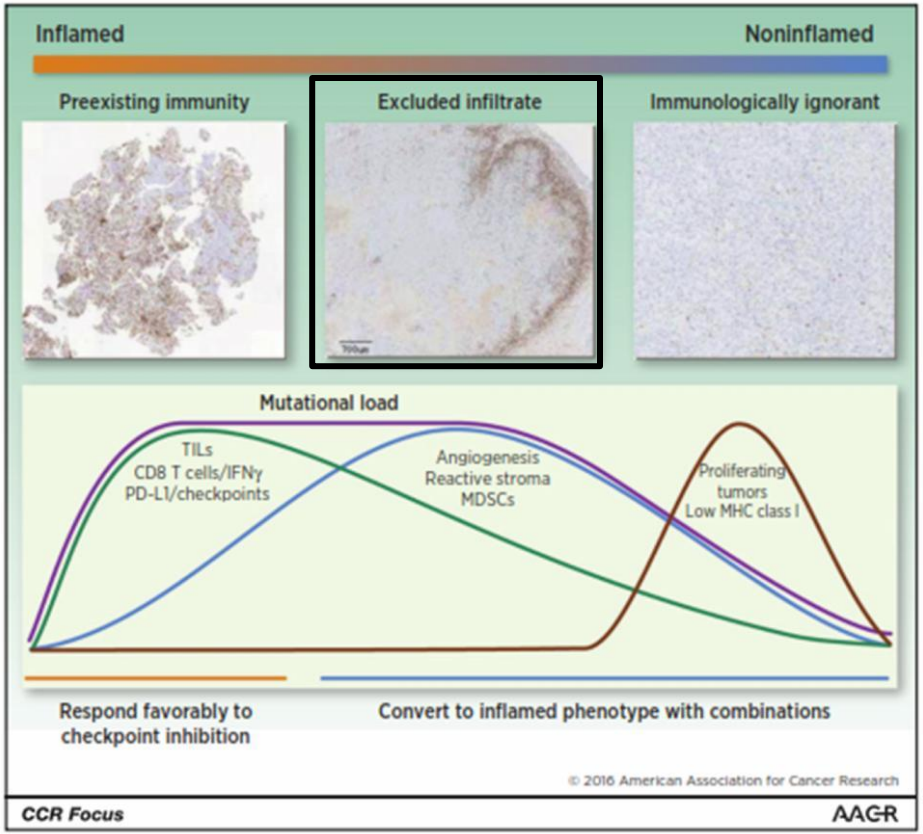
Gentles et al. Nature Medicine 2015

MDSC AND NEUTROPHILS ARE OFTEN ASSOCIATED WITH A POOR PROGNOSIS



Gentles et al. Nature Medicine 2015

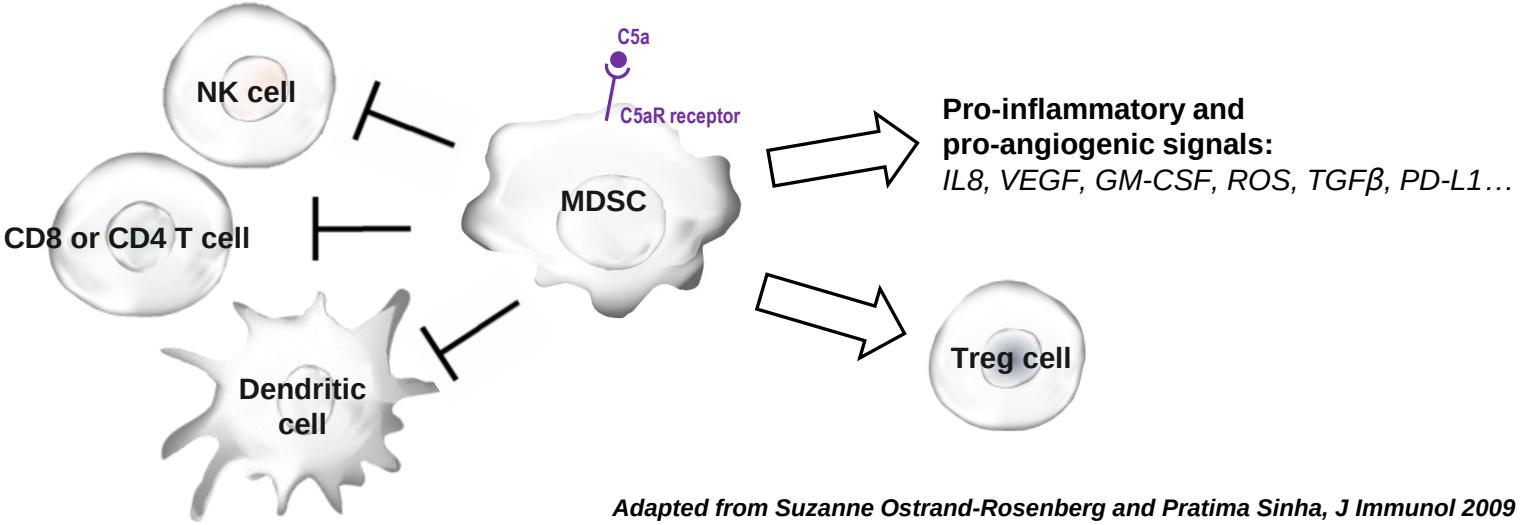
MDSC MAY RENDER PATIENTS REFRACTORY TO CHECKPOINT BLOCKERS



Hedge et al., Clin Cancer Res 2016

MDSC PROMOTE TUMOR GROWTH AND SUPPRESS ANTI-TUMOR IMMUNE RESPONSES

- MDSC and neutrophils suppress effector T and NK cells by the production of cytokines and induction of Tregs
- MDSC secrete pro-inflammatory and pro-angiogenic factors that promote tumor growth and metastasis
- C5a is a potent chemo-attractant and activator of MDSC and neutrophils



