R&D Update 2014

Onno van de Stolpe, CEO
Piet Wigerinck, CSO

New York City, 17 June 2014
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R&D Update

- Company strategy  Onno van de Stolpe, CEO

- R&D strategy & portfolio  Piet Wigerinck, CSO

- Outlook  Onno van de Stolpe, CEO
Galapagos at a glance

- Founded in 1999 as joint venture of Crucell and Tibotec
  - 400 staff, research sites in 4 countries with HQ in Belgium
- Focus on novel mode of action medicines
  - proprietary target discovery platform, broad pipeline:
    - three Phase 2 drugs (one owned by GSK), one Phase 1 drug
    - six pre-clinical candidates, >20 programs in discovery
- Major alliances with AbbVie, JnJ, GSK, Servier
- Market cap ~ $650 M
How we built Galapagos – the business

1999
- Founded by Tibotec and Crucell
- VC financing

2002
- Acquired BioFocus

2005
- IPO on Euronext
- 1st pharma alliance

2008
- 1st profitable year
- Acquired Argenta and GSK Zagreb

2011
- AbbVie deal ‘634

2014
- Sold services to CRL
- AbbVie deal CF
How we built Galapagos – the science

Target


Identified 1st novel target
How we built Galapagos – the science

- Target
- Hit-to-lead
- Lead optimization
- Pre-clinical

1999
- Identified 1st novel target

2002

2005
- Nominated 1st pre-clinical candidate

2008

2011

2014
How we built Galapagos – the science

- Identified 1st novel target
- Hit-to-lead optimization
- Lead optimization
- Pre-clinical
- Phase I
- Entered the clinic for 1st time
- Nominated 1st pre-clinical candidate

How we built Galapagos – the science

1999: Identified 1st novel target
2002: Hit-to-lead optimization
2005: Nominated 1st pre-clinical candidate
2008: Entered the clinic for 1st time
2011: Started 1st Phase 2 study
2014: 1st positive Phase 2 PoC
## Our pharma alliances over time

<table>
<thead>
<tr>
<th>Indication</th>
<th>Partner</th>
<th>Signed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inflammation</td>
<td>gsk</td>
<td>2006</td>
</tr>
<tr>
<td>Inflammation</td>
<td>JANSSEN</td>
<td>2007</td>
</tr>
<tr>
<td>Anti-infectives</td>
<td>gsk</td>
<td>2007</td>
</tr>
<tr>
<td>Osteoporosis</td>
<td>Lilly</td>
<td>2007</td>
</tr>
<tr>
<td>Metabolic</td>
<td>MERCK</td>
<td>2009</td>
</tr>
<tr>
<td>Inflammation</td>
<td>MERCK</td>
<td>2009</td>
</tr>
<tr>
<td>Fibrosis</td>
<td>Roche</td>
<td>2010</td>
</tr>
<tr>
<td>Osteoarthritis</td>
<td>SERVIER</td>
<td>2010</td>
</tr>
<tr>
<td>Oncology</td>
<td>SERVIER</td>
<td>2011</td>
</tr>
<tr>
<td>Autoimmune</td>
<td>abbvie</td>
<td>2012</td>
</tr>
<tr>
<td>Cystic fibrosis</td>
<td>abbvie</td>
<td>2013</td>
</tr>
</tbody>
</table>

- Alliances have brought in ~ $465 M in cash since 2006
- Source of promising molecules and targets for GLPG
Sale of services to Charles River Labs

• Sales price: $178 M in cash + $7 M earnout after 1 year

• Charles River acquired:
  - all operations of BioFocus & Argenta in the UK
  - BioFocus activities in Leiden

• Galapagos continues outsourcing to BioFocus/Argenta over next 3 years
  - $12 million in total

