FORWARD LOOKING STATEMENT

This document has been prepared by Innate Pharma S.A. (the “Company”) solely for the purposes of a presentation to investors concerning the Company. This document is not to be reproduced by any person, nor to be distributed.

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 these data comes from external sources that are recognized in the field or from Company’s estimates based on such sources.

The information contained herein has not been independently verified. No representation, warranty or undertaking, express or implied, is made as to, and no reliance should be placed on, the fairness, accuracy, completeness or correctness of the information or opinions contained herein. The Company is under no obligation to keep current the information contained in this presentation and any opinion expressed is subject to change without notice. The Company shall not bear any liability whatsoever for any loss arising from any use of this document or its contents or otherwise arising in connection therewith.

Please refer to the Document de Référence filed with the Autorité des marchés financiers (“AMF”) on April 7, 2014, 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.
THERAPEUTIC POTENTIAL OF NK CELLS IN ACUTE MYELOID LEUKEMIA

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

Effects are:
- Durable
- Safe
- Controlled by KIR
- Mediated by NK cells

Velardi et al., Science, 2002 (not shown)
Ruggeri et al, Blood, 2007
<table>
<thead>
<tr>
<th>PROGRAM</th>
<th>TARGET</th>
<th>INDICATIONS AND SETTING</th>
<th>ONGOING STUDIES</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Lirilumab</strong></td>
<td>KIR2DL1,2,3</td>
<td>AML, single agent</td>
<td>• Randomized Phase II</td>
</tr>
<tr>
<td>(IPH2102/BMS-986015)</td>
<td></td>
<td>Solid &amp; heme tumors</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Multiple combinations</td>
<td></td>
</tr>
<tr>
<td>licensed to</td>
<td></td>
<td></td>
<td>• 4 Phase I</td>
</tr>
<tr>
<td>Bristol-Myers</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Squibb</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>IPH2201</strong></td>
<td>NKG2A</td>
<td>Head and Neck, CLL, Ovarian</td>
<td>• First Phase II start in 2H14</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Single agent and combinations</td>
<td></td>
</tr>
<tr>
<td><strong>IPH4102</strong></td>
<td>KIR3DL2</td>
<td>Cutaneous T-cell lymphomas</td>
<td>• Phase I start in 2015</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>IPH33</strong></td>
<td>TLR3</td>
<td>Inflammation / Autoimmunity</td>
<td>• Preclinical</td>
</tr>
<tr>
<td><strong>IPH43</strong></td>
<td>MICA</td>
<td>Cancer</td>
<td>• Preclinical</td>
</tr>
<tr>
<td><strong>Other / Discovery</strong></td>
<td>Undisclosed</td>
<td>Cancer / Inflammation</td>
<td>• Preclinical</td>
</tr>
</tbody>
</table>
### Clinical Pipeline of Immunomodulating mAbs in Cancer

<table>
<thead>
<tr>
<th>Target</th>
<th>Phase I</th>
<th>Phase II</th>
<th>Phase III</th>
<th>Market</th>
</tr>
</thead>
<tbody>
<tr>
<td>CTLA-4</td>
<td></td>
<td>AZN</td>
<td></td>
<td>BMS</td>
</tr>
<tr>
<td>PD-1 / PD-L1</td>
<td>Merck KGaA, AZN, AMP/GSK, BMS</td>
<td></td>
<td>Roche, AZN</td>
<td>BMS/ONO, Merck</td>
</tr>
<tr>
<td>KIR</td>
<td>IPH/BMS</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LAG-3</td>
<td>BMS</td>
<td></td>
<td>IMP</td>
<td></td>
</tr>
<tr>
<td>NKG2A</td>
<td>IPH</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Checkpoint Inhibitors**

- CD137: Pfizer, BMS
- B7-H3: Servier/MGNX
- CD40: CRUK, Roche
- OX40: AZN, Roche
- GITR: GITR Inc, Merck
- CD27: Cellidex