Adapted from Suzanne Ostrand-Rosenberg and Pratima Sinha, J Immunol 2009

RECENT DATA PROVIDE RATIONALE FOR C5AR-BLOCKADE IN ONCOLOGY

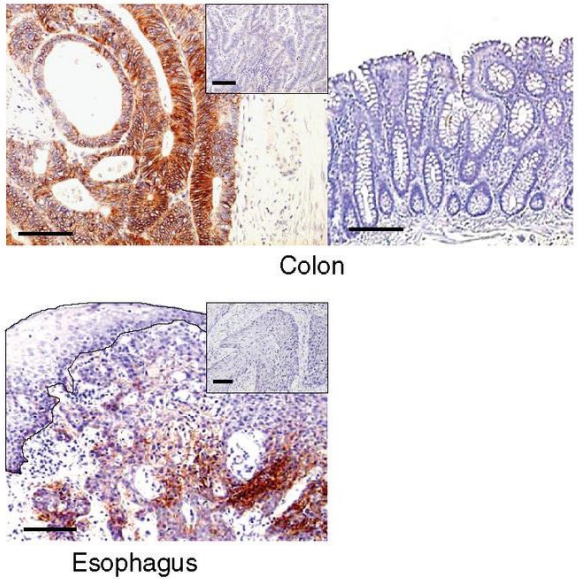
- C5a and its receptor are upregulated in several types of cancer[1,2]
- C5aR mediates accumulation of MDSC in mouse tumors[3]
- M2 macrophages express high levels of C5aR and respond strongly to C5a-induced chemotaxis[4]
- C5aR signaling stimulate secretion of TGFβ by MDSC which suppress T and NK cells[5,6]
- C5aR induces PD-L1 expression on monocytes[2,7]
- C5aR depletion or pharmacological inhibitors slow tumor growth and synergize with anti-PD1[3,8,9,10]

