CTCL LUNCHEON
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PROF. OF
DERMATOLOGY,
STANFORD CANCER
INSTITUTE & SCHOOL
OF MEDICINE
NEW YORK,
OCTOBER 2, 2015
FORWARD LOOKING STATEMENT

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INNATE PHARMA AT A GLANCE

- Leading scientific edge in innate immunity pharmacology
- Primary focus in immuno-oncology
- Portfolio of first-in-class checkpoint inhibitors
- Partnerships with leaders in IO BMS and AZN
A LONG TERM STRATEGY
CREATE AND RETAIN MAXIMAL VALUE

NK Cell
R&D collaboration
2003 - 2009

Lirilumab (PII)
• License (2011)
• Upfront payment: $35 million
• Milestone payments: up to $430 million
• Double-digit royalties on sales

IPH2201 (PII)
• Global co-development and commercialization (2015)
• Initial payment: $250 million
• Further milestone payments: up to $925 million
• Double digit royalties on sales
• Right to co-promote in Europe (50% profit share)

IPH4102
In house development

Inception

2000
2005
2010
2015
2020

NK Cell
R&D collaboration
2003 - 2009

Lirilumab (PII)
• License (2011)
• Upfront payment: $35 million
• Milestone payments: up to $430 million
• Double-digit royalties on sales
<table>
<thead>
<tr>
<th>PROGRAM</th>
<th>TARGET</th>
<th>INDICATIONS AND SETTING</th>
<th>ONGOING STUDIES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lirilumab</td>
<td>KIR2DL1,2,3</td>
<td>AML, single agent</td>
<td>• Randomized Phase II</td>
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<tr>
<td>(IPH2102/BMS-986015)</td>
<td></td>
<td>Solid &amp; heme tumors</td>
<td>• 6 Phase I and II trials</td>
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<tr>
<td>licensed to</td>
<td></td>
<td>Multiple combinations</td>
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<td>Bristol-Myers Squibb</td>
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<tr>
<td>IPH2201</td>
<td>NKG2A</td>
<td>Solid &amp; heme tumors</td>
<td>• Phase II</td>
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<td>co-development with</td>
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<td>Multiple combinations</td>
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<td>AstraZeneca</td>
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<tr>
<td>IPH4102</td>
<td>KIR3DL2</td>
<td>Cutaneous T-cell lymphomas</td>
<td>• Phase I to start in 2015</td>
</tr>
<tr>
<td>IPH33</td>
<td>TLR3</td>
<td>Inflammation / Autoimmunity</td>
<td>• Preclinical</td>
</tr>
<tr>
<td>IPH43</td>
<td>MICA</td>
<td>Cancer</td>
<td>• Preclinical</td>
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<tr>
<td>Other / Discovery</td>
<td>Undisclosed</td>
<td>Cancer / Inflammation</td>
<td>• Preclinical</td>
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</table>
Youn H. Kim, M.D., is an internationally renowned expert in cutaneous lymphomas.

She serves as Director of the Multi-disciplinary Cutaneous Lymphoma Program, Director of the Residency Program in the Department of Dermatology, and Medical Director of the Photopheresis Unit at Stanford Medical Center. Dr. Kim is also Co-Director of the Lymphoma Research Program at the Stanford Cancer Institute and the first recipient of Stanford University's Joanne and Peter Haas Jr. Professorship for Cutaneous Lymphoma Research. She is a member of the National Comprehensive Cancer Network Non-Hodgkin's Lymphoma Panel.

Dr. Kim received her medical degree and completed her residency at Stanford University School of Medicine. She is Board Certified in Dermatology.
TARGETING KIR3DL2
WITH IPH4102 IN CTCL

Youn H Kim, MD

Department of Dermatology
Director, Multidisciplinary Cutaneous Lymphoma Group
Stanford Cancer Institute & School of Medicine
Youn Kim, MD

• **Steering Committee**
  > Eisai, Kyowa Hakko Kirin, Takeda/Millennium

• **Consultant or Advisory Board**
  > Actelion, Celgene, Galderma, Soligenix, Neumedicine, Seattle Genetics, MiRagen, Horizon

• **Investigator**
  > Kyowa Hakko Kirin, Merck, Takeda/Millennium, Seattle Genetics, Actelion, Eisai, Genentech, Tetralogic, Innate