**Agonist Antibodies**

AMP: Amplimmune; CRUK: Cancer Research UK; MGNX: MacroGenix; IMP: Immutep
INNATE PHARMA VALUE PROPOSITION
FIRST-IN-CLASS AGENTS IN IMMUNO-ONCOLOGY

• Heterogeneity in cancers, between patients or intra tumoral opens opportunity for differentiation for novel agents

Patient stratification and Combination therapy

• Multiple agents needed to address large population of patients and maximize outcome
  > IPH compounds target untapped part of the immune system
  > Targeting NK cells expected to be very well tolerated
LIRILUMAB
FIRST-IN-CLASS NK CELL CHECKPOINT INHIBITOR

- Development and commercialization rights licensed to Bristol-Myers Squibb
  - $35 million upfront, up to $430 million in milestone payments, double-digit royalties (signed July 2011)
RATIONALE FOR COMBINING LIRILUMAB AND T-CELL CHECKPOINT AGENTS

Activation through KIR-blockade

Stimulation of T cells by NK cells

Activation through CTLA-4 or PD-1-blockade
LIRILUMAB ENHANCES ADCC FUNCTION OF NK CELLS

Kohrt et al., Blood, 2013
<table>
<thead>
<tr>
<th></th>
<th>SETTING</th>
<th>PATIENTS (PLANNED)</th>
<th>INDICATION</th>
<th>STATUS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Monotherapy</strong></td>
<td>Randomized Phase II</td>
<td>150</td>
<td>Acute Myeloid Leukemia Maintenance setting</td>
<td>LFS expected end of 2015</td>
</tr>
<tr>
<td><strong>Nivolumab</strong></td>
<td>Phase I with cohort expansion</td>
<td>162</td>
<td>Selected solid tumors: MEL, NSCLC, GI, SCCHN, HCC</td>
<td>Enrolment close to completion</td>
</tr>
<tr>
<td><strong>Ipilimumab</strong></td>
<td>Phase I with cohort expansion</td>
<td>125</td>
<td>Selected solid tumors: NSCLC, Castrate resistant Prostate cancer, Melanoma</td>
<td>In dose escalation</td>
</tr>
<tr>
<td><strong>Elotuzumab</strong></td>
<td>Phase I with randomized cohort expansion</td>
<td>136</td>
<td>Multiple myeloma: Relapsed/refractory MM Post autologous transplant</td>
<td>Started in October 2014</td>
</tr>
<tr>
<td><strong>Nivolumab</strong></td>
<td>Phase I</td>
<td>180</td>
<td>Selected hematologic tumors: Relapsed/refractory NHL, HL, MM or CML</td>
<td>Started in October 2014</td>
</tr>
</tbody>
</table>
ONGOING CLINICAL PROGRAM WITH LIRILUMAB

• 5 ongoing trials with lirilumab
  > 500+ patients, heme & solid tumors, single-agent and combinations

• Multiple rationale of NK cell activation explored
  > Direct cytotoxic activity
  > Interplay with T cells
  > ADCC enhancement

• Well tolerated in Phase I trial
  > Highest dose selected in nivolumab + liriumab dose escalation

• Start of activity read out in 2015

ClinicalTrials.gov Identifier: NCT01714739
EFFIKIR PHASE II TRIAL
DOUBLE-BLIND PLACEBO-CONTROLLED RANDOMIZED TRIAL IN AML

• Target enrolment completed in July 2014 (150 patients)
• Data on LFS expected end of 2015 (100 events)

Minimization
- Elderly
  - 1st complete remission
  - Max 2 consolidations
  - Not eligible for HST
- Center
  - 1º vs 2º AML
  - No. consolidations
  - Cytogenetics

1:1:1

Randomize
- Lirilumab 0.1 mg/kg q 12 weeks
  - Intermittent full KIR occupancy
- Lirilumab 1.0 mg/kg q 4 weeks
  - Continuous full KIR occupancy
- Placebo q 4 weeks

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
Maximum follow-up period: 24 months after last patient entry