1 Hidetoshi Nitta et al., Clin Cancer Res, 2013 ; 2 Corrales et al. J. Immunol. 2012; 3 Markiewski et al. Nature Immunology 2008; 4 Internal data ; 5 Qing et al. Eur J. Immunol. 2012; 6 Janelle et al. Cancer Immunol Research 2014; 7 Ling-Ling An et al., Sci Rep., 2016; 8 Vadrevu et al. Cancer research. 2014; 9 Ajona D et al; Cancer Discovery 2017; 10 Internal data (not shown)

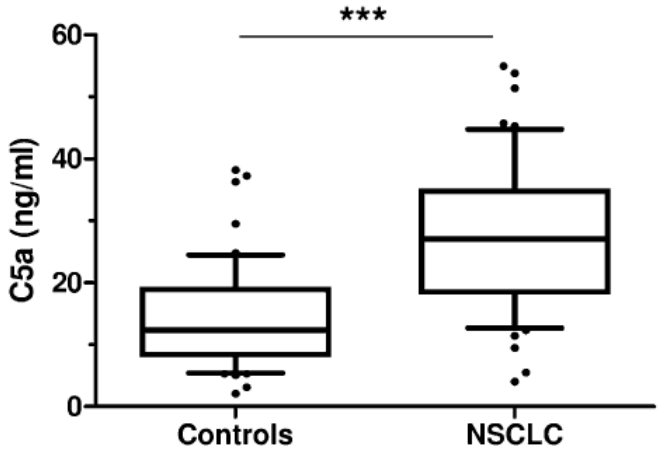
C5A AND ITS RECEPTOR ARE UPREGULATED IN SEVERAL TYPES OF CANCER

- C5aR appears to be highly express in several cancer types with abundant MDSC

1 IHC for C5aR in colorectal and esophagus



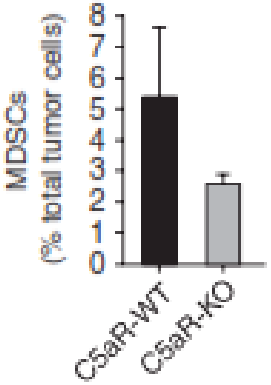
2 Elevated levels of C5a in circulation of NSCLC patients

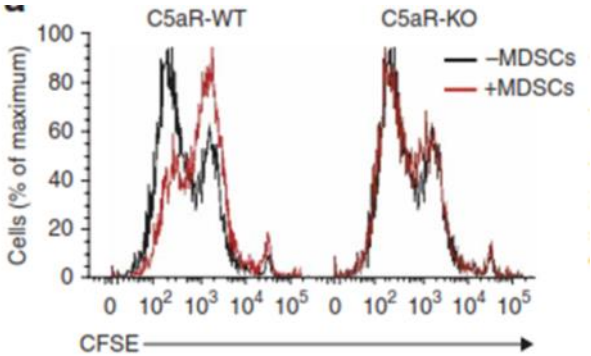


1 Hidetoshi Nitta et al., Clin Cancer Res, 2013; 2 Corrales et al J. Immunol 2012

C5AR KO MICE: FEWER AND LESS FUNCTIONAL MDSCs, AND REDUCED TUMOR GROWTH

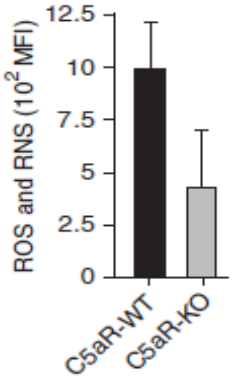
C5aR KO mice have reduced numbers of tumor infiltrating MDSCs