• **No financial compensation or honorarium from Innate**
FIRST-IN-HUMAN TRIAL OF ANTI-KIR3DL2 IPH4102 IN CTCL

SEPTEMBRE 2015
A RARE DISEASE WITH HIGH UNMET MEDICAL NEED
Cutaneous T-cell Lymphomas (CTCL)

<table>
<thead>
<tr>
<th>New WHO-EORTC Classification</th>
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</thead>
<tbody>
<tr>
<td>Mycosis fungoides and variants/subtypes</td>
</tr>
<tr>
<td>Sézary syndrome</td>
</tr>
<tr>
<td>PC CD30+ lymphoproliferative disorders</td>
</tr>
<tr>
<td>Subcutaneous panniculitis-like T-cell lymphoma</td>
</tr>
<tr>
<td>Cutaneous γ/δ T-cell lymphoma</td>
</tr>
<tr>
<td>Adult T-cell leukemia/lymphoma</td>
</tr>
<tr>
<td>PC peripheral T-cell lymphoma, unspecified</td>
</tr>
<tr>
<td>• Aggressive epidermotropic CD8+ T-cell lymphoma</td>
</tr>
<tr>
<td>• CD4+ sm/med-sized pleomorphic T-cell LPD</td>
</tr>
<tr>
<td>• PTCL, other</td>
</tr>
</tbody>
</table>

CTCL is heterogeneous group of NHL presenting primarily in the skin, of malignant clonal T cells

Blood 2005;105:3768-85
WHO monogram, 4th Ed, 2008
MYCOSIS FUNGOIDES & SÉZARY SYNDROME
Most common of the cutaneous T-cell lymphomas

• Continued rising annual incidence in US (SEER)$^1$
  > 0.96 per 100,000
    • 3,000 new cases
  > ↑ by 2.9 per million per decade
  > 4% of NHLs
• Median age at diagnosis is 55-65 yrs
  > Two-thirds present with early stage disease (IB-IIA)
• Factors predictive of disease progression or survival$^{2,3}$
  > Advanced skin involvement (greater BSA, tumors/erythroderma)
  > Involvement of sites other than skin
  > Older age, male gender, blacks
  > Folliculotropism (hair follicle involvement- deeper)
  > Large cell transformation (change to larger cells, rapid growth)
  > Increased LDH (blood marker of lots of disease, more than skin)

Mycosis Fungoides
Treatment of Varying Skin Manifestations

Patch T1-2

Plaque T1-2

Erythroderma T4

Tumor T3
MANAGEMENT OF EXTRACUTANEOUS DISEASE

Blood

Viscera

Lymph node

T6 (52.293, 22.150)
Sézary syndrome—
generalized erythroderma, keratoderma, *severe itching*;
freq staph aureus infection
Mycosis fungoides (MF) and Sézary syndrome (SS), its leukemic variant, are the most common CTCL subtypes.

Overall survival depends in part on disease subtype.

Survival decreased with advancing T class and clinical stage DSS utilizing revised staging system

Plaque worse than patch disease

Agar et al. J Clin Oncol 2010;28:4730
<table>
<thead>
<tr>
<th>IA</th>
<th>IB/IIA</th>
<th>IIB</th>
<th>III</th>
<th>IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Limited patch/plaque</td>
<td>Generalized patch/plaque</td>
<td>Tumors</td>
<td>Erythroderma</td>
<td>Extracutan disease</td>
</tr>
</tbody>
</table>

- **IA** Limited patch/plaque: Topical steroid, retinoid (bex), NM phototherapy, local RT, imiquimod
- **IB/IIA** Generalized patch/plaque: ECP + IFN, bexarotene
- **IIB** Tumors: Phototherapy + bexarotene or IFN, TSEBT + ECP, IFN
- **III** Erythroderma: Bexarotene, methotrexate, IFN, vorinostat, romidepsin, Alemtuzumab
- **IV** Extracutan disease: Combination chemo

No standard of care in CTCL. Many drugs are used, depending on stage and line of therapy, and often off label.

