

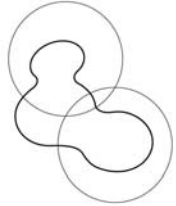
A hand holding a small glass vial with a white label, set against a background of overlapping white circles. The vial label contains handwritten text: "Lot 15", "Conc 6.26 (µg/ml)", "Date 02/10/09", and "PE".

innate pharma

Strategic update and 2009 Financial Results

March 5, 2010

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innate pharma

First-in-class
immunotherapeutics

- Immuno-pharmacology company:
 - > Therapeutic focus on cancer and inflammation
 - > Technology focus on antibody
- First-in-class immunotherapeutics
 - > Two proprietary Phase II drug candidates
- Track-record in translational research up to clinical proof-of-concept
- 80 FTEs, based in Marseilles, Lyon and New York
- Founded in 1999
- Public company listed on NYSE-Euronext in Paris (IPH) since 2006



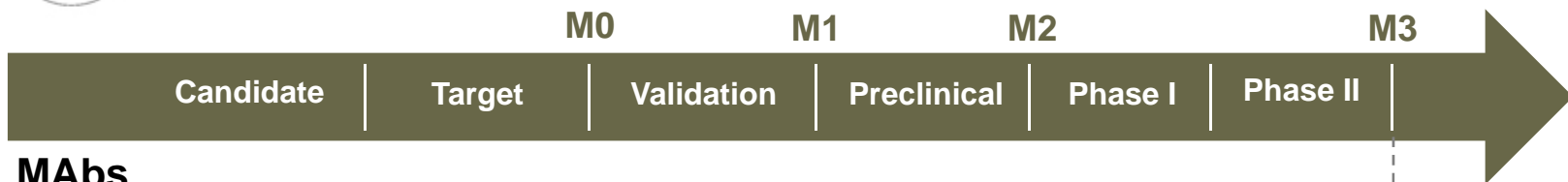
Key highlights of 2009

- Key clinical achievements:
 - Completion of IPH 2101 initial Phase I program, initiation of Phase II program
 - Encouraging Phase IIa results with IPH 1101 in HCV and fNHL
 - Final fNHL results expected mid-2010
- Focus on antibody development :
 - 2 proprietary mAb drug candidates – Termination of IPH 4201
 - 2 other mAb programs licensed to Novo Nordisk A/S
 - Agreement with Inserm Tranfert to feed portfolio with new targets in oncology and inflammation / auto-immunity
- Strengthening of the cash situation through a “PIPE” deal in December 2009:
 - €49.2m as at the end of the year



Portfolio of core drug candidates

From novel target to clinical proof-of-concept



MAbs

Oncology
Inflammation

IPH 2101	KIR2DL1,2,3	Multiple Myeloma / MMy	
		Acute Myeloid Leukemia / AML	
IPH 4101	KIR3DL2	CTCL	
IPH 2201 (NN8765)	undisclosed	Inflammation, autoimmunity	



Small molecule immune modulator

Oncology
Infections

IPH 1101	$\gamma\delta$ TCR	Follicular Lymphoma / fNHL	
		Type C Hepatitis / HCV	

Clinical proof-of-concept

- **To meet its objective, Innate Pharma's strategy is to:**

- > Bring assets to **clinical POC***
- > **Find partners:**
 - In cancer when access to global development capabilities is needed
 - In inflammation and infectious diseases
- > **Build its portfolio through:**
 - In-house capabilities
 - Acquisition and/or in-licensing

- **In the short run, the Company intends to:**

- > Execute Phase II program of IPH 2101
- > Complete the Phase II program for IPH 1101 in order to partner the program
- > Move IPH 4101 to clinical stage
- > Leverage on the newly signed collaboration with Inserm and other to source new antibody targets

**Proof-of-concept*



Update on drug candidates



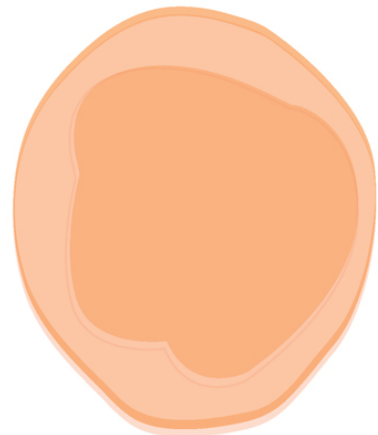
IPH 2101

Anti KIR

First mAb candidate
potentiating NK cells

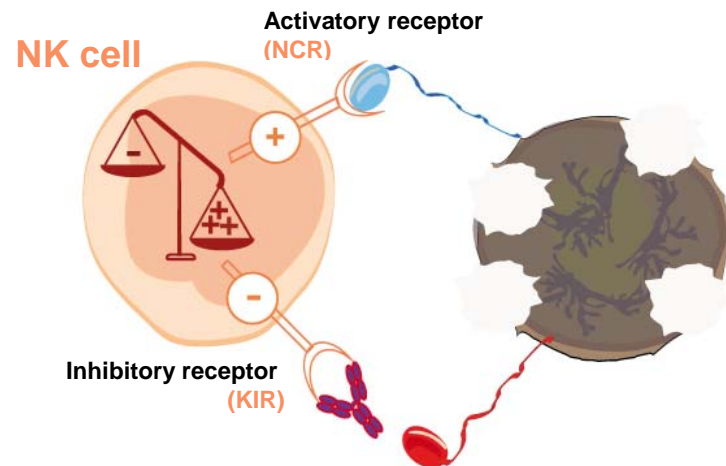
*Mechanism of action supported by clinical evidence
(Science, 2002; Blood, 2007 for AML and MMy)*

Next step: clinical proof-of-concept





Compound class:	Fully human antibody
Target / Mechanism of action:	Anti-KIR: potentiates the anti-tumor activity of natural killer (NK) cells
Indications in development:	Multiple Myeloma Acute Myeloid Leukemia
Development status:	Phase II