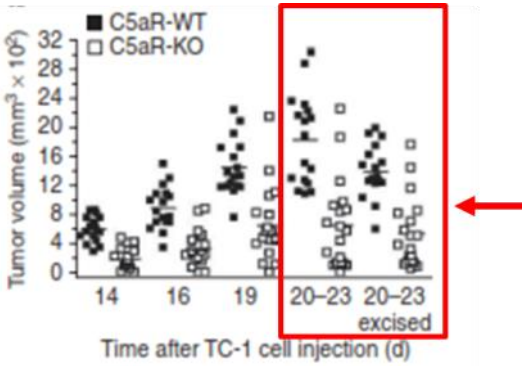




MDSCs from C5aR KO mice are unable to suppress T cell proliferation

MDSCs from C5aR KO mice produce reduced levels of ROS and RNS

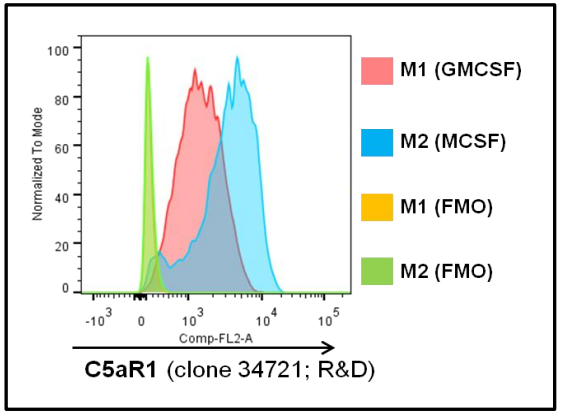


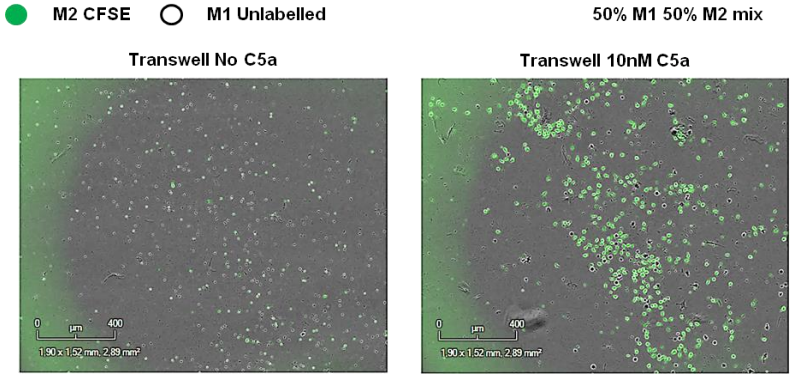


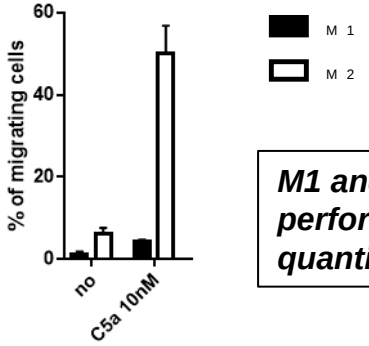
C5aR KO mice have reduced tumor growth

Markiewski et al., Nat Immunol., 2008

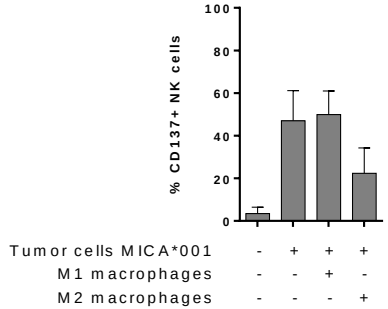
HUMAN M2 MACROPHAGES EXPRESS HIGH LEVELS OF C5AR AND RESPOND STRONGLY TO C5A-INDUCED CHEMOTAXIS







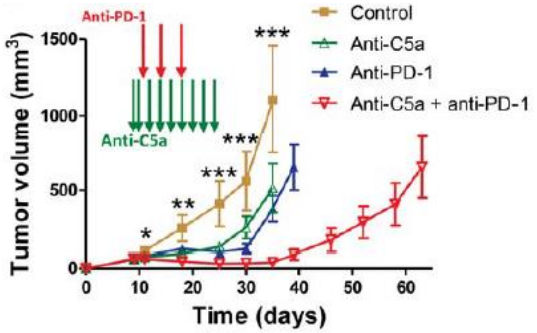
M1 and M2 migrations were performed individually and quantified with Cell Titer Glo

