Turn innovative science into value for patients by characterizing the therapeutic potential of our products.
OUR CORPORATE STRATEGY DRIVES ALL DEVELOPMENT PRIORITIES

Astellas Strategy

Maximize the Product Value

Create Innovation

Pursue Operational Excellence
STRATEGIC PRIORITY: MAXIMIZE THE PRODUCT VALUE

Astellas Strategy

Maximize the Product Value

Create Innovation

Pursue Operational Excellence

Development Priorities

- Expand current indications, explore future indications, evaluate new formulations: enzalutamide
- Meet pediatric regulatory requirements: mirabegron; solifenacin
- Evaluate combination therapy in underserved patient populations: mirabegron + solifenacin
STRATEGIC PRIORITY: CREATE INNOVATION

Astellas Strategy

Maximize the Product Value

Create Innovation

Pursue Operational Excellence

Development Priorities

- Assess full treatment paradigm: gilteritinib
- Recognize competitive environment: ASP8273
- Explore opportunities in treatment-resistant patients: enfortumab vedotin (ASG-22ME)
- Explore new approaches in areas of unmet needs: IMAB362*
- Take flexible licensing approach for novel assets: roxadustat, ASP0113
- Leverage Japan expertise: Amgen Astellas joint venture

*Transaction announced, completion pending
STRATEGIC PRIORITY: PURSUE OPERATIONAL EXCELLENCE

Astellas Strategy

Maximize the Product Value

Create Innovation

Pursue Operational Excellence

Development Priorities

- Build speed and efficiency into pre-POC activities, in light of historical attrition rates in early-stage development
- Leverage global reach and balance internal and external capabilities to execute late stage studies
ASTELLAS STRATEGY ALIGNS WITH EVOLUTION OF EXTERNAL REGULATORY ENVIRONMENT

More consistent approval time across regulatory authorities

Increasing use of expedited review for novel compounds in areas of high unmet medical need

STRATEGIC FOCUS WILL ENABLE EFFECTIVE AND EFFICIENT DELIVERY OF OUR EXPANDING PIPELINE

**Phase 1**
- enfortumab vedotin (ASG-22ME)
- ASG-15ME
- ASP5878
- AGS67E
- ASP4132
- gilteritinib (NSCLC)
- AGS62P1
- ASP2205
- ASP6282
- YM311/FG-2216 (JP)
- ASP7398
- ASP6294
- ASP8302
- ASP5094
- ASP3662
- ASP4345
- ASP4070
- ASP7266
- ASP0892
- ASP1807/CC8464

**Phase 2**
- enzalutamide (Breast cancer, HCC)
- AGS-16C3F (Renal cell carcinoma)
- blinatumomab (AMG 103)
- YM311/FG-2216 (Renal anemia, EU)
- ASP8232 (Diabetic nephropathy)
- bleselumab (ASKP1240)
- peficitinib (ASP015K)
- ASP7962 (Osteoarthritis)
- ASP8062 (fibromyalgia)
- ASP0819 (fibromyalgia)
- ASP1707 (Endometriosis, rheumatoid arthritis)
- ASP7373 (H5N1 influenza, JP)
- CK-2127107 (SMA, COPD)
- RPE cell program (Dry AMD etc.)

**Phase 3**
- enzalutamide (M0 CRPC, M0 BCR: US/EU/Asia, M1 HSPC, TNBC: US/EU/JP/Asia)
- degarelix (3-month, JP)
- gilteritinib (ASP2215)
- ASP8273 (NSCLC, US/EU/JP/Asia)
- solifenacin (Pediatric NDO, US/EU)
- solifenacin/mirabegron (Concomitant use, US/EU/Asia)
- mirabegron (Pediatric NDO, EU)
- roxadustat (ASP1517/FG-4592)
- ASP0113/VCL-CB01 (CMV-HCT, US/EU/JP)
- peficitinib (ASP015K)
- romosozumab (AMG 785)
- fidaxomicin (Infectious enteritis: JP, pediatric: EU)
- ipragliflozin/sitagliptin (Fixed dose combination, JP)
- ipragliflozin (Type 1 diabetes, JP)
- linaclotide (Chrono constipation, JP)

**Filed**
- enzalutamide (Tablet, EU/JP)
- quetiapine (BP-D, JP)
- ASP7374 (Seasonal influenza, JP)
- linaclotide (ASPD0456)

**Therapeutic Area:**
- Oncology
- Urology, Nephrology
- Immunology, Neuroscience
- Others

New molecular/biological entity
Outcome of the projects are shown. Please refer to pipeline list for details including target disease.
AGENDA

I Establishing a Leadership Position in Oncology

II Potential for Gilteritinib in Acute Myeloid Leukemia (AML)

III Advancing Other Late-Stage Oncology Programs

IV Update on Other Late-Stage Programs
ESTABLISHING A LEADERSHIP POSITION IN ONCOLOGY
ESTABLISHING A LEADERSHIP POSITION IN ONCOLOGY

**2007**
- **Agensys Acquisition**
  - Added large-molecules and Antibody Drug Conjugates (ADCs) to development portfolio

**2009**
- **Enzalutamide Licensure**
  - Gained rights to a leading prostate cancer therapy, with potential applicability in other tumor types

**2010**
- **OSI Pharmaceuticals Acquisition**
  - Established expertise in lung cancer; added on-market infrastructure

**2016**
- **Ganymed AG Pharmaceuticals Acquisition***
  - Added novel pipeline of Ideal Monoclonal Antibodies (IMABs), including late-stage asset IMAB362

Established strategic collaborations with world-class research institutions, such as Dana Farber Cancer Institute and MD Anderson Cancer Center, among others

*Transaction announced; completion pending*
DEVELOPMENT STRATEGY IN ONCOLOGY

- Demonstrate impressive efficacy and safety in treatment-resistant populations and areas of highest unmet need
- Expand into earlier stages of disease and/or other tumor types if appropriate
- Utilize precision / targeted approaches if appropriate
- Consider combinations or immuno-oncology (I/O) approaches
FULLY EXPLORING THE THERAPEUTIC POTENTIAL OF ENZALUTAMIDE IN PROSTATE CANCER