ClinicalTrials.gov Identifier: NCT01687387
NKG2A IS A INHIBITORY RECEPTOR ON TUMOR INFILTRATING CD8 T CELLS AND NK CELLS

Upregulation of NKG2A on NK cells inside tumors

NKG2A on tumor infiltrating CD8+ T cells

Lung carcinoma

Blood NK (HC)

Intratumoral NK

Cervical cancer

Blood

Tumor

From L to R: Platonova et al. 2011, Sheu et al. 2005
IPH2201 TARGETS BOTH NK AND CD8 T CELLS

NK and T-cell inhibition by NKG2A

Activation through NKG2A-blockade
**BROAD POTENTIAL FOR DEVELOPMENT OF IPH2201**

- HLA-E upregulated on a wide variety of tumor types

<table>
<thead>
<tr>
<th>Tumor</th>
<th>Healthy tissue</th>
</tr>
</thead>
<tbody>
<tr>
<td>(17/21) 81%</td>
<td>Colorectal</td>
</tr>
<tr>
<td>(39/55) 71%</td>
<td>Ovarian</td>
</tr>
<tr>
<td>(22/33) 67%</td>
<td>Oesophagus</td>
</tr>
<tr>
<td>(13/33) 39%</td>
<td>Lung</td>
</tr>
<tr>
<td>(38/79) 48%</td>
<td>Melanoma</td>
</tr>
</tbody>
</table>

*Internal data*
## DEVELOPMENT PLAN

<table>
<thead>
<tr>
<th>Year</th>
<th>Events</th>
</tr>
</thead>
<tbody>
<tr>
<td>2014</td>
<td>H&amp;N Phase II single agent</td>
</tr>
<tr>
<td>2015</td>
<td>H&amp;N Phase II combo cetuximab</td>
</tr>
<tr>
<td>2016</td>
<td>CLL Phase II combo ibrutinib</td>
</tr>
<tr>
<td>2017</td>
<td>Ovarian Phase II single agent</td>
</tr>
<tr>
<td></td>
<td>Ovarian Phase II combo SOC</td>
</tr>
</tbody>
</table>

### Opportunities
- Integrated biomarker studies
- Other relevant indications and combinations (e.g., with checkpoint inhibitors)
KIR3DL2 is specifically expressed on cutaneous and circulating CTCL cells

- Irrespectively of disease grade and on most subtypes
- Restricted expression on normal tissues

Patients biopsies stained with Innate’s anti-KIR3DL2 mAb

SS pt #1, grade IIIB 86.5% KIR3DL2⁺ tumor cells
tMF pt #1, grade IIIB 96% KIR3DL2⁺ tumor cells
CD30⁺ LPD pt #3, grade IB 81% KIR3DL2⁺ tumor cells
POTENT ANTITUMOR ACTIVITY IN MODELS OF ADVANCED CTCL

- Humanized cytotoxic IgG1
- Start of Phase I expected in 2015
- Orphan drug status in EU for the treatment of cutaneous T-cell lymphoma

RAJI-KIR3DL2 SC xenograft model

Autologous ADCC with CTCL patient cells

Patient #10 (representative of n = 15)

WHAT’S NEW IN 2014 / WHAT TO EXPECT IN 2015

Lirilumab
• 3 ongoing trials: single agent in AML and solid tumors in combination
• 2 new combination trials in heme malignancies

Lirilumab clinical trials read-out

IPH2201
• Acquisition of full rights to IPH2201
• Phase II clinical development plan

IPH2201 Phase II trials roll out

IPH4102
• Orphan drug designation in the European Union

IPH4102 Phase I trial start

Corporate
• €70m raised from specialist investors
• Expanded clinical team

Further pipeline growth
CLINICAL EXPERIENCE WITH HYBRIDOMA ANTI-KIR IPH2101 PHASE I IN ACUTE MYELOID LEUKEMIA