• Deal closed on 1 April 2014
Strengthening the balance sheet

<table>
<thead>
<tr>
<th>Year</th>
<th>Cash</th>
<th>CIR Receivables</th>
</tr>
</thead>
<tbody>
<tr>
<td>2010</td>
<td>56</td>
<td>13</td>
</tr>
<tr>
<td>2011</td>
<td>46</td>
<td>24</td>
</tr>
<tr>
<td>2012</td>
<td>132</td>
<td>35</td>
</tr>
<tr>
<td>2013</td>
<td>197</td>
<td>46</td>
</tr>
<tr>
<td>2014E</td>
<td>236</td>
<td>46</td>
</tr>
</tbody>
</table>

in $ millions on 31 December
Growth strategy

- Execute development of JAK1 Phase 2 programs in RA & Crohn’s disease
- Build mature clinical portfolio
- Take programs in R&D focus areas further on our own
- Continue pharma alliances
- Sign new alliances and partnerships

Strong balance sheet for unlocking shareholder value in the pipeline
R&D Update

• Company strategy  Onno van de Stolpe, CEO

• R&D strategy & portfolio  Piet Wigerinck, CSO

• Outlook  Onno van de Stolpe, CEO
A novel mode of action company

- Pioneering at every step
- Aim for disease-modifying drugs
- Novelty affords long market exclusivity
- Portfolio higher risk with higher potential
- Our approach validated in clinic & by deals

Our aim: novel targets, better molecules
Human primary cell expertise
Single cells & co-cultures

<table>
<thead>
<tr>
<th>Human Cell Type</th>
<th>Human Cell Type</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adipocytes</td>
<td>Hepatocytes</td>
</tr>
<tr>
<td>Astrocytes$^1$</td>
<td>Keratinocytes</td>
</tr>
<tr>
<td>Basophils</td>
<td>Macrophages</td>
</tr>
<tr>
<td>Beta cells</td>
<td>Mast cells</td>
</tr>
<tr>
<td>Bronchial epithelial cells</td>
<td>Mesenchymal stem cells</td>
</tr>
<tr>
<td>Cardiac fibroblasts</td>
<td>Motor neurons$^1$</td>
</tr>
<tr>
<td>Cardiomyocytes$^1$</td>
<td>Osteoblasts</td>
</tr>
<tr>
<td>Chondrocytes</td>
<td>Skeletal muscle</td>
</tr>
<tr>
<td>Dendritic cells</td>
<td>Striatal neurons$^1$</td>
</tr>
<tr>
<td>Embryonic stem cells</td>
<td>Synoviocytes</td>
</tr>
<tr>
<td>Endothelial cells</td>
<td>T-cells</td>
</tr>
<tr>
<td>Fibroblasts$^1$</td>
<td>1) cell line</td>
</tr>
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</table>

$^1$) cell line
Disease model expertise

<table>
<thead>
<tr>
<th>Disease</th>
<th>Model Expertise</th>
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<tbody>
<tr>
<td>ALS</td>
<td>Immunity</td>
</tr>
<tr>
<td>Alzheimer’s</td>
<td>Inflammation</td>
</tr>
<tr>
<td>Asthma / Allergy</td>
<td>Oncology</td>
</tr>
<tr>
<td>Atherosclerosis</td>
<td>Osteoarthritis</td>
</tr>
<tr>
<td>Cardiovascular</td>
<td>Osteoporosis</td>
</tr>
<tr>
<td>COPD</td>
<td>Parkinson’s</td>
</tr>
<tr>
<td>Cystic Fibrosis</td>
<td>Psoriasis</td>
</tr>
<tr>
<td>Diabetes / Obesity</td>
<td>Renal failure</td>
</tr>
<tr>
<td>Duchenne / SMA</td>
<td>Rheumatoid Arthritis</td>
</tr>
<tr>
<td>Fibrosis</td>
<td>Viral infections</td>
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<tr>
<td>Huntington’s</td>
<td></td>
</tr>
</tbody>
</table>
‘634 validates our approach
8 years from assay development to Ph2 PoC data

2003
Target discovery

2005
Hit finding

2007
JAK1 selected

2009
Assay development

2011
Start Phase 1

2013
End PoC

2014
Start Phase 2B in RA

PCC nomination

Start Phase 2 PoC

Start Phase 2 in Crohn’s

Galapagos most advanced in JAK1 space today
R&D strategy

Pharma: focus on marketing & sales, seeking innovation for pipelines

Galapagos discovers disease-modifying novel modes-of-action:

- select disease areas with unmet medical need
- develop assays to identify novel targets to address these diseases
- discover & develop selective molecules directed toward novel targets
- partner at optimal stage, ring fence some proprietary programs
## Galapagos R&D focus areas

<table>
<thead>
<tr>
<th>Focus area</th>
<th>Rationale</th>
<th>Stage most advanced program</th>
</tr>
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<tbody>
<tr>
<td>Inflammation</td>
<td>• historical focus</td>
<td>JAK1: Phase 2b</td>
</tr>
<tr>
<td></td>
<td>• built strong expertise</td>
<td></td>
</tr>
<tr>
<td>Orphan diseases</td>
<td>• unique assay toolkits</td>
<td>CF potentiator: candidate</td>
</tr>
<tr>
<td></td>
<td>• built strong expertise</td>
<td></td>
</tr>
<tr>
<td>Anti-infectives</td>
<td>• discovered unique platform</td>
<td>DnaE: candidate drug</td>
</tr>
<tr>
<td>Fibrosis</td>
<td>• novel modes of action can make a difference</td>
<td>lead compounds</td>
</tr>
<tr>
<td></td>
<td>• unique assay toolkits</td>
<td></td>
</tr>
</tbody>
</table>
Steady flow of Phase 2 readouts

2014

GSK2586184 – psoriasis
Phase 2 topline results

‘974 - UC
Phase 2 topline results

2015

‘634 – RA
Darwin 1 &2 topline results

2016

‘634 – Crohn’s
Phase 2

‘1205 – IBD - JnJ
Phase 2 topline
R&D highlights

- '634
- '974
- Cystic Fibrosis
- GSK2856184
- Anti-infectives
- '1790
- R&D overview
‘634: the most selective JAK1 inhibitor

- Novel mode of action for autoimmune
- Confirmed safety & efficacy in 2 short-term studies in RA
- Oral treatment with opportunity for once-daily dosing
Selectivity of JAK inhibitors
GLPG in-house data

Ratio JAK1/JAK2 in human whole blood assay

- baricitinib
- ruxolitinib (Jakafi™)
- ABT-494 series
- decernotinib (VX-509)
- tofacitinib (Xeljanz™)
- INCB039110
- 634
'634 gives continuous target inhibition
Unique PD profile in JAK field – PD modelling data

% pSTAT1 inhibition

Hours post dose

Tofacitinib - 5 mg bid
GLPG0634 - 200 mg qd
'634 phase 2b program in RA
Moderate to severe RA with inadequate MTX response

- **Darwin**
  - **Add-on to MTX**: 595 patients
  - **Monotherapy**: 280 patients
  - **Long term extension**
‘634 global DARWIN program

3 studies, 23 countries and > 200 sites
## Status overview

<table>
<thead>
<tr>
<th>Study</th>
<th># of countries</th>
<th>average # sites per country</th>
<th>% sites open</th>
</tr>
</thead>
<tbody>
<tr>
<td>Darwin 1</td>
<td>22</td>
<td>6.7</td>
<td>93%</td>
</tr>
<tr>
<td>Darwin 2</td>
<td>19</td>
<td>4.6</td>
<td>77%</td>
</tr>
</tbody>
</table>

- Longer than expected approval cycles
Planning ‘634 in RA

### Previous

- **2014**: Last patient in Darwin 1
- **2015**: Topline 12 wk Darwin 1 & 2
- **2016**: Licensing decision AbbVie
- **2017**: Possible start Ph3

### Updated

- **2014**: Last patient in Darwin 1
- **2015**: Topline 12 wk Darwin 2
- **2016**: Licensing decision AbbVie
- **2017**: Possible start Ph3
JAK1 inhibition improves inflammation in GI DSS mouse inflammation model

**STAT activation**

- H2O intact
- 4% DSS
- DSS + '634

**GLPG0634 (ng/ml)**

- JAK2 IC50
- JAK1 IC50
- Plasma [ '634 ]

**Time (hours)**

- 30 mg/kg dose

**Strong pre-clinical data for '634 in GI inflammation**
Phase 2 study in Crohn’s

- 180 patients with CDAI score between 220 – 450
- Two-part study: 10 week induction & 10 week (early) maintenance
- Primary endpoint at week 10: CDAI <150
- Data on both induction & maintenance enables fast move into Phase 3