- Clinical Trials
- **brentuximab, pralatrexate**, liposomal doxorubicin, gemcitabine, other
- **Allo-HSCT**

**CURRENT CLINICAL MANAGEMENT OF CTCL, 2015**

WWW.NCCN.ORG => NHL => MF/SS
**EFFICACY OF SYSTEMIC AGENTS IN CTCL**

ONLY 4 AGENTS WITH FDA APPROVAL

<table>
<thead>
<tr>
<th>Agent (Class)</th>
<th>Indication</th>
<th>Year</th>
<th>Study</th>
<th>N</th>
<th>ORR</th>
<th>DOR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Romidepsin (HDAC inhibitor)</td>
<td>CTCL with prior systemic therapy</td>
<td>2009</td>
<td>Pivotal</td>
<td>96</td>
<td>34%</td>
<td>15 mo</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Supportive</td>
<td>71</td>
<td>35%</td>
<td>11 mo</td>
</tr>
<tr>
<td>Denileukin diftitox (Fusion protein)</td>
<td>Tumors that express CD25</td>
<td>1999, 2008</td>
<td>Pivotal</td>
<td>71</td>
<td>30%</td>
<td>4 mo</td>
</tr>
<tr>
<td>Bexarotene (RXR activator)</td>
<td>Cutaneous manifestations</td>
<td>1999</td>
<td>Pivotal</td>
<td>62</td>
<td>32%</td>
<td>5+ mo</td>
</tr>
<tr>
<td>Vorinostat (HDAC inhibitor)</td>
<td>Cutaneous manifestations</td>
<td>2006</td>
<td>Pivotal</td>
<td>74</td>
<td>30%</td>
<td>6+ mo</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Supportive</td>
<td>33</td>
<td>24%</td>
<td>4 mo</td>
</tr>
</tbody>
</table>

**Need better therapies, more options:**

**Brentuximab vedotin (anti-CD30 ADC)**

**Mogamulizumab (anti-CCR4 mab)**

*Both in phase 3 trials*
IMMUNE MODULATION WITH MABS IN CTCL

Tumor cell-specific:
tumor surface
cellular molecules
(e.g., CD4, CD25,
CD30, CD52, CCR4,
KIR3DL2/CD158k)

Monoclonal antibodies

Microenvironment
Immune modulation
(e.g. PD-1, PD-L1,
CTLA-4, CD47/SIRP, IDO, MDSC, Tregs)

CD8+ TILs

CTCL
DRUGS IN DEVELOPMENT IN CTCL
MOGAMULIZUMAB (ANTI-CCR4)

• CCR4: trafficking receptor for systemic memory Th2 and regulatory T cells to skin and lung.

• Preliminary results:
  > Phase I/II study in MF + SS patients (38 evaluable pts):
    • ORR = 36.8% (28.6% in 21 MF patients and 47.1% in 17 SS patients)
    • Median PFS = 11.4 months
    • Median DOR = 10.4 months
    • Mogamulizumab well tolerated

• Ongoing clinical development
  > randomized Phase III against vorinostat (NCT01728805)
  > Relapsed/refractory stage ≥ IB CTCL, excluding transformed MF
DRUGS IN DEVELOPMENT IN CTCL
BRENTUXIMAB VEDOTIN (ANTI-CD30 DRUG CONJUGATE)

• CD30: is expressed on activated B, T and NK cells, and activated monocytes in hematopoietic tissues.

• Targeted delivery of potent tubulin disrupting drug (MMAE)

• Preliminary results:
  > 2 Phase II studies (48 pts with CD30+ MF/SS and LPD; 30 pts with MF/SS with various degree of CD30 expression)
  > ORR ~ 70%; CR rate 35 and 3% respectively
  > Median duration of response ~ 32+ weeks
  > Peripheral neuropathy reported in ~ 2/3 of the patients with a median time to improvement of 42-49 weeks

• Ongoing developments
  > Randomized Ph III against bexarotene or methotrexate (NCT01578499)
  > Relapsed/refractory CD30-positive CTCL, excluding Sézary syndrome

Kim et al, JCO 2015; Duvic et al, JCO 2015
51 yo F stage IVA2 MF with LCT in skin/LNs: response to brentuximab vedotin

Pre-treatment 12/20/2012
Post 2 cycles 1/29/2013

Responses are not durable
Preliminary Results of a Phase I Study of Nivolumab in Patients with Relapsed or Refractory Lymphoid Malignancies