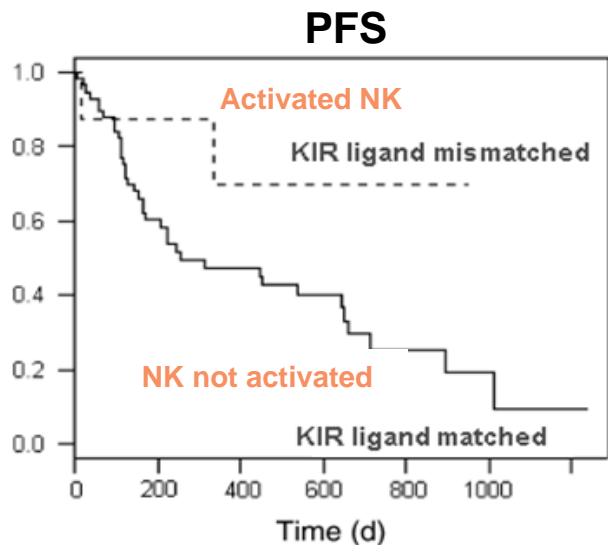


- Immune modulator, first antibody drug candidate to target KIR
 - > Closer competitors with respect to MOA* are anti-CTLA4 mAbs (potentiate T cells anti-tumor activity): ipilimumab (BMS, Phase III) and tremelimumab (Pfizer, Phase II)
- Long patent life providing market exclusivity
- Potential for horizontal market expansion in onco-hematology and then in solid tumors and possibly in chronic inflammation
- Bio-equivalent candidate IPH 2102 with improved manufacturing process

*Mechanism-of-action

Proven mechanism of action in AML and MMy

- MOA* supported by clinical evidence in studies on haploidentical hematopoietic transplantation for AML (*Science, 2002; Blood, 2007*) and MMy (*British Journal of Haematology, 2005*)



Demonstrated effect of NK cells on relapse and survival after ASCT for Multiple Myeloma

Kröger et al., Br. J. Hematol. 2005

- First indications selected based on scientific rationale, medical need and potential for rapid registration
- Phase I studies (ASCO, ASH 2009) demonstrated good safety profile as single agent, suggesting potential for combination with other agents

*Mechanism-of-action

Development strategy in MMy:

- Established biological endpoint in MMy (M-Protein) providing short term signal correlated with time-to-event endpoints such as PFS, TTP and OS

Three Phase IIa studies planned

Single agent in non-progressing settings: maintenance post first line therapy in stable residual disease (initiated) and smoldering myeloma (to be initiated)

Combination with standard of care lenalidomide (Revlimid[®], Celgene Corporation), in patients in first relapse (to be initiated, collaboration with Celgene Corporation)

Development strategy in AML:

- Envisaged positioning is extension of first remission requiring time-to-event endpoints (PFS, OS, etc) which can be addressed only through Phase III trials

Next study

Phase I extension in patients in first CR to confirm dose and schedule and document efficacy signals on DFS possibly leading to registration study (initiated)

Multiple Myeloma

Second hematological cancer, still incurable

Description of the disease and natural history :

- Monoclonal proliferation of plasma cells (differentiated B-cells producing immunoglobulins - Ig)
- Smoldering myeloma evolving into symptomatic myeloma – Smoldering myeloma is under-diagnosed
- OS < 2 years (< 1y if renal failure)

Population:

- Incidence / Mortality (G7): 40,900 / 27,400 patients/year
- Median age at diagnosis: 65-70y

Patients < 65 yrs:
5-yr survival 20-30%

Patients > 65 yrs:
5-yr survival 5-15%

Significant unmet medical needs:

- Newly approved treatments have improved patients' outcome but medical needs remain high:
 - > Drugs with lower toxicity, notably for elderly patients
 - > Consolidation / Maintenance after complete response
 - > Treatment of relapse (combination)
 - > Prevention of progression of smoldering MMy into symptomatic MMy

**IPH 2101
may bring significant
improvement in
these settings**

IPH 2101's new MOA:

- May shrink residual disease resistant to cytotoxic agents or targeted therapies
- Is expected to be well tolerated, key consideration notably for elderly patients (not suitable for intensification regimen)

Acute Myeloid Leukemia

Strong medical need for the most common type of adult leukemia

Description of the disease and natural history:

- Rapid growth and accumulation of abnormal and immature myeloblasts in the bone marrow; very heterogeneous disease
- Median age at diagnosis: 65 years; Overall survival: ~20%

Patients <60 yrs:
5-yr survival 20-30%

Patients >60 yrs:
5-yr survival 5-15%

Population:

- Incidence / Mortality (US): 13,000 / 9,000 patients/year

Significant unmet medical needs:

- No major progress for the last 10 years, no drug currently in development anticipated to have a large impact on patient outcomes
- Major medical needs in the following areas:
 - > Curative therapy beside transplant
 - > Non-cytotoxic drugs to overcome multidrug resistance
 - > Well tolerated drugs to expand remission particularly for elderly patients
 - > Novel therapies for relapsed / refractory disease

**Strong
rationale for
IPH 2101**

IPH 2101's new MOA:

- May shrink residual disease resistant to cytotoxic agents
- Is expected to be well tolerated, key consideration notably for elderly patients



IPH 1101

IPH 1201

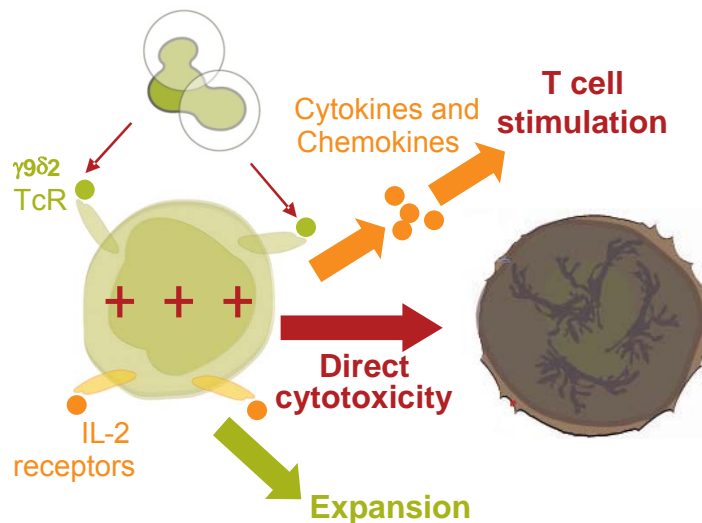
Small molecule
stimulating $\gamma\delta$ T cells