**Local Therapy**

- Castration
- Anti-Androgens

**Castration**

- M0 BCR EMBARK PIII study
- M0 CRPC PROSPER PIII study

**Ant-Androgens**

- M1 HSPC ARCHES PIII study
- Post-chemo AFFIRM PIII study

**Chemotherapy**

- Chemo-naive PREVAIL PIII study

**PSA/Tumor volume**

- Asymptomatic
- Symptoms

**Time**

- Non-Metastatic
- Metastatic

- Hormone Sensitive
- Castration Resistant

**Mulders et al., EAU2012; Modified by Astellas**

*1 Radiotherapy, prostatectomy
*2 Metastatic at the time of diagnosis

PSA: Prostate-specific antigen, M0 CRPC: Non-metastatic castration-resistant prostate cancer
M0 BCR: Non-metastatic biochemical recurrence prostate cancer
M1 HSPC: Metastatic hormone sensitive prostate cancer
<table>
<thead>
<tr>
<th>Phase 3 Development Program: Triple Negative Breast Cancer</th>
</tr>
</thead>
<tbody>
<tr>
<td>Precision medicine approach</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Phase 2 Development Program: Breast Cancer sub-types</th>
</tr>
</thead>
<tbody>
<tr>
<td>ER/PR+, AR+/Her-2+</td>
</tr>
</tbody>
</table>

| Phase 2 Development Program: Hepatocellular Carcinoma     |
## ASTELLAS’ ONCOLOGY PIPELINE

<table>
<thead>
<tr>
<th>Project</th>
<th>Target Cancer</th>
<th>Characteristics</th>
<th>P1</th>
<th>P2</th>
<th>P3</th>
<th>Filed</th>
</tr>
</thead>
<tbody>
<tr>
<td>enzalutamide</td>
<td>Prostate cancer (M0 CRPC, M0 BCR, M1 HSPC), Breast cancer, Hepatocellular carcinoma</td>
<td>Androgen receptor inhibitor</td>
<td>PC, TNBC</td>
<td>BC, HCC</td>
<td></td>
<td></td>
</tr>
<tr>
<td>degarelix</td>
<td>Prostate cancer</td>
<td>GnRH antagonist</td>
<td></td>
<td></td>
<td>3-month: JP</td>
<td></td>
</tr>
<tr>
<td>gilteritinib</td>
<td>Acute myeloid leukemia, Non-small cell lung cancer</td>
<td>FLT3/AXL inhibitor</td>
<td>AML</td>
<td>NSCLC</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ASP8273</td>
<td>Non-small cell lung cancer</td>
<td>Mutant-selective irreversible EGFR inhibitor</td>
<td></td>
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</tr>
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<td>ASP4132</td>
<td>Advanced cancer</td>
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<td></td>
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<tr>
<td>IMAB362*</td>
<td>Gastroesophageal adenocarcinoma</td>
<td>Ideal Monoclonal Antibody (target: CLDN18.2)</td>
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<tr>
<td>AGS-16C3F</td>
<td>Renal cell carcinoma</td>
<td>Antibody utilizing ADC (target: ENPP3)</td>
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<td>blinatumomab</td>
<td>Acute lymphoblastic leukemia</td>
<td>Anti-CD19 BiTE</td>
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<td>enfortumab vedotin (ASG-22ME)</td>
<td>Urothelial cancer, Solid tumors</td>
<td>Antibody utilizing ADC (target: Nectin-4)</td>
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<tr>
<td>ASG-15ME</td>
<td>Urothelial cancer</td>
<td>Antibody utilizing ADC (target: SLITRK6)</td>
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<tr>
<td>AGS67E</td>
<td>Lymphoid malignancy</td>
<td>Antibody utilizing ADC (target: CD37)</td>
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<tr>
<td>AGS62P1</td>
<td>Acute myeloid leukemia</td>
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*Transaction announced; completion pending
# ASTELLAS’ ONCOLOGY PIPELINE

## Small molecule

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## Antibody

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*Transaction announced; completion pending*
POTENTIAL FOR GILTERITINIB IN AML
ACUTE MYELOID LEUKEMIA
AND GILTERITINIB

Jessica K. Altman, M.D.
Director, Acute Leukemia Program
Robert H. Lurie Comprehensive Cancer Center
Associate Professor of Medicine
Feinberg School of Medicine, Northwestern University, Chicago, IL
December 8, 2016
AGENDA

I. Current treatment landscape in AML and unmet medical needs

II. Characteristics of gilteritinib

III. Expectation for gilteritinib as a clinical physician
“Deb” (alias), 52-year-old female, presented to her primary care physician with a week of fever of 103° F, generally feeling unwell;

Because of the persistent symptoms, a complete blood count (CBC) is drawn revealing white blood cells (WBC) of 196,000/uL, Hemoglobin (Hgb) of 5.7 g/dL, and platelet (PLT) count of 80,000/uL;

She is instructed to go to the Emergency Room (ER) for urgent evaluation. At the ER, her exam is notable only for scattered bruises and mild gingival hyperplasia;

She undergoes bone marrow evaluation and is diagnosed with AML with Normal karyotype (NK) and a FLT3 ITD
ACUTE MYELOID LEUKEMIA (AML) INTRODUCTION

- Estimated new cases/deaths (US) 2016: 19,950/10,430
- ~25% will survive 5 years
- Median age: 67 years
- Heterogeneity in genetics, clinical manifestations, and outcome
- New targeted agents promising

APPROACH TO NEWLY DIAGNOSED PATIENT

- History and physical (organomegaly, extramedullary disease)
- CBC with differential, chemistry panel including uric acid
- Smear review
- PT, PTT, fibrinogen (Disseminated intravascular coagulation (DIC) panel)
- Bone marrow aspirate and biopsy
  - Morphology and flow cytometry
  - Cytogenetics – prognosis, treatment, role of transplant
  - Molecular studies – prognosis, role of transplant, targeted treatment (had been restricted to trials but not for long)
- Risk assessment and transplant planning
- Discussion of fertility
## TOWARDS A RECLASSIFICATION OF CYTOGENETIC (AND MOLECULAR) RISK GROUPS

<table>
<thead>
<tr>
<th>Risk Group</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Very favorable</td>
<td>t(15;17) with any abn</td>
</tr>
<tr>
<td>Favorable</td>
<td>inv(16) lacking c-KIT; t(8;21) lacking del(9q) or complex karyotype or c-KIT; Mutated NPM1 without FLT3-ITD (normal karyotype); Mutated CEBPα+ (double mutation) (normal karyotype)</td>
</tr>
<tr>
<td>Intermediate</td>
<td>Normal or +8 or +21 or others</td>
</tr>
<tr>
<td>Unfavorable</td>
<td>-5/del(5q), -7/del(7q), inv(3) or t(3;3), t(v;11)(v;q23), 17p, t(6;9), t(9;22), complex karyotypes with ≥ 3 abn; inv(16) or t(8;21) with c-KIT; normal karyotype with FLT3+; monosomal karyotype</td>
</tr>
</tbody>
</table>

Continued modification with the recognition of new prognostic markers

Adapted from Slovak Blood 2000 and Dohner Blood 2010
TREATING AML IN YOUNGER ADULTS

- **Induction**: dauno 90 mg/m²/d x 3d (or ida) + ara-C 100 mg/m²/d x 7d continuous infusion;

- **Consol**: multiple cycles (3-4) of HIDAC in younger pts fav-risk, NK FLT3-/NPM1+, or biallelic CEBPα+; 3-4 for CBF

- Allogeneic HCT for intermed- and high-risk (consider alternative donor if no sib); including FLT3 ITD +

- No maintenance

- Relapse: Re-induction chemo then allogeneic transplantation

A GENERAL APPROACH TO THE OLDER ADULT WITH AML

AML Diagnosis: cyto, molecular, co-morbidities and social assessment

Not Eligible for Intensive Treatment

Unfavorable Risk Profile

Donor?