<table>
<thead>
<tr>
<th>Placebo (n=45)</th>
<th>100 mg QD</th>
<th>Placebo</th>
<th>100 mg QD</th>
<th>200 mg QD</th>
</tr>
</thead>
<tbody>
<tr>
<td>'634 200 mg QD (n=135)</td>
<td>Placebo</td>
<td>Placebo</td>
<td>Placebo</td>
<td>Placebo</td>
</tr>
</tbody>
</table>

Time (weeks)
Phase 2 study with ‘634 in Crohn’s

9 countries in Europe
Timelines ‘634 in Crohn’s

- 2014: Last patient in
- 2015: Topline 10 weeks
- 2016: Licensing decision AbbVie
- 2017: Topline 20 weeks

Crohn’s topline to be delivered in Q2 ‘15
R&D highlights

- '634
- '974
- Cystic Fibrosis
- GSK2856184
- Anti-infectives
- '1790
- R&D overview
Ulcerative colitis
Neutrophil infiltration in gut wall

Neutrophilic inflammation
'974: novel mode of action in UC

Short Chain Fatty Acids

- FFA2
  - Pro-inflammatory
    - Neutrophil migration
    - Neutrophil activation
    - IL-8 release
- Other
  - Anti-inflammatory
    - Inhibition of release of:
      - cytokines
      - chemokines
      - prostaglandins
Phase 1: multiple ascending dose

Dose selection for Proof-of-Concept study

- PK/PD: plasma levels vs. inhibition of acetate-induced CD11b[AE]
- Maximal PD ($E_{\text{max}}$) achieved at plasma levels > 500 ng/ml
- At 200 mg BID, plasma exposure exceeds 500 ng/mL for 24 h per day
The bar in UC is set high
Pfizer IgG2 antibody, PhIb trial results in UC

<table>
<thead>
<tr>
<th></th>
<th>Placebo</th>
<th>PF-00547,659</th>
</tr>
</thead>
<tbody>
<tr>
<td>Week 4</td>
<td>n=19</td>
<td>n=60 (all groups)</td>
</tr>
<tr>
<td>Clinical Response %</td>
<td>32</td>
<td>52</td>
</tr>
<tr>
<td>Clinical Remission %</td>
<td>11</td>
<td>13</td>
</tr>
<tr>
<td>Mucosal healing %</td>
<td>16</td>
<td>37</td>
</tr>
<tr>
<td>Fecal calprotectin; % change</td>
<td>-18</td>
<td>-63</td>
</tr>
</tbody>
</table>

*Source: Vermeire, Gut, 2011*
‘974: Proof-of-Concept study

- Randomized, double-blind, placebo-controlled study
  - 4 countries, 16 sites
- 45 patients with mild to moderate UC with active disease
  - 4 weeks treatment: oral dosing
  - 200 mg BID ‘974 or placebo (2:1)
  - safety parameters & pharmacokinetics
  - efficacy parameters: fecal calprotectin, neutrophils in biopsies (MPO), (partial) Mayo score
- Independent, unblinded safety monitoring throughout the study
<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Placebo n = 15</th>
<th>'974 200 mg BID n = 30</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>39.1</td>
<td>40.2</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>25.1</td>
<td>26</td>
</tr>
<tr>
<td>Ethnicity (Caucasian)</td>
<td>15</td>
<td>30</td>
</tr>
<tr>
<td>Sex (F/M)</td>
<td>5 / 10</td>
<td>8 / 22</td>
</tr>
<tr>
<td>Duration of UC (y)</td>
<td>9.4</td>
<td>10.1</td>
</tr>
<tr>
<td>Acute exacerbations last 12 m</td>
<td>0.9</td>
<td>1.1</td>
</tr>
<tr>
<td>Baseline FC (mg/kg) mean</td>
<td>742</td>
<td>1,594</td>
</tr>
<tr>
<td>Baseline MPO (/Field) mean</td>
<td>67</td>
<td>70</td>
</tr>
<tr>
<td>Baseline CRP (mg/L)</td>
<td>11.1</td>
<td>8.3</td>
</tr>
</tbody>
</table>
Safety findings