Most advanced program

POC data in 2009/2010 in NHL and in HCV

Objective is partnering-out

Compound class:	Small molecule (NCE)
Target / Mechanism of action:	Agonist of $\gamma\delta 2$ T-cells
Indications in development:	Follicular Lymphoma and Type C viral Hepatitis
Development status:	Phase II POC data in HCV reported POC data in NHL exp. 2010

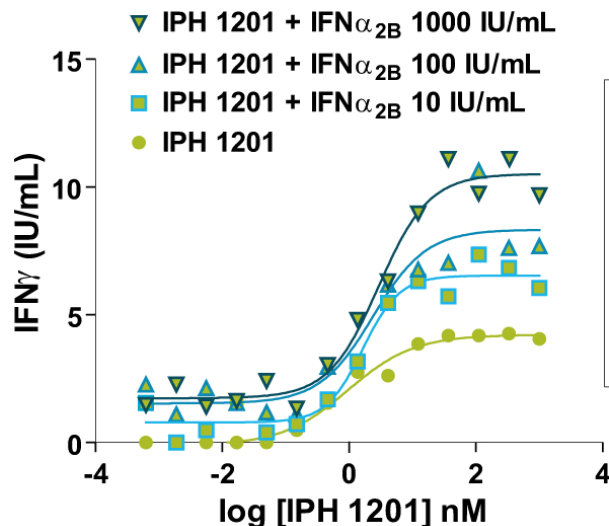


- Immune modulator, first molecule to specifically target $\gamma\delta$ T-cells
- Long patent life providing market exclusivity
- Proof-of-concept data in HCV reported (AASLD 2009)
- Encouraging interim data in FL reported (ASH 2009)

Perspective in Type C viral Hepatitis

Progresses with the emergence of the protease inhibitors

- New class of drug in late Phase III trial (protease inhibitors) in combination settings
- IFN α and ribavirin will remain the backbone of future treatment at all stages
- Evidence of antiviral activity with IPH 1101, very good tolerance
- IPH 1101 could synergize with current and future SOC notably by enhancing efficacy of IFN α and exerting both an anti viral and immunomodulatory effect
- IPH 1101 may provide an option to overcome virus mutations leading to resistance to targeted therapies (such as protease inhibitors)





Perspective in Follicular Lymphoma

Rituximab to remain key in the treatment

- Multiple agents in late development stage reflecting rituximab commercial success and persistent need to achieve sustained remission
- Rituximab status as market leader is unlikely to be challenged within the next 5-10 years
- Encouraging interim data with 32% complete response in the first set of 34 patients assessed by the independent central review (reference paper Davis et al. (*JCO*, 2000): 11% CRR expected in this population)
- Satisfactory and manageable overall tolerance and safety
- Enhancement of the effector function of $\gamma\delta$ T cells in combination with a cytotoxic antibody could have a significant clinical impact in the disease treatment



Potential positioning:

> Follicular Lymphoma:

- Combination with rituximab in first line or relapsing patients
- Combination with rituximab in advanced disease elderly patients

> Other indications:

- Combination with other cytotoxic mAbs (trastuzumab, etc)

> HCV:

- Combination with IFN α and ribavirin in non-responder patients to current and future SOC

Next steps:

- > Final data on primary efficacy endpoint in 2010
- > Controlled trial of IPH 1101 in combination with rituximab versus rituximab alone

- > Consolidation of clinical POC in a combination setting

Main objective is partnering-out once full set of cancer Phase II data is available



R&D update



Scientific positioning

Immune modulation and tumor targeting

TARGETED IMMUNOTHERAPY

Innate Pharma, Micromet, BMS, Pfizer, Roche/Genentech...