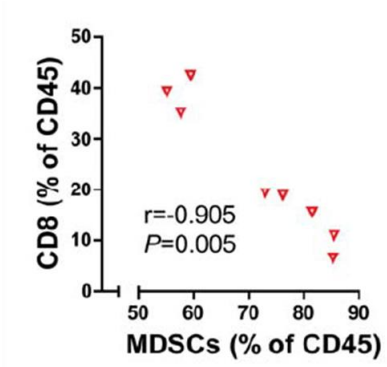


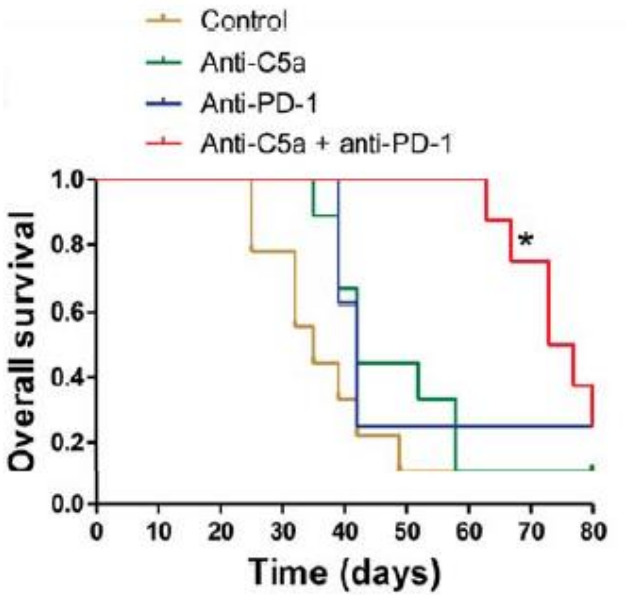
Innate Pharma data

C5A BLOCKADE CONTROLS THE GROWTH OF ESTABLISHED LUNG TUMORS, AND SYNERGIZES WITH ANTI PD-1

393P model: Kras driven highly aggressive lung adenocarcinoma







Similar efficacy observed in a model of Lewis lung cancer

Ajona D et al; Cancer Discovery 2017

CONCLUSIONS

- Tumor microenvironment and infiltration by immunosuppressive cells (e.g. MDCS) are increasingly viewed as a key tumor escape mechanism

- Anti-C5aR is a novel approach for reducing infiltration and activation of MDSC
  - > Inhibits both MDSC and suppressor neutrophils

- IPH5401 is a first-in-class, clinical-stage anti-C5aR blocking antibody
  - > Favorable safety profile in single- and multi-dose Phase I trials in arthritis patients
  - > In-house validation in cancer
  - > Potential for single-agent and combination with PD-1 checkpoint blockade

- Trials initiation with IPH5401 in oncology expected in 2018

BROAD, INNOVATIVE AND DIVERSIFIED PORTFOLIO

CLINICAL PROGRAM	TARGET	INDICATION	STAGE	OWNERSHIP
Lirilumab	KIR2DL-1,-2,-3	Squamous cell carcinoma of the head and neck (SCCHN) Solid and hematological tumors - Multiple combinations	Randomized Phase I/II Phase I & II trials	Licensed to Bristol-Myers Squibb
Monalizumab	NKG2A	Solid and hematological tumors - Single agent and multiple combinations	Phase I/II trials	Co-development with AstraZeneca
IPH4102	KIR3DL2	Cutaneous T-cell lymphomas (CTCL)	Phase I including cohort expansion	Innate Pharma
IPH5401	C5aR	Cancer	Trials to start in 2018	Innate Pharma
PRECLINICAL PROGRAM				
IPH4301	MICA/B	Cancer	IND-enabling studies	Innate Pharma
IPH52	CD39	Cancer	Research	Innate Pharma
IPH53	CD73	Cancer	Research	Innate Pharma