- 2 “treatment related” AEs reported
  - placebo: worsening of colitis, leading to treatment discontinuation
  - ‘974: nausea, vomiting & dyspepsia leading to treatment discontinuation

- Low frequency of common infections: 3 on placebo, 1 on ‘974

- Glucose: unaffected

- Hematology: unaffected

‘974 was well tolerated & safe
Evaluation of read-outs

- MPO in lamina propria
- Fecal calprotectin
- CRP
- Partial Mayo score
- Mayo score

MoA

Clinical efficacy
MPO in biopsies
Absolute & categorical shifts

Mean % MPO positive cells

Categorical shifts of patients

'974 shows MPO biomarker effect in UC patients
Fecal calprotectin
Changes & shifts from baseline

Mean change from baseline

Categorical shifts of patients

‘974 shows fecal calprotectin biomarker effect in UC patients
Mayo score

Mean Mayo score

Categorical shifts of patients

Placebo

'974 shows no clinical effect in UC patients
Summary & conclusions
‘974 Proof-of-Concept study

- Efficient Proof-of-Concept study with all appropriate endpoints
- ‘974 was safe & well tolerated in UC patients
- Trend shown for reduction in MPO in colon biopsies & fecal calprotectin
  - related to the novel mode-of-action: neutrophil migration
- Reduction of neutrophil influx did not induce improvement of clinical signs and symptoms within 4 weeks

Some evidence for mode-of-action, no competitive clinical effect
Next steps with ‘974

- Perform subgroup analyses
- Consider other indications
- Discuss further development with partners
- Present PoC data at a medical conference
R&D highlights

- '634
- '974
- Cystic Fibrosis
- GSK2856184
- Anti-infectives
- '1790
- R&D overview
Deal structure in cystic fibrosis (CF)

- Both companies contribute funding & science
- AbbVie commercializes
  - GLPG retains China/South-Korea, co-promotion rights in Benelux
- Upfront payment $45 M
  - plus an additional $360 M in future milestones + double digit royalties
About CF

- Fatal inherited disease of the lungs & digestive system
- Debilitating disease, impairs quality of life
- Life expectancy: 37 years
- 70,000 patients worldwide, 30,000 in the US
- Patients carry a defective gene/protein (CFTR)
  - CFTR channel transports chloride across cell membrane
  - 1900 mutations identified grouped into 5 different classes
Most CF patients are Class II (F508del)
We target the main mutation

<table>
<thead>
<tr>
<th></th>
<th>Normal</th>
<th>Class I</th>
<th>Class II</th>
<th>Class III</th>
<th>Class IV</th>
<th>Class V</th>
</tr>
</thead>
<tbody>
<tr>
<td>CF mutation</td>
<td>W1282X</td>
<td><strong>F508del</strong></td>
<td>G551D</td>
<td>R117H D1152H</td>
<td>3849+10kb C→T</td>
<td></td>
</tr>
<tr>
<td>Allele frequency</td>
<td>~6%</td>
<td><strong>~87%</strong></td>
<td>~3%</td>
<td>&lt;2%</td>
<td>&lt;1%</td>
<td></td>
</tr>
<tr>
<td>Approved drugs</td>
<td></td>
<td></td>
<td>Kalydeco®</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Patient cell data predict clinical outcomes

**Clinical outcome:**
- **55%** responders (FEV1 ≥ 5%) on F508del/F508del
- **75%** responders (FEV1 ≥ 5%) on G551D/F508del

**Patient cells:**
- **F508del**
  - Treated with: VX-809 + Kalydeco
  - ~20% of WT
- **G551D**
  - Treated with: Kalydeco
  - ~30% of WT
'1837

Positioning a potentiator for clinical development

- Improved efficacy in primary cells
- Stable over time
- Favorable metabolic profile
  - reduced DDI liabilities
- Targeting initiation of FIH by end 2014
Goal: improve on VX-809/Kalydeco

Current example

- F508del

<table>
<thead>
<tr>
<th>Healthy</th>
<th>Patient</th>
<th>VX-809</th>
</tr>
</thead>
<tbody>
<tr>
<td>30</td>
<td>10</td>
<td>0</td>
</tr>
</tbody>
</table>

~20% of WT

Room for improvement

Opened with Kalydeco

Possible treatment modes

(I)
- Potentiator
- Corrector 1

Superior potentiatior
Superior corrector

(II)
- Potentiator
- Corrector 1
- Corrector 2

Superior combination
**Vertex combo therapy in Phase III**

Pre-clinical evaluation of F508del-CFTR homozygous primary cells corrected with VX-809 for 24 h. Current after adding 10 µM Forskolin & 500 nM Kalydeco.