- Elderly AML patients in complete remission after induction and consolidation treatment - maintenance setting
- Phase I dose-escalation including 23 patients in first CR, and extension including 12 patients
- Doses ranged from 0.0003 to 3 mg/kg – Full KIR saturation at doses ≥1mg/kg
- Good tolerance with mild and transient adverse events. MTD not reached. Clear PK/PD relationship
- Clinical outcome (2 patients from extension excluded, one in CR2 and one for early relapse within 5-days)

<table>
<thead>
<tr>
<th>Dose</th>
<th>N*</th>
<th>PFS (months)</th>
<th>OS (months)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;1 mg/kg</td>
<td>16</td>
<td>2.3</td>
<td>12.6</td>
</tr>
<tr>
<td>1-3 mg/kg</td>
<td>16</td>
<td>9.5</td>
<td>20.0</td>
</tr>
<tr>
<td>HR (95%CI)</td>
<td>16</td>
<td>0.515</td>
<td>0.490</td>
</tr>
<tr>
<td>P-value</td>
<td></td>
<td>0.075</td>
<td>0.076</td>
</tr>
</tbody>
</table>

Sources: Vey et al., Blood Sept. 21 and ASH 2013 poster
CLINICAL EXPERIENCE WITH HYBRIDOMA ANTI-KIR IPH2101 COMBINATION PHASE I IN MULTIPLE MYELOMA

• Phase I dose-escalation of IPH201 in combination with lenalidomide (LEN) in 15 patients with relapsed/refractory MM
  > Prior therapies: one prior line: 10 pts; 2 prior lines: 5 pts; prior LEN: 10 pts
• Treatment:
  > 4 cycles of IPH2101 and LEN; 5 pts received 4 additional cycles
  > No use of corticosteroids
• Results
  > Combination generally well tolerated
  > IPH2101 PK and PD not affected by co-administration of LEN
  > Objective responses observed in 33.3% of patients with and without prior LEN exposure. Median PFS of 24 months

<table>
<thead>
<tr>
<th>Overall best response</th>
<th>Total</th>
<th>Dose lirilumab - lenalidomide</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>0.2mg/kg - 10mg</td>
</tr>
<tr>
<td>VGPR</td>
<td>2 (13.3%)</td>
<td>1</td>
</tr>
<tr>
<td>PR</td>
<td>3 (20%)</td>
<td>1</td>
</tr>
<tr>
<td>MR/SD</td>
<td>7 (46.7%)</td>
<td>3</td>
</tr>
</tbody>
</table>

Source: Benson et al., ASH 2013 poster, manuscript in preparation
PHASE I WITH LIRILUMAB

• Single-agent Phase I with a variety of hematologic and solid tumors
  > Slowly progressive or stable disease or in complete response
  > Does not allow measurement of tumoral response

• Primary endpoint: safety
  > Secondary endpoints: PK/PD

• 6 dose levels from 0.015 mg/kg to 10 mg/kg

• 37 patients treated

• Well tolerated, maximum tolerated dose not reached

• Safety profile consistent with earlier observations with IPH2101

• This study paved the way for the randomized Phase II EffiKIR trial with lirilumab
EFFIKIR POSITIONING IN ACUTE MYELOID LEUKEMIA
STRONG MEDICAL NEED IN ELDERLY PATIENTS

- 5-year survival rate in elderly patients with AML is 5 to 15%

- No current standard of care for elderly patients in post-induction setting

- Intensive development effort in AML focused on relapsed / refractory disease

- Lirilumab tested in maintenance for elderly patients

Treatment paradigm

<table>
<thead>
<tr>
<th>Patients &lt;60y (&lt;50%)</th>
<th>Patients &gt;60y (&gt;50%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Induction chemotherapy</td>
<td>Induction chemotherapy</td>
</tr>
<tr>
<td>CR 65-85%</td>
<td>CR ~ 50%</td>
</tr>
<tr>
<td>Consolidation then transplantation</td>
<td>Consolidation</td>
</tr>
<tr>
<td>Maintenance chemotherapy</td>
<td>Maintenance chemotherapy</td>
</tr>
<tr>
<td>~ 50%</td>
<td>~ 50%</td>
</tr>
<tr>
<td>EffiKIR trial</td>
<td>EffiKIR trial</td>
</tr>
</tbody>
</table>