1Memorial Sloan Kettering Cancer Center and Weill Cornell Medical College, New York, NY; 2Mayo Clinic, Rochester, MN; 3Dana-Farber Cancer Institute, Boston, MA; 4Oregon Health and Science University and the Knight Cancer Institute, Portland, OR; 5University of Utah Huntsman Cancer Institute, Salt Lake City, UT; 6John Theurer Cancer Center, Hackensack University Medical Center, Hackensack, NJ; 7Fox Chase Cancer Center, Philadelphia, PA; 8Abramson Cancer Center, University of Pennsylvania, Philadelphia, PA; 9University of Michigan Hematology, Ann Arbor, MI; 10Yale Cancer Center, New Haven, CT; 11Beth Israel Deaconess Medical Center, Boston, MA; 12Brigham and Women’s Hospital Clinical Cytogenetics Laboratory, Boston, MA; 13Bristol-Myers Squibb, Princeton, NJ; 14Johns Hopkins University School of Medicine and the Sidney Kimmel Comprehensive Cancer Center, Baltimore, MD; 15Jonsson Comprehensive Cancer Center, University of California, Los Angeles, CA

56TH ANNUAL ASH MEETING
DECEMBER 2014
SAN FRANCISCO, CA
Study Design

Relapsed or Refractory HM (N=105)
- No autoimmune disease
- No prior organ or stem cell allografting
- No prior checkpoint blockade

Dose Escalation
Nivolumab
1mg/kg→3mg/kg
Wks 1,4 then q2w
(N=13)
- B-Cell Lymphoma (n=8)
- CML (n=1)
- Multiple Myeloma (n=4)

Dose Expansion (3mg/kg)
Hodgkin Lymphoma (n=23)
(N=69)
- B-Cell Lymphoma (n=23)
- T-Cell Lymphoma (n=23)
- Multiple Myeloma (n=23)

Endpoints

Primary
- Safety and Tolerability

Secondary
- Best Overall Response
  - Investigator assessed
- Objective Response
- Duration of Response
- PFS
- Biomarker studies
# Best Overall Response

<table>
<thead>
<tr>
<th>Disease</th>
<th>Objective Response Rate, n (%)</th>
<th>Complete Responses, n (%)</th>
<th>Partial Responses, n (%)</th>
<th>Stable Disease, n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>B-Cell Lymphoma* (n=29)</td>
<td>8 (28)</td>
<td>2 (7)</td>
<td>6 (21)</td>
<td>14 (48)</td>
</tr>
<tr>
<td>Follicular Lymphoma (n=10)</td>
<td>4 (40)</td>
<td>1 (10)</td>
<td>3 (30)</td>
<td>6 (60)</td>
</tr>
<tr>
<td>Diffuse Large B-Cell Lymphoma (n=11)</td>
<td>4 (36)</td>
<td>1 (9)</td>
<td>3 (27)</td>
<td>3 (27)</td>
</tr>
<tr>
<td>T-Cell Lymphoma† (n=23)</td>
<td>4 (17)</td>
<td>0 (0)</td>
<td>4 (17)</td>
<td>10 (43)</td>
</tr>
<tr>
<td>Mycosis Fungoides (n=13)</td>
<td>2 (15)</td>
<td>0 (0)</td>
<td>2 (15)</td>
<td>9 (69)</td>
</tr>
<tr>
<td>Peripheral T-Cell Lymphoma (n=5)</td>
<td>2 (40)</td>
<td>0 (0)</td>
<td>2 (40)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Multiple Myeloma (n=27)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>18 (67)</td>
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<tr>
<td>Primary Mediastinal B-Cell Lymphoma (n=2)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>2 (100)</td>
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*includes other B-cell lymphoma (n=8)
†includes other cutaneous T-cell lymphoma (n=3) and other non-cutaneous T-cell lymphoma (n=2)
INTRODUCTION

KIR3DL2 &
IPH4102 IN
CTCL
KIR3DL2, UNIQUE THERAPEUTIC TARGET IN CTCL

- Inhibitory receptor, member of the Killer Immunoglobulin-like Receptor (KIR) family
- In healthy individuals, there is limited expression of KIR3DL2 by normal blood cells (~25% NK cells and <15% T cells)
- No KIR3DL2 expression on the FDA panel of human tissues (IHC)
- KIR3DL2 is expressed by up to 95% CTCL cells irrespectively of disease stage and CTCL subtype (IHC study, N = 107 CTCL patients)
- Skin-resident CD4+ T cells express KIR3DL2 and may be the normal counterparts of CTCL (Sako et al, CytomA 2014)