Kalydeco + VX-809 = ~ 20% WT
‘1837 is a superior potentiator

Pre-clinical evaluation of F508del-CFTR homozygous primary cells corrected with VX-809 for 24 h. Current after adding 10 μM Forskolin & 500 nM GLPG1837.
We have superior correctors...

Pre-clinical evaluation of F508del-CFTR homozygous primary cells corrected with compound A or VX-809 for 24 h. Current after adding 10 µM Forskolin & 500 nM GLPG1837.
We have superior corrector combinations

Pre-clinical evaluation of F508del-CFTR homozygous primary cells corrected with compound A, B, C, B+C, or VX-809 for 24 h. Current after adding 10 μM Forskolin & 500 nM GLPG1837.
Corrector combination data
Strong activity in F508del cells, opened with ‘1837

<table>
<thead>
<tr>
<th>Lead series</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>VX-809</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
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<td>2</td>
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</table>

10 μM cpd concentration – 3 μM VX-809
Assay: Transepithelial Clamp Circuit

- >50% of wild type CFTR current
- ~25-50% of wild type CFTR current
- <25% of wild type CFTR current

Multiple corrector combinations result in >50% wild type CFTR current
Timelines CF

- **2013**: Delivered PCC potentiator ‘1837
- **2014**: GLPG starts phase 1 potentiator ‘1837
- **2015**: GLPG starts phase 2 potentiator ‘1837
- **2016**: Deal with AbbVie in CF

**Strong position with own potentiator, multiple correctors**
R&D highlights

- ‘634
- ‘974
- Cystic Fibrosis
- GSK2856184
- Anti-infectives
- ‘1790
- R&D overview
Phase 2 studies with GSK2586184

- Selective JAK1 inhibitor GLPG0778 out licensed Jan 2012
- GSK responsible for further research
- Galapagos eligible to receive milestones & royalties
- Ph2 in psoriasis met primary endpoint
  - significantly higher proportion met targeted PASI75% at 400 mg BID dose
  - most common adverse events were headache, common cold
  - GSK considering next steps with GSK2586184
R&D highlights

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New paradigm needed

- One drug to fight one bug
- Narrow spectrum compounds with rapid companion diagnostic
- Decision to treat in less than 1 hour
- Initiate therapy with narrow spectrum antibiotic
  - keep broad spectrum antibiotics as 2\textsuperscript{nd} line
New path for antibacterial approval
Shift to pathogen-focused development

- Disease focused
  - 2 phIII studies

- Pathogen focused
  - 1 phIII study, plus small studies
  - Small studies
  - Animal data

Possibility to rely on human PK data combined with animal model data

Quantity of clinical data vs. unmet medical need
DnaE offers new mode of action

- Confirmed mode of action
  - resistant mutant analysis and sequencing
  - site directed mutagenesis
- Ubiquitous in bacterial world
  - no mammalian homologue
- Essential for Gram-positive and Gram-negative bacteria

GLPG has unique platform for novel class of antibiotics
**Current MRSA strains susceptible to ‘1492**

<table>
<thead>
<tr>
<th>Phenotype</th>
<th>Amoxicillin</th>
<th>Ciprofloxacin</th>
<th>Linezolid</th>
<th>‘1492</th>
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<td>No cross-resistance</td>
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</table>

No cross-resistance with current antibiotics
Strong cidal activity on *S. aureus*

GLPG1492 causes rapid kill of *S. aureus* at 4-fold MIC even at high inoculum
**S. aureus** infection mouse model

‘1492 dose response

Efficacy demonstrated in dose-dependent manner >15 mg/kg
‘1492: an attractive profile