INVESTOR RELATIONS

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Chief Financial Officer

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Tel: +33 (0)4 30 30 30 87

APPENDIX

BIBLIOGRAPHY: TARGETING MDSC

- The role of myeloid cells in cancer therapies (Engblom et al. Nat. Rev. Cancer 2016)
- Eradication of metastatic mouse cancers resistant to immune checkpoint blockade by suppression of myeloid-derived cells (Kim et al. PNAS 2014)
- Prostate: Effective combinatorial immunotherapy for castration-resistant prostate cancer (Lu et al. Nature 2017)
- Breast and melanoma: Overcoming resistance to checkpoint blockade therapy by targeting PI3Kγ in myeloid cells. (De Henau O et al., Nature 2016)
- Pancreatic: CSF1/CSF1R Blockade Reprograms Tumor-Infiltrating Macrophages and Improves Response to T Cell Checkpoint Immunotherapy in Pancreatic Cancer Models. Yu Zhu et al., Cancer res (2014)
- Colorectal: Tumoral Immune Cell Exploitation in Colorectal Cancer Metastases Can Be Targeted Effectively by Anti-CCR5 Therapy in Cancer Patients. (Halama et al. Cancer Cell 2016)

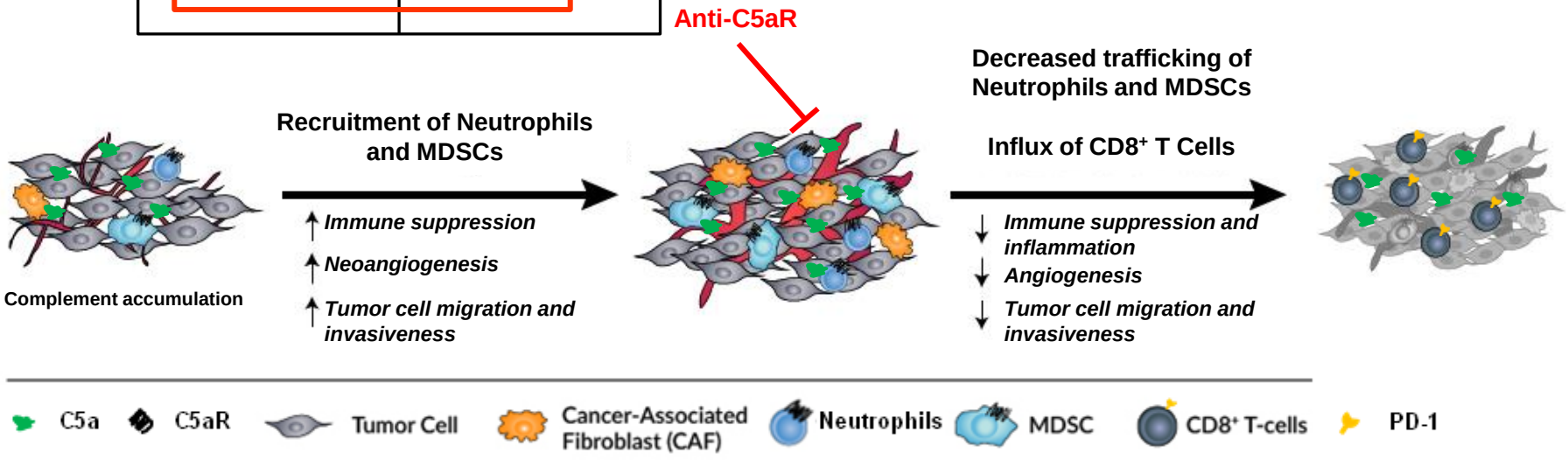
BIBLIOGRAPHY: C5AR IN CANCER

- Complement C5a Receptor (C5aR) promotes myeloid-derived suppressor cells (MDSC) and reduces T cells (Markiewski et al Nature Immunology 2008)
- C5aR ko mice exhibit reduced tumor grwoth and in mouse TC1 solid tumor model, single-agent C5aR antagonist peptide inhibited tumor growth, w efficacy similar to Paclitaxel (Markiewski et al Nature Immunology 2008)