No

Standard induction treatment

Yes

Standard induction treatment with planned alloHCT

Eligible for Intensive Treatment

Consider alloHCT

Well designed clinical trial always preferred

Non-intensive regimen (ex: HMA)

In part adapted from: Ossenkoppele and Löwenberg *Blood* 2015 125:767-774
FLT3 AS A TARGET IN AML

- Promotes proliferation and blocks differentiation
- Activating mutations present in ~30% of AML (ITD and activation loop)
- Patients with FLT3/ITD mutations have a worse prognosis – increased relapsed rate, lower OS
- Associated with leukocytosis and high percentage of bone marrow blasts, de novo AML
- *FLT3* inhibitors in development; single agent and combination studies


FLT3 INHIBITOR DEVELOPMENT

### Table 1. Most frequently used FLT3 inhibitors

<table>
<thead>
<tr>
<th>Name of the drug</th>
<th>Kinase inhibitory profile</th>
<th>Disease under evaluation</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sorafenib (Nexavar)</td>
<td>CRAF and BRAF, KIT, FLT3, VEGFR-2, VEGFR-3, and PDGFR-β</td>
<td>AML, Hepatocellular carcinoma, Renal cell carcinoma</td>
<td>Most of these kinases are involved in angiogenesis.</td>
</tr>
<tr>
<td>Quizartinib (AC220)</td>
<td>FLT3/STK1, CSF1R/FMS, SCFR/KIT, PDGFRs</td>
<td>AML, Thyroid carcinoma</td>
<td>It is the most potent <em>in vitro</em> FLT3 inhibitor.¹⁶</td>
</tr>
<tr>
<td>Midostaurin (PKC412)</td>
<td>FLT3, KIT, PDGFR-β, VEGFR-2, PKC</td>
<td>AML, MDS, Aggressive systemic mastocytosis and mast cell leukemia⁸¹</td>
<td>Inhibits FLT3 at very low doses, generally in the nanomolar range.⁴⁸</td>
</tr>
<tr>
<td>Lestaurnib (CEP701)</td>
<td>FLT3, JAK2, TRK A/TRK B/TRK C</td>
<td>AML and MPN⁸²,⁸³</td>
<td>—</td>
</tr>
<tr>
<td>Crenolanib (CP868596)</td>
<td>FLT3-ITD, FLT3-DB85, PDGFR-α, PDGFR-β</td>
<td>AML, GIST, Glioma</td>
<td>—</td>
</tr>
<tr>
<td>Gilteritinib (ASP2215)</td>
<td>FLT3, AXL, ALK</td>
<td>AML</td>
<td>—</td>
</tr>
</tbody>
</table>

Abbreviations: FLT3 = FMS-like tyrosine kinase 3; GIST = gastrointestinal stromal tumor; ITD = internal tandem duplications; MDS = myelodysplastic syndrome; MPN = myeloproliferative neoplasms.

Antar A et al; Bone Marrow Transplantation (2016) 1-8.
**PHASE 3 RATIFY TRIAL SCHEMA**

Prospective Phase 3, double-blinded randomized study of induction and consolidation +/- midostaurin (PKC412) in newly diagnosed adults <60 years old with FLT3 mutated AML

**Pre-Registration**
- FLT3 WILD TYPE not eligible for enrollment

**Randomize**
- FLT3 ITD or TKD
  - Daunorubicin ARA-C Midostaurin
  - Daunorubicin ARA-C Placebo

**CR**
- HiDAC Midostaurin
- HiDAC Placebo

**MAINTENANCE 12 Months**
- Midostaurin
- Placebo

Stratification: TKD; ITD with allelic ratio <0.7 vs. ≥0.7
RATIFY TRIAL RESULTS

• *FLT3* centrally (48 hr)

• CR by day 60 in midostaurin arm 59% vs. 53% in placebo arm (NS)

• Median OS: Midostaurin 74.7 months; placebo 25.6 mo (p = 0.0074)

• Midostaurin improves OS when added to standard chemotherapy with maintenance in newly diagnosed patients aged 18-60 years old with ITD and TKD *FLT3* mutant AML
“DEB’S” TREATMENT

• Enrolled on C10603: A Phase 3 Randomized, Double-Blind Study of Induction (Daunorubicin/Cytarabine) and Consolidation (High-Dose Cytarabine) Chemotherapy + Midostaurin (PKC412) or Placebo in Newly Diagnosed Patients < 60 Years of Age with FLT3 Mutated AML

• Attained aplastic marrow at day 14 and then entered CR ~ day 28

• Matched sibling donor allogeneic stem cell transplant in CR1

• (Deb’s disease recurred ~ day 100 and was treated off study w 5-aza and sorafenib)
GILTERITINIB: A HIGHLY SELECTIVE FLT3/AXL INHIBITOR

- Activating mutations of **FLT3** occur in ~30% of AML cases\(^1\)
  - Internal tandem duplications (ITD) in the juxtamembrane domain confer a poor prognosis\(^1,2\)
  - Point mutations (especially D835) in the tyrosine kinase domain induce resistance to FLT3 inhibitors\(^3\)

- Gilteritinib (ASP2215) is a highly potent, selective FLT3/AXL inhibitor that has demonstrated consistent and sustained inhibition of FLT3 in vitro\(^4-6\)

- **CHRYSALIS** is a first-in-human, pharmacodynamic-driven, open-label Phase 1/2 trial (NCT02014558) of once-daily oral gilteritinib in relapsed/refractory (R/R) AML
  - Adults with R/R AML irrespective of FLT3 mutation status were enrolled from 28 sites across the US and Europe
  - Primary end points were safety, tolerability, and pharmacokinetic profile
  - The key secondary end point was antileukemic activity; pharmacodynamic effects were an exploratory end point
  - Data locked June 2016