Mortality (at 5-years):

- 70-80% (pts <60y)
- 85-95% (pts >60y)
EFFIKIR PHASE II TRIAL
DOUBLE-BLIND PLACEBO-CONTROLLED RANDOMIZED TRIAL IN AML

- Target enrolment completed in July 2014 (150 patients)
- Data on LFS expected end of 2015 (100 events)

Minimization

Elderly
1st complete remission
Max 2 consolidations
Not eligible for HST

Center
1st vs 2nd AML
No. consolidations
Cytogenetics

1:1:1

Lirilumab 0.1 mg/kg q 12 weeks
Intermittent full KIR occupancy

Lirilumab 1.0 mg/kg q 4 weeks
Continuous full KIR occupancy

Placebo q 4 weeks

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
Maximum follow-up period: 24 months after last patient entry

ClinicalTrials.gov Identifier: NCT01687387
KIR AND NKG2A EXPRESSION ON DIFFERENT SUBSETS OF NK CELLS

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

Internal data
NKG2A EXPRESSION ON TUMOR INFILTRATING LYMPHOCYTES

Upregulation of NKG2A on NK cells inside tumors

NKG2A on tumor infiltrating CD8+ T cells

Lung carcinoma

Blood NK (HC)

Intratumoral NK

Cervical cancer

Blood

Tumor

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

<table>
<thead>
<tr>
<th>In thousands of euros (IFRS)</th>
<th>2014</th>
<th>2013</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Current revenue and other income</strong></td>
<td>4,137</td>
<td>6,978</td>
</tr>
<tr>
<td>Research and development</td>
<td>(10,890)</td>
<td>(7,003)</td>
</tr>
<tr>
<td>General and administrative</td>
<td>(2,310)</td>
<td>(2,152)</td>
</tr>
<tr>
<td><strong>Operating expenses</strong></td>
<td>(13,200)</td>
<td>(9,155)</td>
</tr>
<tr>
<td><strong>Operating income/(loss)</strong></td>
<td>(9,063)</td>
<td>(2,177)</td>
</tr>
<tr>
<td>Financial income, net, and others</td>
<td>25</td>
<td>-145</td>
</tr>
<tr>
<td><strong>Net loss</strong></td>
<td>(9,039)</td>
<td>(2,323)</td>
</tr>
<tr>
<td>Weighted average number of shares (in thousands):</td>
<td>47,337</td>
<td>38,003</td>
</tr>
<tr>
<td>Net loss per share</td>
<td>(0.19)</td>
<td>(0.06)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>June 30, 2014</th>
<th>Dec. 31, 2013</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cash, cash equivalents and financial instruments</strong></td>
<td>78,913</td>
<td>41,348</td>
</tr>
<tr>
<td>Total financial debt</td>
<td>4,425</td>
<td>4,819</td>
</tr>
</tbody>
</table>

- Cash position 3Q 2014: €74.7m / Cash horizon: end 2017
SHARE INFORMATION

- Listed on Euronext, IPO in November 2006
  - Euronext Paris: FR0010331421 – IPH
  - €178m raised since inception

- Stock liquidity (in 2014)
  - 53.0m outstanding shares (54.4m diluted)
  - Average daily trading volume >500,000

- Analyst coverage:
  - Citi Research
  - Gilbert Dupont
  - Goldman Sachs
  - Leerink Partners
  - Oddo Securities

- Shareholders represented at the Supervisory Board:
  - Bpifrance Participations *
  - Novo Nordisk A/S *
  - Wellington Management
  - OrbiMed
  - Fidelity Management
  - Other

* Shareholders represented at the Supervisory Board
Yannis Morel
PhD,
Chief Business Officer
Innate Pharma