- Highly selective for *S. aureus* including MRSA
- Strong bactericidal activity
- Oral, intravenous, subcutaneous routes available
- No cross-resistance to existing antibiotics

- MRSA associated with a variety of serious indications
  - ABSSSI (formerly cSSSI)
  - bacteremia & endocarditis
  - pneumonia
  - osteomyelitis
  - central nervous system infections

‘1492 is a powerful narrow spectrum agent against MRSA
Anti-infectives expected timelines

- **2014**: Start Ph1 studies ‘1492
- **2015**: Topline Ph1 studies ‘1492
- **2016**: Filing Ph2 Proof of Concept ‘1492

More pathogens to follow
1492 scalable into discovery platform

DnaE

- P. aeruginosa
- S. aureus
- Entero-bacteriaceae
- Other ESKAPE pathogens
- C. difficile
R&D highlights

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‘1790 *in vivo* activity via EPHA2

**Triple-negative breast cancer**

Tumor growth inhibition in MDA-MB-231 xenograft mouse model

- **EPHA2 target engagement *in vivo***
- **Normalized phospho/total EPHA2**
- **‘1790 tumor concentration (ng/g)**

- ‘1790 completely blocks tumor growth at 30 mg/kg
- EPHA2 target inhibited in a dose-dependent manner

---

Mean ± sem, one-way ANOVA + Dunnett’s

***: p<0.001 versus Vehicle

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76
Metastatic melanoma

'1790 on tumor material derived from 19 patients

Ex-vivo tumor cell growth inhibition

Source 1

Source 2

Certain patient types susceptible to '1790

- High sensitivity to '1790 in metastatic melanoma, incl. wild-type BRAF
- Ongoing effort to identify predictive biomarker
‘1790: novel MoA in oncology

Differentiated profile:

- strongly inhibits EPH-R family
- *in vivo* effects in triple negative breast cancer model via inhibition of EPHA2
- shows response in 20% of panel of 100 patient-derived solid tumors

Next steps

- identify a predictive biomarker to stratify patients
- look for an experienced partner to perform clinical trials
R&D highlights

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- R&D overview
Our scientific achievements
Pre-clinical programs since March 2013

- Alliances: progressed programs toward the clinic, achieved milestones
- Alliance with MorphoSys: advanced toward candidate MAb
- Cystic fibrosis: delivered PCC ('1837)
- Anti-infectives: pre-clinical development on track to enter clinic in 2014
- Oncology: delivered PCC ('1790) for triple-negative breast cancer
- Announced three IWT grants for €7.6 M total for early research
Stopped programs since March 2013

- '187
- '1577 (GSK)
- '1179 (GSK)
- '1332 (Servier)

Galapagos aims to make the call early on programs
Progress in discovery R&D
By stage

March 2013
- Target discovery: 54%
- Hit finding: 19%
- Hit to lead: 12%
- Lead Optimization: 15%

June 2014
- Target discovery: 27%
- Hit finding: 39%
- Hit to lead: 9%
- Lead Optimization: 15%
Discovery FTE resources by indication
March 2014

- Cystic fibrosis: 27%
- Inflammation: 24%
- Anti-infectives: 10%
- Fibrosis: 9%
- Metabolic: 9%
- Osteoarthritis: 9%
- Other: 12%

Key focus areas get 80% of FTEs
## Therapeutic scope

<table>
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<th>Early development</th>
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= Research alliance with partner
First into humans in next 18 months

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Preclinical  Healthy volunteers
## Phase 2 planning: topline results 2014-2015

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* = Topline results
R&D Update

- Company strategy
  Onno van de Stolpe, CEO

- R&D strategy & portfolio
  Piet Wigerinck, CSO

- Outlook
  Onno van de Stolpe, CEO
Short term outlook

- Possible $250 M in payments from AbbVie for ‘634 in 2015
- Multiple Phase 2 readouts with novel modes of action
- CF program on track to deliver combination therapy for main mutation
- Strong balance sheet to support our R&D strategy
Five year scenario

• At least one commercial product
• Late stage development pipeline
  ➢ partners plus internal Phase 3 programs
• Fully integrated biotech: discovery, development, marketing & sales
• Focus on innovation with new mode of actions

Galapagos in excellent position to unlock more pipeline value