---

\(^5\)Perl, A et al. *Haematologica*. 2015;100(Suppl 1);
MY EXPECTATION FOR TREATING AML IN YOUNGER ADULTS IN THE FLT3 INHIBITOR ERA (IN THE NEAR FUTURE)

• **Induction:** dauno x 3d (or ida) + ara-C x 7d c.i + FLT3i;

• **Post remission therapy:**
  – HiDAC + FLT3i
  – Allogeneic HCT

• Maintenance post transplant or consolidation with FLT3i

• Relapse: FLT3i alone or re-induction chemo with FLT3i
NOT TOO DISTANT HORIZON: OLDER ADULT WITH FLT3 MUTATED AML

AML Diagnosis: cyto, molecular, co-morbidities and social assessment

Not Eligible for Intensive Treatment
  Non-intensive regimen (ex: HMA) with a FLT3i

Eligible for Intensive Treatment
  Eligible for alloHCT
    No
      FLT3i-based maintenance
    Yes
      Consider alloHCT and FLT3i maintenance

Well designed clinical trial always preferred

In part adapted from: Ossenkoppele and Löwenberg *Blood* 2015 125:767-774
FUTURE OF GILTERITINIB AND REMAINING QUESTIONS

• Eagerly awaiting approval for patients with recurrent FLT3 mutated disease
  – single agent activity
  – tolerance
• Await data from combination studies in newly diagnosed patients with standard chemotherapy
  – midostaurin + 7+3 data
  – data needed to understand comparison
  – will specificity of inhibitor matter in upfront setting?
• Await results from combination studies with HMA
  – set apart than other available agents
• Post transplant maintenance
• All settings where clinicians will want to utilize gilteritinib if activity confirmed
DEVELOPMENT PORTFOLIO HIGHLIGHTS CONTINUED

R&D meeting 2016

Bernie Zeiher, M.D.
President, Development
Astellas Pharma Inc.
December 8, 2016
POTENTIAL FOR GILTERITINIB

CONTINUED
GILTERITINIB: TREATMENT ALGORITHM AND DEVELOPMENT PROGRAM

AML Patients (~35,000)*
mFLT3 ~30%

High-Intensity Induction / Consolidation

- Phase 1
  - Chemo Consolidation
  - Transplant
    - Maintenance
      - GOSSAMER study Phase 3
    - Maintenance
      - MОРPHО study Phase 3
  - Salvage Therapy
    - CHRYСALIS study (Phase 1), ADMIRAL study (Phase 3)

Low-Intensity Chemo

- LACEWING study Phase 2/3
ADVANCING OTHER LATE-STAGE ONCOLOGY PROGRAMS
ASP8273: JAPANESE PHASE 2 DATA FOR NSCLC FIRST LINE TREATMENT PRESENTED AT 17TH WORLD CONFERENCE ON LUNG CANCER

Best percentage change from baseline in target-lesion size

Waterfall plot shows investigator-assessed tumour response. Thirty subjects had evaluable target lesion data.

*Denotes patients with de novo T790M mutation; △patient who experienced progressive disease; ▲patients who discontinued due to progressive disease.

Treatment-related adverse events occurring in >=15% of the ASP8273 300 mg population

<table>
<thead>
<tr>
<th>Treatment-Related Adverse Events, n (%)</th>
<th>Grade 1</th>
<th>Grade 2</th>
<th>Grade 3</th>
<th>Grade 4</th>
<th>Overall</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diarrhoea</td>
<td>14 (45)</td>
<td>5 (16)</td>
<td>2 (6)</td>
<td>0</td>
<td>21 (68)</td>
</tr>
<tr>
<td>Peripheral sensory neuropathy</td>
<td>11 (36)</td>
<td>1 (3)</td>
<td>0</td>
<td>0</td>
<td>12 (39)</td>
</tr>
<tr>
<td>Elevated ALT</td>
<td>6 (19)</td>
<td>3 (10)</td>
<td>2 (6)</td>
<td>0</td>
<td>11 (35)</td>
</tr>
<tr>
<td>Hyponatraemia</td>
<td>3 (10)</td>
<td>–</td>
<td>6 (19)</td>
<td>1 (3)</td>
<td>10 (32)</td>
</tr>
<tr>
<td>Nausea</td>
<td>7 (23)</td>
<td>3 (10)</td>
<td>0</td>
<td>0</td>
<td>10 (32)</td>
</tr>
<tr>
<td>Dry mouth</td>
<td>6 (19)</td>
<td>2 (6)</td>
<td>0</td>
<td>0</td>
<td>8 (26)</td>
</tr>
<tr>
<td>Elevated AST</td>
<td>5 (16)</td>
<td>2 (6)</td>
<td>1 (3)</td>
<td>0</td>
<td>8 (26)</td>
</tr>
<tr>
<td>Decreased appetite</td>
<td>5 (16)</td>
<td>1 (3)</td>
<td>1 (3)</td>
<td>0</td>
<td>7 (23)</td>
</tr>
<tr>
<td>Dry skin</td>
<td>5 (16)</td>
<td>2 (6)</td>
<td>0</td>
<td>0</td>
<td>7 (23)</td>
</tr>
<tr>
<td>Stomatitis</td>
<td>5 (16)</td>
<td>1 (3)</td>
<td>0</td>
<td>0</td>
<td>6 (19)</td>
</tr>
<tr>
<td>Dysgeusia</td>
<td>3 (10)</td>
<td>2 (6)</td>
<td>0</td>
<td>0</td>
<td>5 (16)</td>
</tr>
<tr>
<td>Malaise</td>
<td>4 (13)</td>
<td>1 (3)</td>
<td>0</td>
<td>0</td>
<td>5 (16)</td>
</tr>
<tr>
<td>Rash</td>
<td>4 (13)</td>
<td>1 (3)</td>
<td>0</td>
<td>0</td>
<td>5 (16)</td>
</tr>
<tr>
<td>Vomitting</td>
<td>5 (16)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>5 (16)</td>
</tr>
</tbody>
</table>

TRAE occurring in ≥15% of subjects;

*No classification of Grade 2 hyponatremia within the NCI-CTCAE.

Date of data cut off: 23 February 2016.