→ KIR3DL2 is a very restricted and specific marker of CTCL
KIR3DL2 is expressed in ~65% of all CTCL, irrespectively of disease subtype. Expression is more prominent in Sézary syndrome, transformed mycosis fungoides and CD30+ LPD (ALCL subtype).
IHC staining of KIR3DL2 on skin biopsies of CTCL patients with Sézary syndrome, mycosis fungoides and CD30+ lymphoproliferative disorder (ALCL subtype)
Correlation between KIR3DL2 and TCR-Vβ expression in flow cytometry on blood CTCL cells in Sézary syndrome patients (n = 32)

Spearman $r = 0.6609$

$p < 0.0001$
IPH4102 HAS THE POTENTIAL TO BE A HIGHLY TARGETED CTCL THERAPY

• KIR3DL2 exhibits homogeneous expression on tumor cells
• KIR3DL2 has very limited expression on normal cells subsets
• Altogether, targeting KIR3DL2 may lead to
  > Improved targeting and better anti-tumoral effect
  > Improved safety profile
  > Higher exposure and longer treatment duration
  > Greater opportunity for combinations
IPH4102 IN CTCL

NON CLINICAL EFFICACY SUMMARY
**IPH4102 KEY FEATURES**

- **Selective binding to human KIR3DL2 with high affinity**
  > No cross-reaction to other human KIRs
- **Humanized IgG1 designed to deplete KIR3DL2-positive tumor cells**
- **Compelling efficacy in non clinical studies (large set of *in vitro, in vivo* and *ex vivo* models)**
  > Main MOA include ADCC and ADCP
  > Reduces tumor growth and improves survival in murine xenograft models of KIR3DL2⁺ tumors
  > Induces killing of primary CTCL tumors in the presence of patient autologous NK cells
- **Orphan Drug designation by the EU in 2014 for the treatment of CTCL**
- **Distinct anti-KIR3DL2 mAbs developed for biomarker purposes** (*IHC and flow cytometry*)
IPH4102 Efficacy in Mouse IV Models

IPH4102 improves survival in a dose-dependent manner.

Mice: SCID (n = 8)
RAJI-KIR3DL2: 5 M IV at D0
IPH4102: single IV admin. at D1
Read-out: survival
IPh4102 Efficacy *Ex Vivo*: Autologous ADCC

**Experimental Conditions**

- Fresh blood taken from Sézary Syndrome patients
- CD4 and NK cells separately sorted from PBMC of the same patient (by negative selection)
- NK and CD4 mixed at various E/T ratios + IPh4102 or alemtuzumab and rituximab as controls
- 4 to 6 hours incubation
- 7AAD incorporation used as marker of cell death
- Flow cytometry read-out
**IPH4102 EFFICACY EX VIVO: AUTOLOGOUS ADCC EFFICACY RESULTS**

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**Patient #10**

<table>
<thead>
<tr>
<th>E/T ratio</th>
<th>% killing</th>
</tr>
</thead>
<tbody>
<tr>
<td>T. only</td>
<td>0</td>
</tr>
<tr>
<td>1/10</td>
<td>20</td>
</tr>
<tr>
<td>1/5</td>
<td>40</td>
</tr>
<tr>
<td>1/1</td>
<td>60</td>
</tr>
<tr>
<td>5/1</td>
<td>80</td>
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- IPH4102
- rituximab
- alemtuzumab

**Patient #11**

<table>
<thead>
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<th>E/T ratio</th>
<th>% killing</th>
</tr>
</thead>
<tbody>
<tr>
<td>T. only</td>
<td>0</td>
</tr>
<tr>
<td>1/10</td>
<td>20</td>
</tr>
<tr>
<td>1/5</td>
<td>40</td>
</tr>
<tr>
<td>1/1</td>
<td>60</td>
</tr>
<tr>
<td>5/1</td>
<td>80</td>
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</table>

- IPH4102
- rituximab
- alemtuzumab

**Patient #17**

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<th>E/T ratio</th>
<th>% killing</th>
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</thead>
<tbody>
<tr>
<td>T. only</td>
<td>0</td>
</tr>
<tr>
<td>1/10</td>
<td>20</td>
</tr>
<tr>
<td>1/5</td>
<td>40</td>
</tr>
<tr>
<td>1/1</td>
<td>60</td>
</tr>
<tr>
<td>5/1</td>
<td>80</td>
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</tbody>
</table>