Immunomodulators*

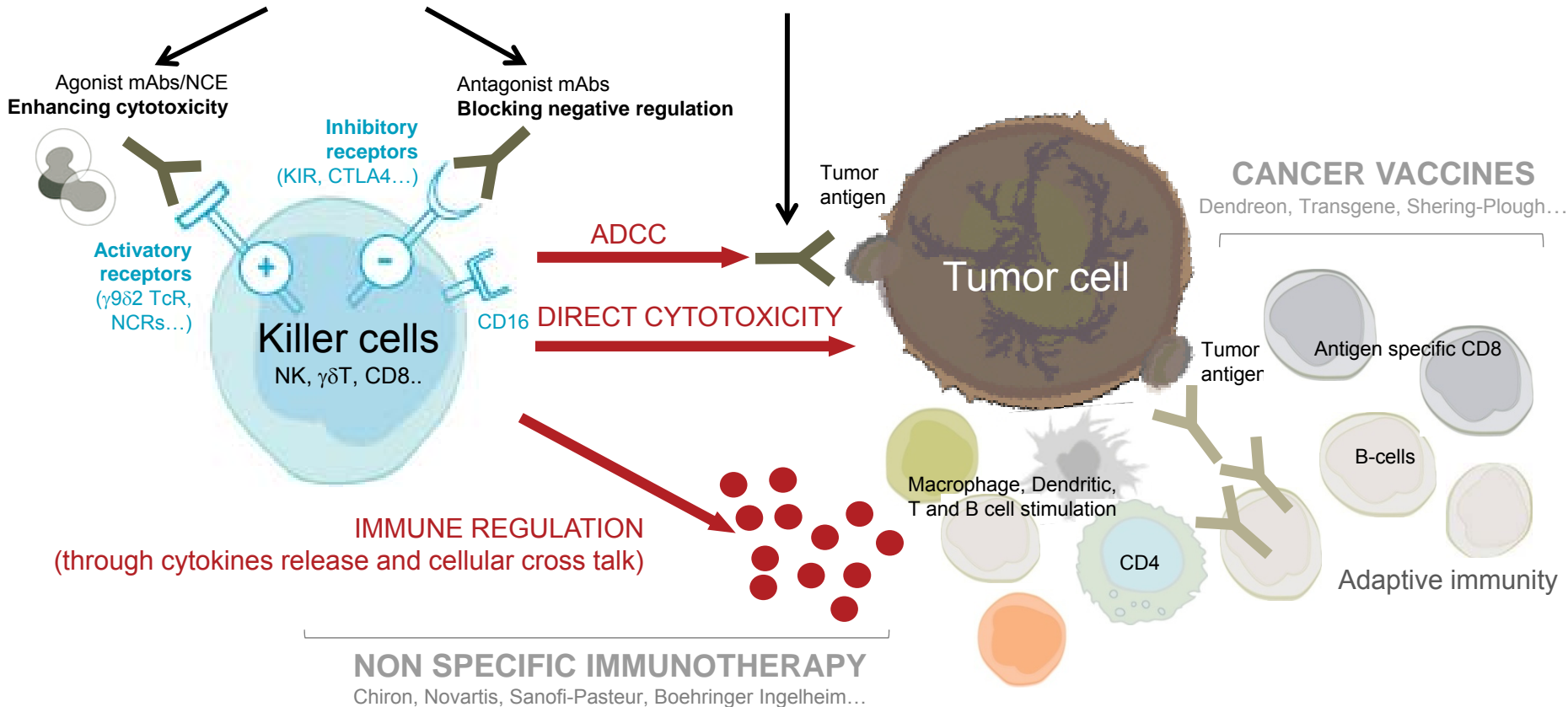
IPH 1101, IPH 2101

Proxy: anti-CTLA4 in Phase III (BMS, Pfizer)

Tumor Targeting with cytotoxic mAbs

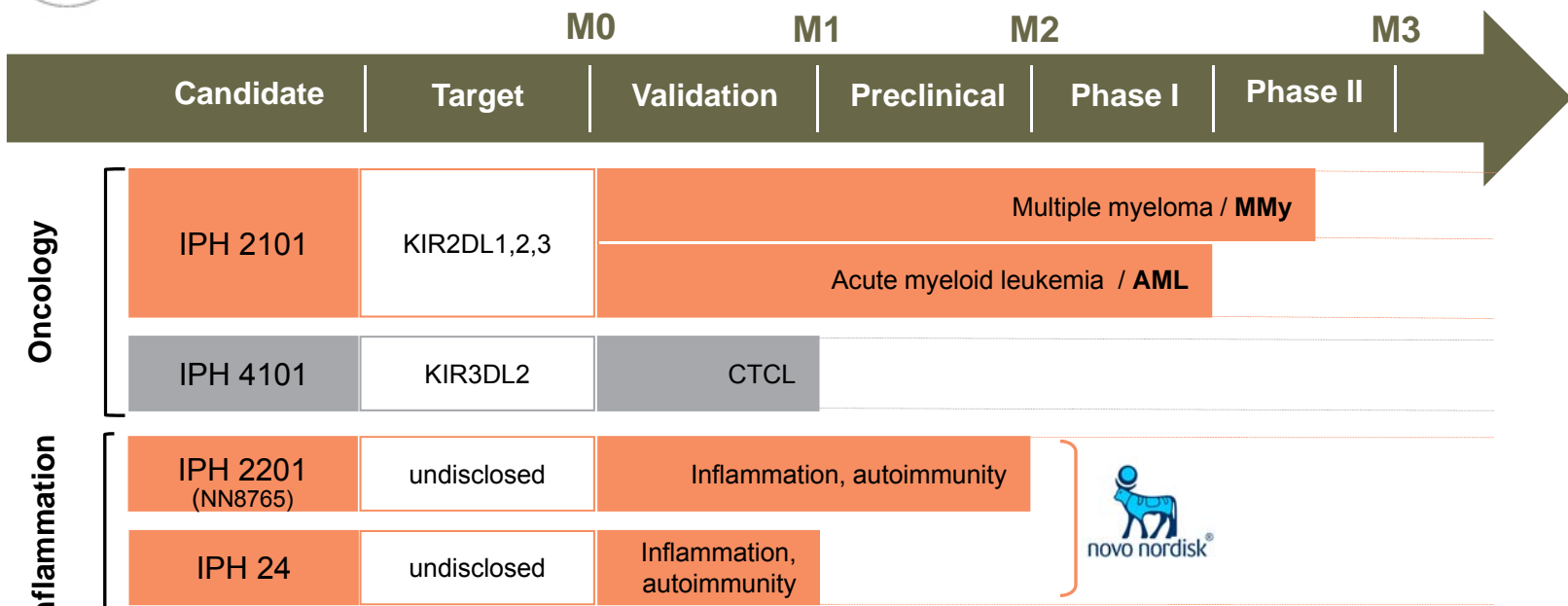
IPH 4101

Proxy: Rituxan® (Roche/Genentech)



* Same targets might be relevant for cancer (agonist) and inflammation (antagonist)

- **Specialization of upstream R&D in antibodies:**
 - Consolidation of in-house antibody capabilities and streamlining of research organization
 - MAbs have a higher probability of success (25% vs 11% for NCE, *source: Datamonitor*)
 - Long in vivo half-life, low risk of off-target toxicity and good tolerance
 - Well established regulatory pathway for pharmaceutical development
 - Suitable for oncology, inflammation, chronic diseases
 - Commoditized technologies
 - Access to a universe of novel targets
- **Target scouting in cancer and inflammation**
 - Leverage on Inserm Transfert partnership as well as other scientific networks
- **No further investment of internal resources in RNA targeting TLR**
 - Strong pharmacology packages for IPH 3102 (TLR3, cancer and vaccine adjuvantation) and IPH 3201 (TLR7/8, vaccine adjuvantation)



■ **IPH 4101 (cutaneous lymphomas):**

- Currently in pre-clinical validation
- Technology agreement with Vivalis and Oséo support announced in early 2009; successful collaboration with first milestone achieved late 2009