Nishio et al., WCLC2016
NSCLC, Non-small cell lung cancer; ALT, alanine aminotransferase; AST, aspartate aminotransferase; NCI-CTCAE, national cancer institute common terminology criteria for adverse events; TRAE, treatment-related adverse events.
ENFORTUMAB VEDOTIN (ASG-22ME): TARGET AND MECHANISM OF ACTION

**Target**

Nectin-4 is a type I transmembrane protein that belongs to the Nectin family of adhesion molecules.

**Normal tissue:**
- Variable, mostly weak or moderate, expression was detected by IHC in transitional epithelium of bladder, skin (epidermis, sweat glands and hair follicles), salivary gland (ducts), esophagus, breast, and stomach.

**Malignant tissue:**
- Highly expressed in bladder cancer with more moderate expression in breast, pancreatic, lung and ovarian cancer tissue microarrays (TMA).
- 83% (434/524) of bladder cancers on TMA were positive, 60% with strong or moderate staining.

**Antibody Drug Conjugate (ADC)**

Enfortumab vedotin is an antibody drug conjugate (ADC) with the following components:
- Fully human monoclonal antibody IgG1k directed against Nectin-4.
- Protease-cleavable linker.
- Microtubule-disrupting agent monomethylauristatin-E (MMAE).

ENFORTUMAB VEDOTIN: PHASE 1 IN METASTATIC UROTHELIAL CARCINOMA SUBJECTS

Waterfall Plot of Maximum Change from Baseline in Phase 1 Metastatic Urothelial Carcinoma Subjects

Overall Response in Evaluable Subjects* with mUC

<table>
<thead>
<tr>
<th>Best Overall Response, N (%)</th>
<th>1.25 mg/kg (n=17)</th>
<th>Total (N=49)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ORR (CR+PR)</td>
<td>10 (59)</td>
<td>18 (37)</td>
</tr>
<tr>
<td>95% CI</td>
<td>32.9, 81.6</td>
<td>23.4, 51.7</td>
</tr>
<tr>
<td>DCR (CR+PR+SD)</td>
<td>14 (82)</td>
<td>37 (76)</td>
</tr>
<tr>
<td>95% CI</td>
<td>56.6, 96.2</td>
<td>61.1, 86.7</td>
</tr>
</tbody>
</table>

**ORR Subcategories, N (%)**

- Subject with liver metastasis: 1/1 (100) vs 5/12 (42)
- Prior taxanes: 4/6 (67) vs 8/20 (40)
- Prior CPI: 4/7 (57) vs 6/16 (38)

*Evaluable Subjects are defined as subjects having at least one post-baseline radiographic assessment; Response assessed per RECIST 1.1 Response rate includes unconfirmed response, study is enrolling.

Rosenberg et al., ESMO2016
CI: Confidence intervals, DCR: Disease Control Rate
ENFORTUMAB VEDOTIN: SAFETY DATA IN PHASE 1 STUDY

Treatment-Emergent Adverse Events (TEAE)* ≥ 20% N=58

- Fatigue: All Events 43, Considered Related 30
- Nausea: All Events 43, Considered Related 28
- Diarrhea: All Events 33, Considered Related 29
- Pruritis: All Events 33, Considered Related 31
- Decreased appetite: All Events 26, Considered Related 17
- Alopecia: All Events 24, Considered Related 21
- Dysgeusia: All Events 22, Considered Related 14
- Rash: All Events 34, Considered Related 26
- UTI: All Events 22, Considered Related 7
- Vomiting: All Events 22, Considered Related 12

*No events were Grade 4 in severity

Rosenberg et al., ESMO2016
ENFORTUMAB VEDOTIN: NEXT STEPS

Consult with regulatory agencies and pursue registrational-directed development plan in patients who have been exposed to check point inhibitor (CPI) therapy.

Continue Phase 1 expansion cohorts in other Nectin 4 expressing solid tumors, including NSCLC and ovarian.
GANYMED: LEVERAGING ACQUISITIONS TO ACQUIRE NEW PLATFORMS AND TARGET NEW TUMOR TYPES

Strategic acquisition*

Would expand oncology pipeline

Includes IMAB362, the late-stage first-in-class antibody against CLDN18.2

Received orphan drug designation in the U.S. and EU for gastric and pancreatic cancers

*Transaction announced; completion pending
IMAB362: THE TARGET OF CLAUDIN18.2

- Member of the claudin family
- Major structural component of tight junctions
  - Seals intercellular space in epithelial sheets
- Broadly express in various cancer types
  - ~70-90% biliary duct, pancreatic, gastric and mucinous ovarian cancer
  - ~10% ovarian cancer and NSCLC
- Not expressed in any healthy tissues, except for stomach mucosa, with limited accessibility to the antibody
**IMAB362: MECHANISM OF ACTION**

- Chimeric IgG1 backbone antibody
- Highly specific for Claudin18.2
- Modes of action:
  - Antibody-dependent cellular cytotoxicity (ADCC)
  - Complement-dependent cytotoxicity (CDC)
  - In combination with chemotherapy:
    - enhances T-cell infiltration
    - induces pro-inflammatory cytokines

---

**Target patients**

- Gastric, esophageal or the gastroesophageal junction adenocarcinoma
- CLDN18.2: 2+/3+ intensity in ≥ 40% tumor cells (centrally measured with analytically validated, CE marked IVD Kit)
- 1st line, no prior CTx for advanced disease
- Locally advanced or metastatic disease

**Design**

- Randomized Phase 2 trial, open-label
- Arm 1, Arm 2 randomized 1:1
- Added exploratory Arm 3, started after 80% of arms 1&2 had been recruited, 1:1:7 randomization for catch up
- At randomization: Stratification according (i) CLDN18.2 positivity, (ii) measurability of disease
- Primary endpoint: Progression-free survival (PFS), Key secondary endpoint: Overall survival (OS)
IMAB362: PFS IN FAST STUDY
TOTAL POPULATION (2+/3+ CLDN18.2 STAINING IN ≥ 40% OF TUMOR CELLS)

*Based on central imaging assessment in patients with 2+/3+ CLDN18.2 staining in ≥40% of tumor cells (total population);
Updated data presented by Al-Batran et al., ASCO2016

PFS* (primary endpoint): Arm 2 vs. Arm 1
mPFS 4.8 vs. 7.9 mo
HR 0.47
P=0.0001

PFS (exploratory): Arm 3 vs. Arm 1
mPFS 4.8 vs 7.1
HR 0.51
p=0.001
IMAB362: PFS AND OS IN PATIENTS WITH 2+/3+ CLDN18.2 STAINING IN ≥ 70% OF TUMOR CELLS (HIGH EXPRESSOR SUBGROUP) IN FAST STUDY