- IPH4102
- rituximab
- alemtuzumab

**Patient #7**

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<thead>
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</tr>
</thead>
<tbody>
<tr>
<td>T. only</td>
<td>0</td>
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<tr>
<td>1/10</td>
<td>20</td>
</tr>
<tr>
<td>1/5</td>
<td>40</td>
</tr>
<tr>
<td>1/1</td>
<td>60</td>
</tr>
<tr>
<td>5/1</td>
<td>80</td>
</tr>
</tbody>
</table>

- IPH4102
- rituximab
- alemtuzumab

**IPH4102 as potent as alemtuzumab in ex vivo autologous ADCC assays**

- mAb: 10 µg/mL
- Incubation time: 4 – 6 hours
- Read-out: 7AAD incorporation
- KIR3DL2 sites per cell: 1,000 to 4,000
- %KIR3DL2+ cells among CD4+ > 85%
- Total n = 15 patients

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Marie-Cardine A. et al, Cancer Res. 2014
OVERVIEW

IPH4102-101 FIH CLINICAL STUDY

OVERVIEW
**IPH4102-101 FIRST-IN-HUMAN STUDY DESIGN**

**OVERVIEW**

- First-in-Human Phase I study of IPH4102
- Dose-escalation + cohort expansion study

**Dose-escalation part:**
- 10 dose levels of repeated administrations of IPH4102
- Modified 3+3 design with accelerated titration
- First patient expected to be treated 4Q15
- Will determine the recommended Phase II dose and schedule

**Cohort expansion part:**
- Selected CTCL subtypes: SS and transformed MF
  - Highest unmet medical need, not fully addressed in the current Phase III trials
  - Most robust KIR3DL2 expression
- Start with n = 10 patients in each cohort, expandable according to signals of activity
Patient population:

- Relapsed/refractory (≥ 2 previous lines of systemic therapy) CTCL patients
  - All subtypes eligible
- For MF/SS patients: clinical stage ≥ IB
- KIR3DL2-positivity on skin biopsies is required for eligibility
  - Centrally assessed expression of KIR3DL2 on tumors
  - Allows more relevant assessment of IPH4102 safety profile
  - Allows detecting early signals of clinical activity
**OBJECTIVES**

- **Primary objective:** to assess safety & tolerability of increasing IV doses of single agent IPH4102 by:
  - characterizing the dose-limiting toxicities (DLT) and (S)AEs
  - identifying the MTD or Recommended Ph 2 Dose (RP2D)

- **Secondary objectives:**
  > To explore antitumor activity
  > To assess pharmacokinetics (PK) and immunogenicity

- **Translational objectives, biomarker exploration:**
  > To monitor the fate of KIR3DL2-expression cells in skin lesions, blood and lymph nodes (pharmacodynamics)
  > To monitor immune cell activation in blood and explore NK cell and macrophage infiltration in skin lesions
  > To assess Minimal Residual Disease (clonal Vβ chain)
  > To assess cytokine release
IPH4102-101 FIRST-IN-HUMAN STUDY DESIGN

STUDY DESIGN

• Dose-escalation Part:
  accelerated 3+3 design
  pts with KIR3DL2+ tumors
  all CTCL subtypes eligible

• Cohort expansion Part:
  same dose for all: RP2D
  pts with KIR3DL2+ tumors
  pre-selected CTCL subtypes

Recommended
Phase II Dose (RP2D)
e.g. n = 10 tMF + 10 SS

The CTCL subtypes and number
of pts will be adjusted based on
the findings during the dose
escalation phase
IPH4102-101 FIRST-IN-HUMAN STUDY DESIGN
PARTICIPATING SITES

- Two-continent Phase I study
- Clinical sites (dose-escalation part):
  - St Louis Hospital, Paris France (M Bagot)
  - UMC Leiden, the Netherlands (M Vermeer)
  - Guy’s and St Thomas’ Hospital, London UK (S Whittaker)
  - Stanford U, Stanford CA, US (Y Kim)
  - MD Anderson, Houston TX, US (M Duvic)
  - OSU, Columbus OH, US (P Porcu)
- Strong collaborative work between Innate Pharma and investigators
- First patient expected to be treated in October 2015
- IPH4102 IND-enabling data and Phase I design presented in Turin EORTC CLTF meeting, September 2015, by Martine Bagot
A NOVEL TARGETED IMMUNOTHERAPY FOR CTCL

- See Pr Martin Bagot presentation at EORTC cutaneous lymphoma task force meeting - Torino sept. 2015