- C5aR blockade by antagonist peptide inhibited accumulation of MDSC and reduced metastasis in mice. C5a upregulated in patients with NSCLC. (Corrales et al. J. Immunol 2012)
- In three mouse models of lung cancer, C5aR antagonist peptide inhibited tumor growth and synergized with anti- PD-1 (Ajona et al.Cancer Discovery 2017)
- C5aR can be expressed on CD8 T cells and blockade enhances anti-tumor activity (Wang et al, Cancer Discovery 2016 – commentary by Hwu et al in same issue)
- A role for the complement system in NK cell-mediated antitumor T-cell responses (Qing et al. Eur. J. Immunol 2012.; Janelle et al. Cancer Immunol Research 2014)

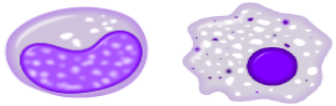
C5A/C5AR PATHWAY IN MDSCs AND CANCER

Neutrophils	MDSC
CD11b$^+$	CD11b$^+$
CD14$^-$	CD14$^+$
CD15$^+$	CD15$^-$
Ly6G$^+$	Ly6G$^-$
Ly6C$^-$	Ly6C$^+$
CD33$^-$	CD33$^+$
C5aR$^+$	C5aR$^+$

C5aR is one of the few markers expressed on both Neutrophils and MDSCs



C5aR FUNCTIONS

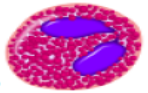


Monocytes and macrophages
- chemotaxis
- phagocytosis ↑
- Cytokine/chemokine release ↑
  - TNFα, IL-1 and IL-6 ↑
  - Synergy with TLR ligands and IC
- CR1, -3 and activating FcγRs ↑

Neutrophils
- adhesion CD11b/CD18↑ – CD62L↓
- transendothelial migration MMP9
- chemotaxis
- phagocytosis ↑
- release of granules ↑
- apoptosis ↓
- NETosis

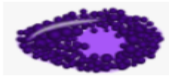
Dendritic cells
- chemotaxis
- maturation ↑

C5a

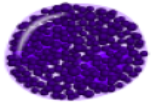


Eosinophils
- chemotaxis
- release of granules ↑

Mast cells

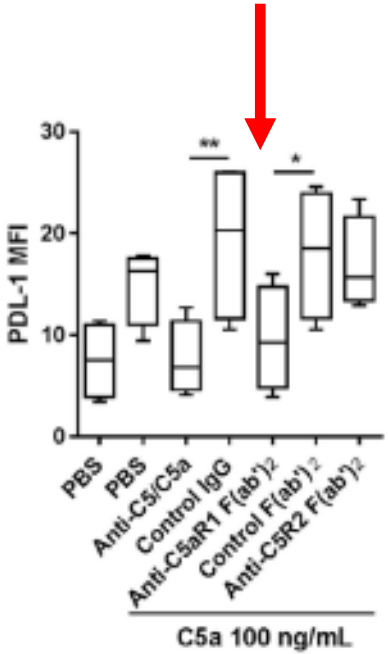


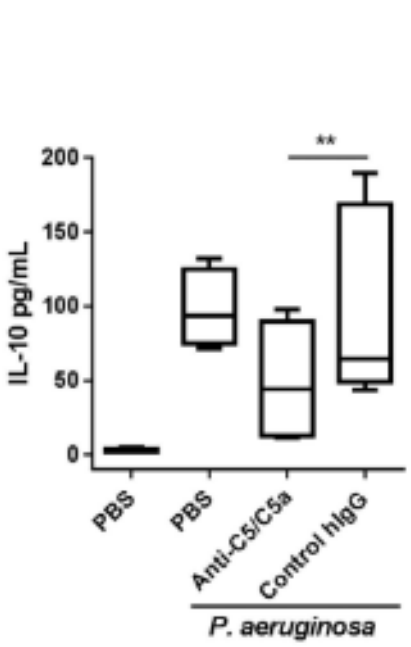
- chemotaxis
- histamine release ↑
- IL-1, -6, -8 and -17A