**PFS**
- mPFS 5.6 vs. 7.2 mo
- HR 0.36
- p=<0.0005

**OS**
- mOS 9 vs. 16.7 mo
- HR 0.45
- p=<0.0005
### IMAB362: SELECTED ADVERSE EVENTS (NCI-CTC CRITERIA) IN FAST STUDY

<table>
<thead>
<tr>
<th>Adverse Event/treatment arm</th>
<th>EOX</th>
<th>EOX+IMAB362</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>G1/2</td>
<td>G3/4</td>
</tr>
<tr>
<td>Anemia</td>
<td>24 (28.6)</td>
<td>6 (7.1)</td>
</tr>
<tr>
<td>Leukopenia</td>
<td>10 (11.9)</td>
<td>5 (6)</td>
</tr>
<tr>
<td>Neutropenia</td>
<td>18 (21.4)</td>
<td><strong>18 (21.4)</strong></td>
</tr>
<tr>
<td>Thrombocytopenia</td>
<td>7 (8.3)</td>
<td>3 (3.6)</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>29 (34.5)</td>
<td>3 (3.6)</td>
</tr>
<tr>
<td>Nausea</td>
<td>52 (61.9)</td>
<td>3 (3.6)</td>
</tr>
<tr>
<td>Vomiting</td>
<td><strong>29 (34.5)</strong></td>
<td><strong>3 (3.6)</strong></td>
</tr>
<tr>
<td>Asthenia</td>
<td>17 (20.2)</td>
<td>2 (2.4)</td>
</tr>
<tr>
<td>Fatigue</td>
<td>14 (16.7)</td>
<td>3 (3.6)</td>
</tr>
<tr>
<td>Infections</td>
<td>9 (10.7)</td>
<td>2 (2.4)</td>
</tr>
</tbody>
</table>

Al-Batran et al., ASCO2016
UPCOMING ONCOLOGY CATALYSTS

FY2016-2017

- IMAB362
  - Closing of Ganymed acquisition*

- Enzalutamide
  - Readout of P2 ER/PR
  - Readout of P2 Her2+

- ASP8273
  - Final results for P1/2

FY2018-2020

- Gilteritinib
  - FPI in GOSSAMER and MORPHO P3 Maintenance Trials

- Enfortumab Vedotin
  - Regulatory discussions
  - Initiate study in CPI treated patients

- Enzalutamide
  - Data readout for PROSPER

- Gilteritinib
  - Data readout for ADMIRAL

- ASP8273
  - Data readout of P3

Note: All dates are approximate. Timing to be based on study progress, event rates and interim analysis triggers.
*Transaction announced; completion pending
UPDATE ON OTHER LATE-STAGE PROGRAMS
ROXADUSTAT: ACTIVATES A NATURAL PATHWAY TO INCREASE RED BLOOD CELL PRODUCTION

NORMAL OXYGEN

HIF-α

HIF-PH Enzymes

HIF-α Degrades Rapidly

Degradation

LOW OXYGEN (e.g., High Altitude) or

ROXADUSTAT

FIBROGen

HIF-α

HIF-PH Enzymes

Roxadustat Stabilizes HIF-α

HIF-α

HIF-α

HIF-α

HIF-β

Gene Transcription

EPO Within or Near Physiological Range

Iron Transport to the Bone Marrow and Hemoglobin (Hb) Synthesis

Iron Absorption

Hepcidin Levels

Red Blood Cell Production

HIF-PH - hypoxia-inducible factor prolyl hydroxylase
ROXADUSTAT: RESULTS FROM NON-DIALYSIS PHASE 2 STUDY IN JAPAN

Primary endpoint: Mean change in Hb from baseline during the fixed-dose period

- Placebo (N=27): -0.052
- Roxadustat 50 mg TIW starting dose (N=27): 0.200
- Roxadustat 70 mg TIW starting dose (N=26): 0.453
- Roxadustat 100 mg TIW starting dose (N=27): 0.570
- Pooled Roxadustat TIW (N=80): 0.407

Adjusted Difference: 0.623*
Adjusted Difference: 0.508*
Adjusted Difference: 0.254*

*P<0.001. Rate of rise was calculated as the slope of a linear regression for each patient using all Hb data collected during the fixed-dose period. Error bars represent standard deviation.

Safety: Roxadustat was well tolerated and had an adverse event profile similar to that observed in previous studies.

Akizawa et al., American Society of Nephrology Kidney Week 2016
Dl: deciliters, g: grams, Hb: hemoglobin, mg: milligrams, TIW: three times weekly
## ROXADUSTAT: ROBUST PHASE 3 PROGRAM TO SUPPORT FILING AND REIMBURSEMENT IN EUROPE AND JAPAN

<table>
<thead>
<tr>
<th></th>
<th>Dialysis</th>
<th>Non-dialysis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Global</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HIMALAYAS:</td>
<td>Incident dialysis, vs epoetin alfa</td>
<td>DOLOMITES, vs darbepoetin</td>
</tr>
<tr>
<td>SIERRAS:</td>
<td>Stable dialysis, vs epoetin alfa</td>
<td>ALPS, vs placebo</td>
</tr>
<tr>
<td>PYRENEES:</td>
<td>Stable dialysis, vs epoetin alfa or darbepoetin</td>
<td>Enrollment completed</td>
</tr>
<tr>
<td><strong>Japan</strong></td>
<td>HD: Conversion, vs darbepoetin</td>
<td>Conversion, vs darbepoetin</td>
</tr>
<tr>
<td>HD: Conversion</td>
<td>HD: Conversion, long-term</td>
<td></td>
</tr>
<tr>
<td>HD: Correction</td>
<td>HD: Correction</td>
<td></td>
</tr>
<tr>
<td>PD</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### For additional anemia indications
Phase 3 study to start for anemia in myelodysplastic syndromes (MDS)
- US FDA has approved an IND for anemia in MDS

**Note:** Company logo in the table shows the sponsor of studies
### SOLIFENACIN/MIRABEGRON: OBTAINED TOP LINE RESULTS FROM PHASE 3 STUDY SYNERGY 2