Pierre Dodion
MD, MBA,
Chief Medical Officer
ARIAD, Pfizer, Novartis, Aventis

Marcel Rozencweig,
MD,
President, Innate Pharma Inc.
Bristol-Myers Squibb

Jérôme Tiollier
PhD,
Chief Development Officer
Pasteur Merieux Sangstat

Catherine Moukheibir
MBA,
Sr Advisor Finance
Movetis, Zeltia, Morgan Stanley

Nicolai Wagtmann
PhD,
Chief Scientific Officer
Novo Nordisk A/S

Hervé Brailly
PhD, CEO & Co-founder
Immunotech SA, Beckman-Coulter
Lirilumab, anti-KIR

> Kohrt et al., 2014. Anti-KIR antibody enhancement of anti-lymphoma activity of natural killer cells as monotherapy and in combination with anti-CD20 antibodies. *Blood*

> Vey et al., 2012. A phase 1 trial of the anti-inhibitory KIR mAb IPH2101 for AML in CR. *Blood*

> Romagne et al., 2009. Preclinical characterization of 1-7F9, a novel human anti-KIR receptor therapeutic antibody that augments natural killer-mediated killing of tumor cells. *Blood*

> Vahlne et al., 2010. In vivo tumor cell rejection induced by NK cell inhibitory receptor blockade: maintained tolerance to normal cells even in the presence of IL-2. *European journal of immunology*

> Moretta et al., 2008. Human NK cells: from HLA class I-specific killer Ig-like receptors to the therapy of acute leukemias. *Immunological reviews*

IPH2201, anti-NKG2A

- Levy et al., 2008. HLA-E protein is overexpressed in primary human colorectal cancer. *International journal of oncology*

- Iwaszko et al., 2011. Clinical significance of the HLA-E and CD94/NKG2 interaction. *Archivum immunologiae et therapiae experimentalis*

- Gunturi et al., 2005. The role of TCR stimulation and TGF-beta in controlling the expression of CD94/NKG2A receptors on CD8 T cells. *European journal of immunology*


- Derre et al., 2006. Expression and release of HLA-E by melanoma cells and melanocytes: potential impact on the response of cytotoxic effector cells. *J Immunol*

- Malmberg et al., 2002. IFN-gamma protects short-term ovarian carcinoma cell lines from CTL lysis via a CD94/NKG2A-dependent mechanism. *The Journal of clinical investigation*

- Levy et al., 2009. Cetuximab-mediated cellular cytotoxicity is inhibited by HLA-E membrane expression in colon cancer cells. *Innate immunity*

- Godal et al., 2010. NK cell killing of AML and ALL blasts by killer cell Ig-like receptor-negative NK cells after NKG2A and LIR-1 blockade. *Journal of the American Society for Blood and Marrow Transplantation*

- Nguyen et al., 2005. NK-cell reconstitution after haploidentical hematopoietic stem-cell transplantations: immaturity of NK cells and inhibitory effect of NKG2A override GvL effect. *Blood*
BIBLIOGRAPHY

**IPH4102, anti-KIR3DL2**

> Sicard et al., 2014. IPH4102, a Humanized KIR3DL2 Antibody with Potent Activity against Cutaneous T-cell Lymphoma. *Cancer Research*

> Bouaziz et al., 2010. Absolute CD3+ CD158k+ lymphocyte count is reliable and more sensitive than cytomorphology to evaluate blood tumour burden in Sezary syndrome. *The British journal of dermatology*

> Bagot et al., 2001. CD4(+) cutaneous T-cell lymphoma cells express the p140-killer cell immunoglobulin-like receptor. *Blood*

**IPH43, anti-MICA**

> Bauer et al., 1999. Activation of NK cells and T cells by NKG2D, a receptor for stress-inducible MICA. *Science*


> Champsaur, M., and L.L. Lanier. 2010. Effect of NKG2D ligand expression on host immune responses. *Immunological reviews*

**IPH33, anti-TLR3**

> Cavassani et al., 2008. TLR3 is an endogenous sensor of tissue necrosis during acute inflammatory events. *The Journal of experimental medicine*