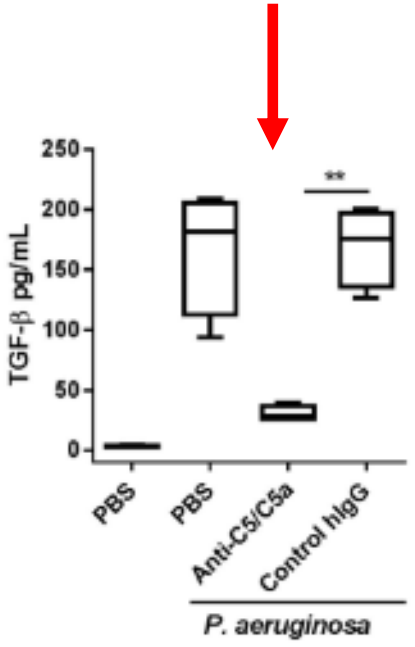


Basophils
- histamine release ↑

C5A INDUCES PD-L1 EXPRESSION AND CYTOKINE RELEASE IN MONOCYTES







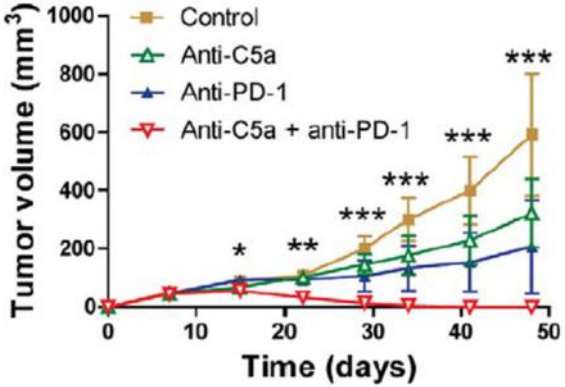
C5a induces PD-L1 expression on human monocytes via C5aR1 but not C5aR2

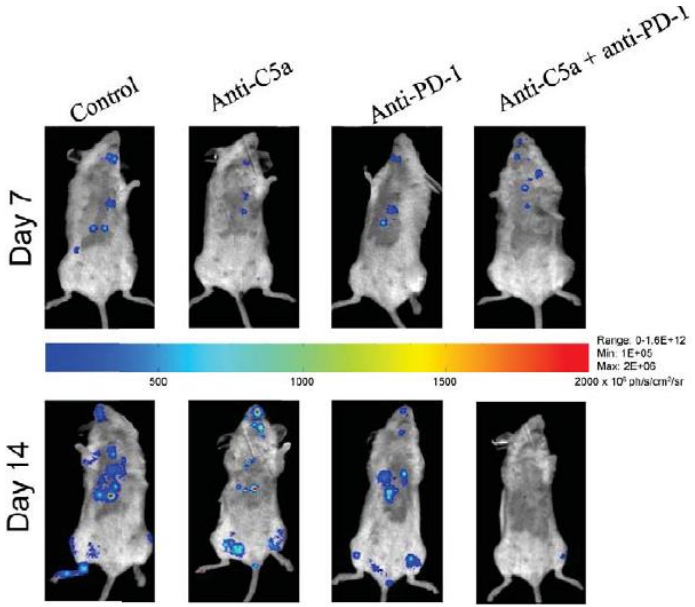
C5a induces the production of immunosuppressive cytokines in human monocytes

Ling-Ling An et al., Sci Rep., 2016

C5A BLOCKADE DECREASES LUNG CANCER GROWTH AND METASTASIS, AND SYNERGIZES WITH ANTI PD-1

393 P model: Kras driven highly aggressive lung adenocarcinoma





Prevention of multiorgan metastasis

Similar efficacy observed in a model of Lewis lung cancer

Ajona D et al; Cancer Discovery 2017