<table>
<thead>
<tr>
<th>Phase 3 program</th>
<th>Safety in SYNERGY 2</th>
<th>Efficacy in SYNERGY 2</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>BESIDE</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Achieved primary endpoints, demonstrating that solifenacin with mirabegron as add-on therapy was superior to solifenacin monotherapy</td>
<td>All treatments were well tolerated.</td>
<td>Combination S5+M50 mg was statistically significantly superior to the M50 mg and S5 mg groups for the primary efficacy endpoints (change in incontinence episodes and change in micturitions per 24 hours).</td>
</tr>
<tr>
<td><strong>SYNERGY</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Did not meet one of primary endpoints (p=0.052), but improvements for a number of efficacy endpoints indicative of additive effects.</td>
<td>The safety profile was as expected based on that of the monotherapies with the frequency of TEAEs (one of the primary endpoints) in the combination group somewhat higher compared to the S5 mg and M50 mg groups.</td>
<td>Efficacy was maintained during the 1-year treatment period for all primary and key secondary efficacy endpoints.</td>
</tr>
<tr>
<td><strong>SYNERGY 2</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Double-blind, active-controlled (vs monotherapies), long term study (n=1,829)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Plan to discuss next steps with health authorities based on results from Phase 3 studies**

S5+M50 mg: solifenacin 5 mg + mirabegron 50 mg, S5 mg: solifenacin 5 mg, M50 mg: mirabegron 50 mg TEAEs: treatment emergent adverse events
UPDATES FOR LATE STAGE PROJECTS

**ASP0113**

**Target:** Cytomegalovirus (CMV) reactivation in hematopoietic cell transplant recipients

**Designed to elicit both T-cell and antibody immune responses against CMV**

**Progress**

- Phase 3 study enrollment completed
- Top line results are expected in FY2017

**Romosozumab**

**Target:** Osteoporosis

**Romosozumab is studied for its potential to increase BMD, improve bone structure and strength and reduce the risk of fractures.**

**Progress**

- We plan to file in Japan later this month.

**Romosozumab**

**Target:** Osteoporosis

**Romosozumab is studied for its potential to increase BMD, improve bone structure and strength and reduce the risk of fractures.**

**Progress**

- We plan to file in Japan later this month.

**Other P2/P3 programs**

**Immunology**

- Peficitinib (ASP015K): Phase 3 for rheumatoid arthritis ongoing in Japan
- Bleselumab (ASKP1240): Initiated Phase 2 with Kyowa Hakko Kirin for recurrence of focal segmental glomerulosclerosis in de novo kidney transplant recipients

**Neuroscience**

- ASP7962 for osteoarthritis
- ASP8062 and ASP0819 for fibromyalgia
Turn innovative science into value for patients by characterizing the therapeutic potential of our products.
AGENDA

I. Oncology marketing strategy and capabilities

II. Potential patient impact in priority cancer types
We dedicate our collective strengths to develop and deliver paradigm changing treatment options for cancer patients globally.

**Mission**

- Drive organic innovation and capture external opportunities
- Deep understanding of customer needs
- Define value based strategy and clear differentiation
- Build Global Marketing excellence
- Develop strong launch capabilities
To secure market access and reimbursement, new products must prove they deliver value to all key stakeholders in the Healthcare System.
<table>
<thead>
<tr>
<th>Drive Growth</th>
<th>Progress Pipeline</th>
<th>Commercial Excellence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Execute XTANDI strategy</td>
<td>Launch preparation</td>
<td>• Global launch excellence</td>
</tr>
<tr>
<td>• Earlier M1 CRPC use</td>
<td>• Enzalutamide BC</td>
<td>• One global voice – strengthen marketing capabilities and center of excellence</td>
</tr>
<tr>
<td>• Strong case for value</td>
<td>• Gilteritinib in r/r AML</td>
<td>• Early value and access decisions – established global function</td>
</tr>
<tr>
<td>• New indications in PC</td>
<td>• ASP8273 in NSCLC</td>
<td>• Start with the patient - understand and focus on their needs</td>
</tr>
</tbody>
</table>

**Business Development**

- Integration of Ganymed
- Continue to amend pipeline

**Select and progress the most differentiated assets**

- Strong scientific evidence and value proposition
- Well defined Life Cycle Plan

PC: Prostate cancer, BC: Breast cancer, M1 CRPC: Metastatic castration-resistant prostate cancer, r/r AML: Relapsed or refractory acute myeloid leukemia, NSCLC: Non-small cell lung cancer
# SELECTED ONCOLOGY PIPELINE OPPORTUNITIES

<table>
<thead>
<tr>
<th>Project</th>
<th>Patient Population</th>
<th>Dev Phase</th>
<th>Patient Number</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Small molecule</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Enzalutamide</strong></td>
<td>Prostate cancer (M0 CRPC, M0 BCR, M1 HSPC), Breast cancer, Hepatocellular carcinoma</td>
<td>Phase 3</td>
<td>●●●</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Phase 2</td>
<td>●●</td>
</tr>
<tr>
<td><strong>Gilteritinib</strong></td>
<td>Acute myeloid leukemia, Non-small cell lung cancer</td>
<td>Phase 3</td>
<td>●●●</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Phase 2</td>
<td>●●●●</td>
</tr>
<tr>
<td><strong>ASP8273</strong></td>
<td>Non-small cell lung cancer</td>
<td>Phase 3</td>
<td>●●●</td>
</tr>
</tbody>
</table>

| **Antibody** |                                                                                      |           |                |
| **IMAB362*** | Gastroesophageal adenocarcinoma                                                       | Phase 2   | ●●●            |
| **Enfortumab vedotin (ASG-22ME)** | Urothelial cancer, Solid tumors                                                       | Phase 1   | ●●●●          |
| **ASG-15ME**  | Urothelial cancer                                                                      | Phase 1   | ●●●●          |

*Transaction of Ganymed announced; completion pending

**Legend:**
- ● ● ● > 50,000 Patients
- ● ● ○ 20,000 - 50,000 Patients
- ● ○ ○ < 20,000 Patients

**Abbreviations:**
- M0 CRPC: Non-metastatic castration-resistant prostate cancer
- M0 BCR: Non-metastatic biochemical recurrence
- M1 HSPC: Metastatic hormone sensitive prostate cancer
XTANDI: MORE THAN 140,000 PATIENTS HAVE BEEN TREATED WITH XTANDI SINCE IT’S LAUNCH IN SEPT. 2012

- Launched in 65 countries
- Strong Y-o-Y growth
- Strengthening market position
- #1 prescribed novel hormone therapy in uro-oncology

Assumes 8 month avg. duration on therapy
Source: Internal sales volumes
ENZALUTAMIDE:～575,000 PROSTATE CANCER PATIENTS DIAGNOSED THROUGH THE DISEASE CONTINUUM IN THE G7 MARKETS IN 2015

- First-line hormonal therapy/castration (M0 BCR)
- Second Line hormonal therapy (M0 CRPC)
- M1 CRPC
- M1 HSPC
- Post-chemo