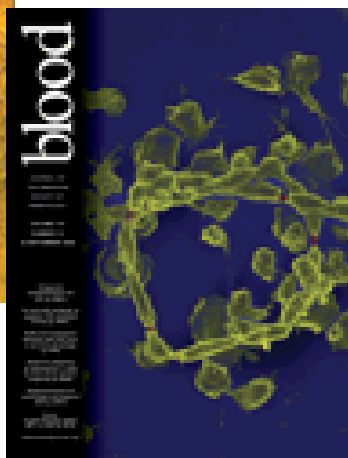
■ **IPH 4201 (pancreas):**

- Detection of unexpected cross-reactivity
- Program terminated early 2010

R&D update

Strong publication level in top-tiers peer review

- >10 publications in 2009/2010 up to date
 - o.w. 4 in Blood, 1 in PNAS, 1 in Cancer Research
- Reflecting top-level scientific work, maturation of research fields as well as development advances





FINANCIAL YEAR 2009





- €24.3m fund raising closed in December 2009
 - FSI as main investor, Novo Nordisk A/S and Alta Partners also participated
 - 10.7 million new shares, 36.6 million shares outstanding
- Operational start of Platine, a joint immuno-monitoring platform
 - In partnership with Transgene, ImmunID, Inserm and Centre Léon-Bérard
 - Leverage on expertise of each partner to (i) save costs and (ii) later deliver services to third parties

Key 2009 accounting figures

IFRS financial statements for 2009

In thousand of euros	2008 Restated	2009
Licensing revenue	7,364	3,243
Others and government funding for research costs	5,560	4,472
Operating revenue	12,924	7,716
Research and development	(20,897)	(18,032)
General and administrative	(5,043)	(5,219)
Net operating expenses	(25,940)	(23,251)
Operating income (loss)	(13,016)	(15,535)
Interest income/(expenses), net	1,154	910
Net loss	(11,862)	(14,626)
Shares outstanding (in thousands) – average	25,665	26,299
Net loss per share	(0.46)	(0.56)
Cash, cash equivalents and financial instruments	33,832	49,194
Total assets	57,288	64,219
Net book value	37,767	47,122
Total financial debt	8,442	8,277



Key 2009 accounting facts

Significant strengthening of the balance sheet

- **Increase in operating loss (€15.5m in 2009 vs. €13.1m in 2008)** in the context of a decrease in operating revenue, following the contractual end of the NK collaboration with Novo Nordisk A/S
- **Decrease in operating expenses (€23.3m in 2009 vs. €25.9m in 2008)** mostly attributable to the accounting for €2.5m in purchase of materials in 2008 following the grant back of the IPH 2101
- **Positive cash flow in the period thanks to:**
 - (i) early CIR refund amounting €10.4m, and
 - (ii) €23.1m in net proceeds of the PIPE deal done in December
- **IPH ended up 2009 with €49.2m in cash**, enough to go into 2012 based on the current business plan

Financial statements available on March 5, 2010
(www.innate-pharma.com)
and annual report available in 2Q 2009

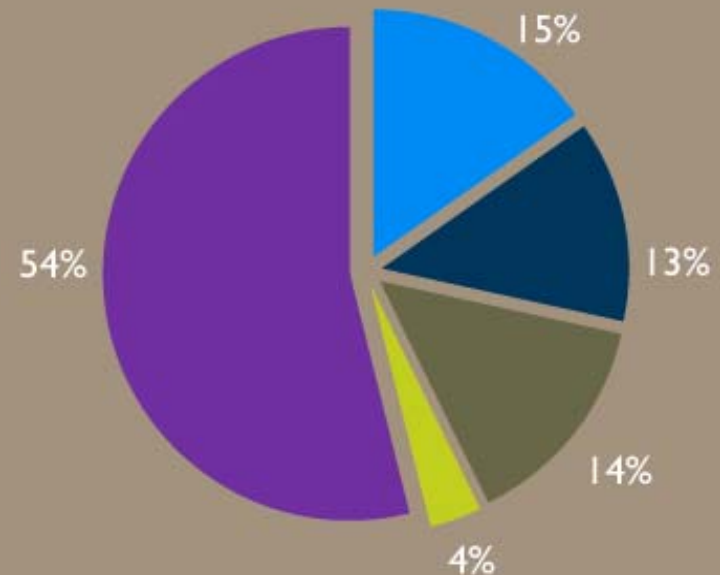


Share information and shareholders

- First day of trading: November 1, 2006
- Number total of shares as at December 31, 2009: 36,636,794
- Shareholders as at December 31, 2009:

New shareholder's structure

- Novo Nordisk A/S
- FSI (French sovereign fund)
- Other investors members seating on the Supervisory Board
- Management
- Free Float



- **A clear positioning:**
 - > First-in-class drug candidates
 - > Specialization in immunology
 - > Therapeutic focus on cancer with development capabilities, inflammation for early licensing
 - > Technologic focus on antibody
- **A key immuno-pharmacology expertise to address significant opportunities**
 - > Cancer immunotherapy could yield breakthrough in cancer treatment (ipilimumab, therapeutic vaccines such as MAGE-3 or TG 4010)
 - > Tumor antigen targeting is a validated pathway (>\$10Bn market), still at the beginning of its expansion
- **Track record of 2 Phase II drugs, including one with proof-of-concept data**
- **Key clinical and corporate news-flow in the 2010-2013 period**
- **Strong cash position to achieve objectives**



innate pharma
the innate immunity company

Appendix

Multiple Myeloma

2nd hematological cancer, still incurable

Description of the disease:

- Monoclonal proliferation of plasma cells (differentiated B-cells producing Ig*)
- Their excessive count is usually associated to an over production of monoclonal Ig (M-protein)

Symptoms of MMy:

- Bone marrow infiltration, M-Protein, renal failure, immunodeficiency
- Poor quality of life: recurring infections, bone pains, fractures, medullar compression

Natural history:

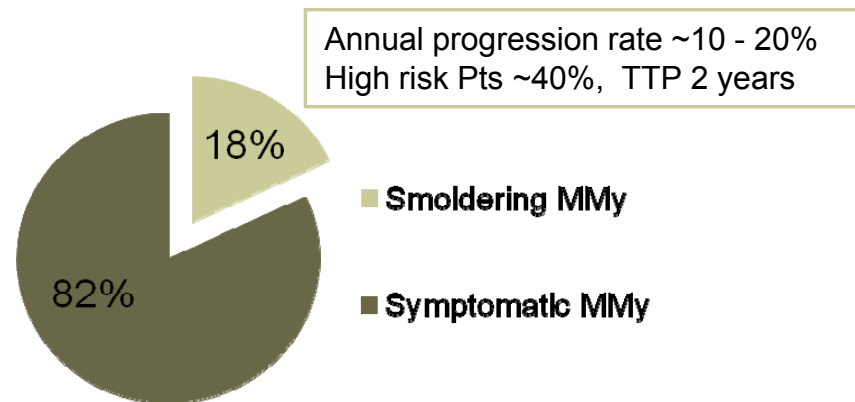
- Smoldering myeloma evolving into symptomatic myeloma – Smoldering myeloma is under-diagnosed
- OS < 2 years (< 1y if renal failure)

Population:

- Incidence (G7): 40,900 patients/year
- Mortality (G7): 27,400 patients/year
- Median age at diagnosis: 65 -70y

Patients <65 yrs:
5-yr survival 20-30%

Patients >65 yrs:
5-yr survival 5-15%

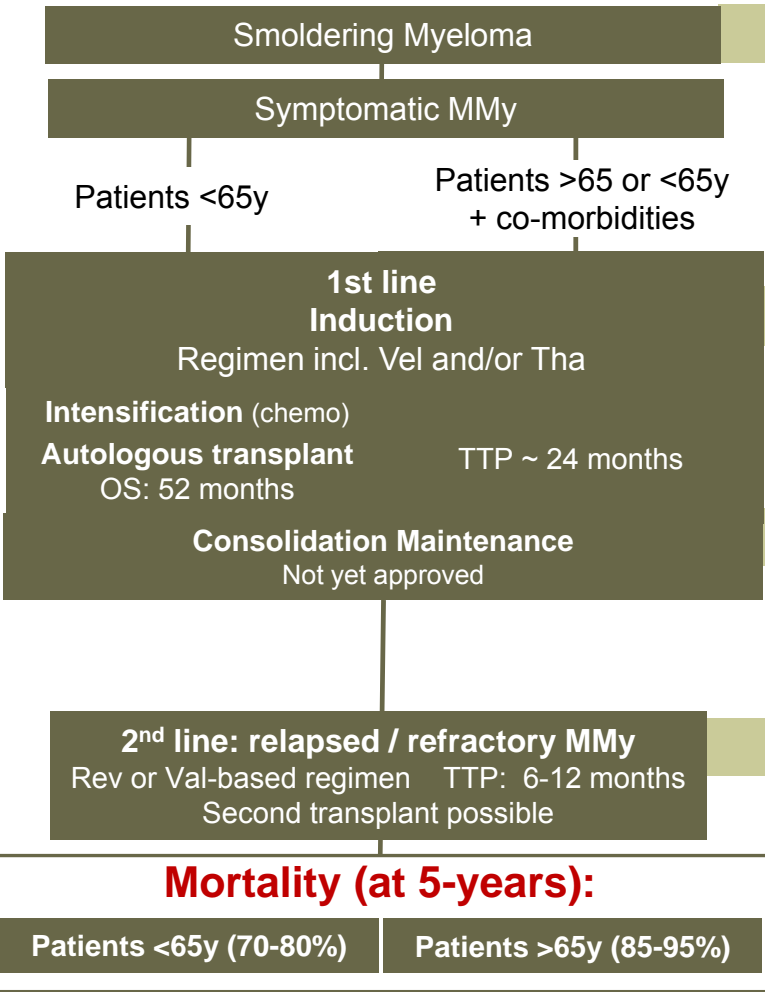


*Ig: immunoglobulins

Multiple Myeloma

IPH 2101 positioning

Treatment paradigm



Persistent medical needs

Likely treatment at 3-5 years

Prevention of progression into symptomatic MMy

Positive interim data with Rev Dex

IPH 2101-203

Increase efficacy for elderly patients

Induction including Rev / Vel (Ph III) For p>60y, TTP remains <20 months

MRD eradication / increase of TTP, good tolerance

Consolidation including Rev / Vel (Ph III)
Rev / Thal: 1/4 MRD eradication

IPH 2101-201

Treatment of relapse

Rev or Vel-based regimen
Efficacy likely to remain suboptimal

**IPH 2101-202
Collaboration with Celgene**

Drugs with **lower toxicity** and regimen that **minimize side effects** for elderly patients

Rev: lenalidomide (Revlimid[®], Celgene)
Thal: thalidomide (Thalomid[®], Celgene)
Vel: bortezomib (Velcade[®], Millenium)



Perspective in Multiple Myeloma

Emerging drugs unlikely to cure disease in the coming years

- There are about 50 drugs in development in MMy, most of them in early development stage (Phase I and II)
- New generations of –imids and proteasome inhibitors in development are not expected to cure the disease
- IPH 2101's new MOA:
 - > may shrink residual disease resistant to cytotoxic agents or targeted therapies, by restoring immune surveillance
 - > expected to be well tolerated, key consideration notably for elderly patients (not suitable for intensification)

Potential market: ~€800m* (newly approved treatments: >\$3Bn in cumulative sales)

Acute Myeloid Leukemia

Strong medical need for the most common type of adult leukemia

Description of the disease and natural history:

- Rapid growth and accumulation of abnormal and immature myeloblasts in the bone marrow; very heterogeneous disease
- Median age at diagnosis: 65 years
- Overall survival: ~20%

Population:

- Incidence (US): 13,000 cases
- Mortality (US): 9,000 patients

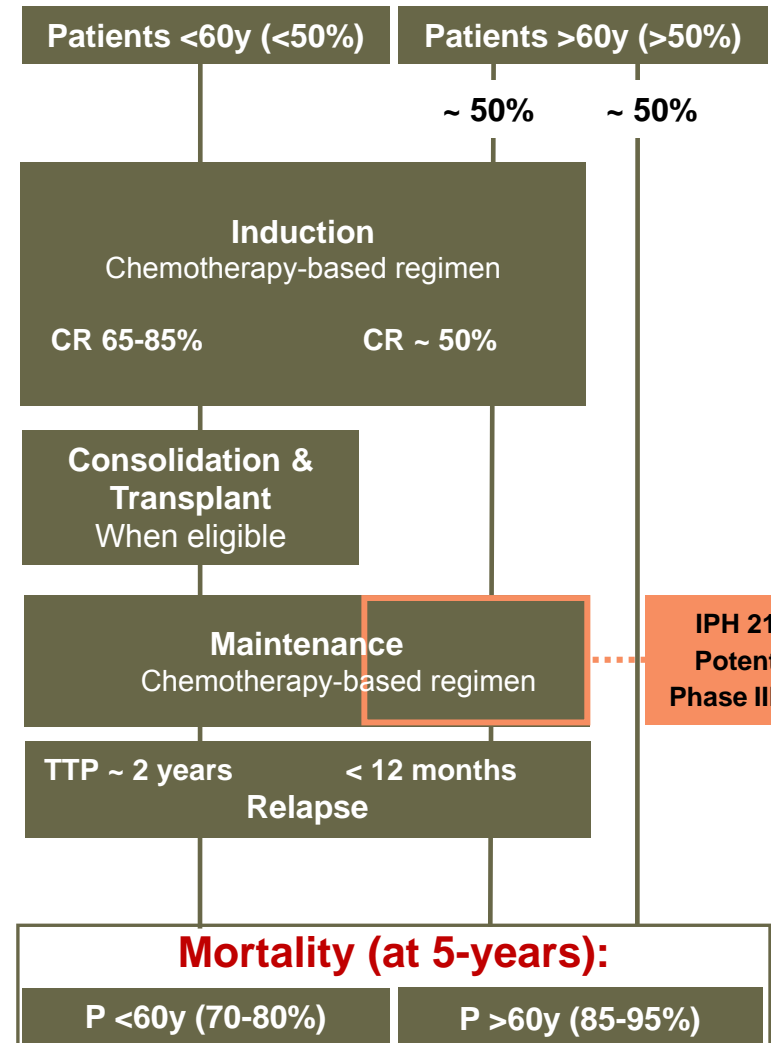
Patients <60 yrs:
5-yr survival 20-30%

Patients >60 yrs:
5-yr survival 5-15%

Significant unmet medical needs:

- No drug currently in development anticipated to have a large impact on patient outcomes
- Need for:
 - >Curative therapy beside transplant
 - >Well tolerated drugs to expand remission as well as for elderly patients
 - >Novel therapies for relapsed / refractory disease
 - >Non-cytotoxic drugs to overcome multidrug resistance

Treatment paradigm*





Perspective in Acute Myeloid Leukemia

Intensive development effort but no revolution expected soon

- Very high unmet medical need, with no major progress for the last 10 years
- A number of drugs is currently in late-stage development, but none of them is anticipated to have a large impact on patient outcomes
- IPH 2101's new MOA:
 - > may shrink residual disease resistant to cytotoxic agents, by stimulating NK mediated immunity as documented in AML
 - > expected to be well tolerated, key consideration notably for elderly patients (not suitable for intensification)

Potential market for post-remission setting: ~€200m*

Source: Datamonitor, *IPH estimates

Type C viral Hepatitis

A significant healthcare concern

Description of the disease:

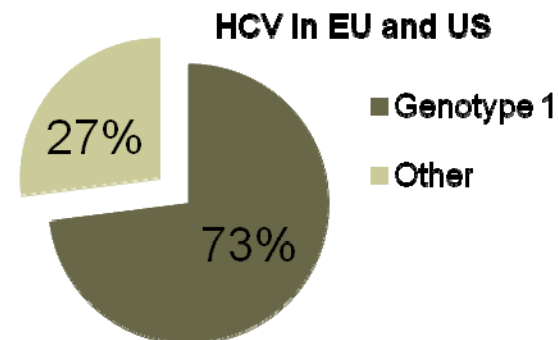
- Chronic infection (RNA virus).
- At the origin of a liver disease (silent destruction of infected hepatocytes and fibrosis), and frequently extra-hepatic manifestations (arthralgia, myalgia, pruritus, fatigue).
- Risk of developing liver cirrhosis and hepatocarcinoma (1-4% per year ; median time between infection and cirrhosis: 30 years with much variability).
- At least 250 000 deaths are attributable yearly and worldwide to HCV infection. An estimated 27% of cirrhosis and 25% of hepatocarcinoma (HCC) occur in HCV infected people.

Population:

- Incidence (WW): 3-4 million patients
- Prevalence (WW): 170 million patients

Segmentation (6 genotypes):

- > Genotype 1 is the most common in EU and US and poorly sensitive to treatment.
- > Genotype 4 to 6 are rare in the EU and US but frequent elsewhere and also poorly sensitive to treatment.
- > Genotype 2 and 3 are sensitive to treatment with 70 to 80% response to SOC.



(Blatt, 2000)



Type C viral Hepatitis

IPH 1101 positioning (genotype 1 HCV)

Treatment paradigm

Genotype 1 HCV Patients

1st line
Peg-IFN α + ribavirine, 48 wks
(Standard of Care - SOC)

SVR* 40~50%

Viral response during treatment

~45%

~15-20%

~35-40%

Relapsers
(SOC 2nd line:
SVR* ~25%)

Non and slow responders

SVR

Risk of cirrhosis and hepatocarcinoma
~2-3% mortality/year

Persistent medical needs

Likely treatment at 3-5 years

Better efficacy, better tolerance and compliance, acceleration of viral load decrease

Protease inhibitors combo with SOC
Exp. SVR ~65%
Other targeted therapies in combo with SOC

Novel therapies for relapsers and non/slow responders

Protease inhibitors and other targeted therapies in combo with SOC

Combination with SOC in non and slow responders

New drugs to overcome resistance associated with virus mutation

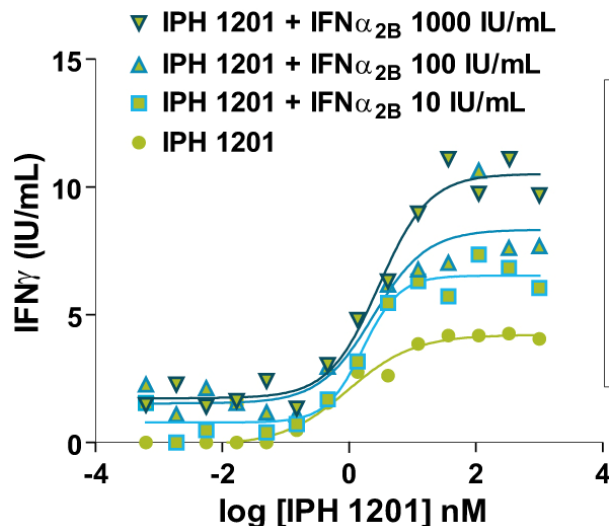
*SVR: Sustained viral response (undetectable viral RNA at 6 months after end of treatment) considered as cure

Perspective in Type C viral Hepatitis

Progresses with the emergence of the protease inhibitors

- New class of drug in late Phase III trial (protease inhibitors) in combination settings
- IFN α and ribavirin will remain the backbone of future treatment at all stages
- Evidence of antiviral activity with IPH 1101, very good tolerance
- IPH 1101 could synergize with current and future SOC notably by enhancing efficacy of IFN α and exerting both an anti viral and immunomodulatory effect
- IPH 1101 may provide an option to overcome virus mutations leading to resistance to targeted therapies (such as protease inhibitors)

Market expected to increase from \$2.3Bn in 2007 to \$4.6Bn in 2017





Follicular Lymphoma

Most frequent hematologic malignancy

Description of the disease:

- Proliferation of lymphocytes (B or T) in the lymphatic system (usually in lymph nodes)
- Heterogeneous disease

Segmentation of NHL:

- B-cells lymphomas: 78% of NHL
 - > Follicular lymphoma (fNHL): 22%
- T-cells lymphomas: 8%
- Other subtypes: 14%

Population:

- Incidence (G7): 122,000 patients
- Mortality (G7): 47,300 patients
- Median age at diagnosis: ~60 years

Prognosis in follicular lymphoma:

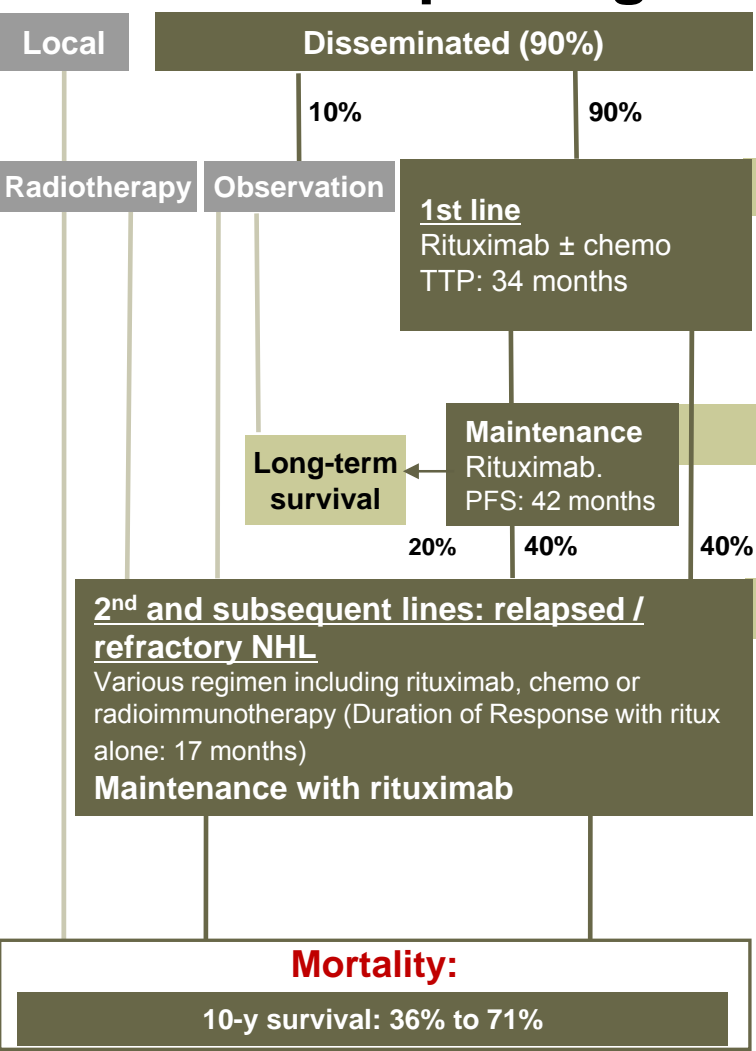
- Follicular lymphoma is indolent (slow evolution) but poorly curable
- 10-y survival: 36% to 71% depending on prognosis factor



Follicular Lymphoma

IPH 1101 positioning

Treatment paradigm



Persistent medical needs

Likely treatment at 3-5 years

IPH 1101/1201

Curative therapy, prolongation of remission

Third generation anti-CD20 mAbs in combo with chemotherapy

First line low-tumor burden in combo with anti-CD20 mAb

Curative therapy, prolongation of remission

Rituximab (PRIMA trial)

Better response and tolerance to replace chemo

Third generation anti-CD20 mAbs in combo with chemotherapy

Relapse low-tumor burden in combo with anti-CD20 mAb

Drugs with **lower toxicity to replace chemo** in combination with mAb and **more efficient treatment** to achieve cure in younger patients

IPH 1101 is administered in combination with low-dose IL-2 in this setting



Perspective in Follicular Lymphoma

Rituximab to remain key in the treatment

- Multiple agents in late development stage reflecting rituximab commercial success and persistent need to achieve sustained remission
- Rituximab status as market leader is unlikely to be challenged within the next 5-10 years
- Encouraging interim data with 32% complete response in the first set of 34 patients assessed by the independent central review (reference paper Davis et al. (*JCO*, 2000): 11% CRR expected in this population)
- Satisfactory and manageable overall tolerance and safety
- Enhancement of the effector function of $\gamma\delta$ T cells in combination with a cytotoxic antibody could have a significant clinical impact in the disease treatment

Potential market: ~€400m*



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