PROSPER
EMBARK
PREVAIL
ARCHES
AFFIRM

Asymptomatic
Non-metastatic
Hormone sensitive
Symptoms
Metastatic
Castration resistant

- Treating Physicians
  - US/EU5/Asia: Urologist, Radiation Oncologist, Urologist, Oncologist
  - Japan: Urologist, Radiation Oncologist, Urologist

*For example, surgery and radiotherapy.
Source: Kohli M, Tindall DJ. Mayo Clin Proc. 2010;85:77-86; CancerMPact; Epiphany; CancerImpact 2015
G7: U.S., EU5 and Japan.
ENZALUTAMIDE: THE CURRENT DEVELOPMENT PROGRAM IS INVESTIGATING ENZALUTAMIDE IN 3 BIOLOGICAL BREAST CANCER SUBTYPES

Women with Metastatic Breast Cancer
Stage IV Incident + Newly Recurrent (2016) 4
US 42,000 | EU 46,000 | JP 11,000

<table>
<thead>
<tr>
<th>Subtype</th>
<th>Phase</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>ER/PR+, HER2-</td>
<td>2</td>
<td>Treated with Hormones</td>
</tr>
<tr>
<td>HER2+, ER/PR+/-</td>
<td>2</td>
<td>Treated with Anti-Her2 +/- Hormones</td>
</tr>
<tr>
<td>TNBC</td>
<td>3</td>
<td>Treated with Cytotoxic agents, Avastin (exUS)</td>
</tr>
</tbody>
</table>

TRIPLE NEGATIVE BREAST CANCER

- Worst prognosis of all Breast Cancer biologic subtypes 2
- Patients diagnosed with TNBC tend to be younger (median age 55-years vs 61 years) 1,3
- ~ 50% of patients test positive to our proprietary diagnostics, which may correlate with enzalutamide responsiveness
- New drugs (PARPS and PD-1s) may enter the market, but expected to demonstrate a benefit in a portion of patients and will leave considerable unmet need


ER: Estrogen receptor, PR: Progesterone receptor, HER2: Human epidermal growth factor receptor 2, TNBC: Triple-negative breast cancer
GILTERITINIB: DEVELOPMENT SEEKS TO ADDRESS KEY PATIENT NEEDS ACROSS FLT3\textsuperscript{MUT} AML

AML Patients (~35,000)\textsuperscript{**}
mFLT3 ~30%

- High-Intensity Induction / Consolidation 60 - 70%*
  - Phase 1 study
- Low-Intensity Chemo 30 - 40%*
  - LACEWING study

- Chemo Consolidation ~50%*
- Transplant ~50%*

- Maintenance 50 - 75%*
  - GOSSAMER study
- Maintenance 50 - 75%*
  - MORPHO study

- Salvage Therapy 50 - 60%*
  - ADMIRAL study

CRITICAL UNMET NEED ADDRESSED BY THE BROAD DEVELOPMENT PROGRAM

- First launch in r/r AML
- Extend indication step by step into earlier lines of treatment
- Create value for FLT3 AML patients


FLT3: FMS-like tyrosine kinase 3, r/r AML: Relapsed or refractory acute myeloid leukemia
EGFR mutations are frequently found in non-small cell lung cancer (NSCLC)

- Most commonly diagnosed cancer worldwide¹
- Accounted for 13% of the global cancer burden with an estimated 1.59 million lung cancer deaths in 2012¹
- Frequency of the EGFR mutations is 10-20% in Caucasians and 30-40% of East Asian NSCLC cases²

- T790M mutations are the primary resistance mechanism for 50%-60% of patients progressing on EGFR TKI’s (erlotinib, gefitinib, afatinib)²
- EGFR T790M mutations are found in ~5% of TKI-naïve NSCLC²

- EGFR TKIs remain the preferred treatment of 1st and 2nd line T790M patients after the launch of PD1/PDL1


**Annual Incidence, EGFRm+³**

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<table>
<thead>
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<tbody>
<tr>
<td>US</td>
<td>44,400</td>
</tr>
<tr>
<td>EU5</td>
<td>42,900</td>
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<tr>
<td>Japan</td>
<td>22,700</td>
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EGFR: Epidermal growth factor receptor
ENFORTUMAB VEDOTIN: UROTHELIAL CANCER IS THE FIFTH MOST COMMON TUMOR TYPE

• Low-grade disease (Ta, Tis, T1) is localized to the urothelium and has not invaded the surrounding muscle
• Intermediate-grade disease (T2, T3a) has invaded the muscle layer of the bladder
• High-grade disease (T3b, T4) has invaded beyond the muscular wall

• Urothelial cancer consists primarily bladder cancer, but also ureter and renal pelvis carcinoma
• Approximately 222,000 new patients are diagnosed annually (US, EU5, JP)
• Patients with early stage disease treated with curative intent, however the recurrence rate is <50%
• Median survival in treated metastatic patients is ~15 months
• Frontline standard of care for metastatic disease is chemotherapy
• PDL-1 and PD-1 inhibitors are emerging as therapeutic options in urothelial cancer, but many patient fail to respond and are in need of improved therapies
• Prescriber group is highly synergistic with our current sales force coverage

Source: 1. SEER; UpToDate; National Cancer Institute; 2. Kantar Cancer Impact 2016
GASTRO ESOPHAGEAL ADENOCARCINOMA REPRESENTS LARGE UNMET NEED WORLDWIDE

- One of the leading causes of cancer death
- Higher incidence in Asia
- First line treatment is combination chemotherapy, or Herceptin (~20% HER2 positive)
- 10-14 months median OS for Stage IV gastric cancer
- Large unmet need remains
- ~50% of the patients is CLDN18.2 positive


CLDN: Claudin
OUR JOURNEY

Turn innovative science into value for patients by delivering paradigm changing treatment options.
CAUTIONARY STATEMENT REGARDING FORWARD-LOOKING INFORMATION

In this material, statements made with respect to current plans, estimates, strategies and beliefs and other statements that are not historical facts are forward-looking statements about the future performance of Astellas. These statements are based on management’s current assumptions and beliefs in light of the information currently available to it and involve known and unknown risks and uncertainties. A number of factors could cause actual results to differ materially from those discussed in the forward-looking statements. Such factors include, but are not limited to: (i) changes in general economic conditions and in laws and regulations, relating to pharmaceutical markets, (ii) currency exchange rate fluctuations, (iii) delays in new product launches, (iv) the inability of Astellas to market existing and new products effectively, (v) the inability of Astellas to continue to effectively research and develop products accepted by customers in highly competitive markets, and (vi) infringements of Astellas’ intellectual property rights by third parties.

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