Corporate Overview

November 2017
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All forward-looking statements contained in this presentation speak only as of the date hereof, and Achillion undertakes no obligation to update any of these statements, except as required by law.
Achillion’s Value Proposition

- Established preliminary PoC with ACH-4471 for the treatment of C3G
  - Significant improvement in proteinuria demonstrating preliminary proof-of-concept (PoC)
  - Significant improvement in complement biomarkers demonstrating target engagement
  - Potential disease-modifying therapy in area where no current treatment available

- Established PoC with ACH-4471 for the treatment of PNH
  - Good tolerability observed at 200mg TID dose after more than six months of treatment
  - Demonstrated both clinical (hemoglobin) and biomarker (C3 fragment deposition on PNH erythrocytes) improvements for balanced ability to address both EVH and IVH in patients

- Factor D platform represents an oral, potentially disease-modifying therapy in multiple diseases

- $353.5 million at 9/30/17 in cash, cash equivalents, and interest receivable to support achievement of value-accreting milestones
Achillion’s Pipeline

**COMPLEMENT FACTOR D PLATFORM**

- **C3 Glomerulopathy (C3G)**
  - **ACH-4471: Factor D Inhibitor** | Oral

- **Paroxysmal Nocturnal Hemoglobinuria (PNH)**
  - **ACH-4471: Factor D Inhibitor** | Oral

- **Immune Complex-mediated Mesangoproliferative Glomerulonephritis (IC – MPGN)**
  - **ACH-4471: Factor D Inhibitor** | Oral

- **AP-mediated diseases**
  - **ACH-5228: Next-Generation Factor D inhibitor** | Oral

- **AP-mediated ophthalmology diseases**
  - **Next-Generation Factor D inhibitors** | Ophthalmic

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fD: Factor D | DMPK: Drug Metabolism/Pharmacokinetics | AP: Alternative Pathway
Mechanism Matters: Factor D Inhibition

FACTOR D
A critical control point specifically within the AP

TRIGGER POINT INHIBITOR
Prevents amplification and modulates downstream complement cascade
FACTOR D INHIBITOR PORTFOLIO
Unlocking the Broader Potential of ACH-4471

PLANS FOR EXPANDING CLINICAL PROGRAM

- C3G & IC-MPGN
  - Phase 2: 14-day dosing
  - Phase 2: 12 month dosing (open-label)
  - Phase 2: 6-month dosing in C3G (double-blind, placebo controlled)
  - Natural history study: Ongoing study conducted by Imperial College of London anticipated to enroll up to 400 patients globally

- PNH
  - Phase 2: Expanding patient enrollment in ongoing monotherapy trial in untreated patients; currently 3 subjects remain on treatment with 200 mg TID
  - Phase 2: Add-on trial to support “switch-strategy” for patients with suboptimal response to eculizumab

We aim to pioneer “best-in-disease” factor D inhibition across multiple indications
FACTOR D INHIBITOR PORTFOLIO
ACH-4471 Summary of Regulatory Status

- Regulatory development planning is focused on US, UK, EU and Japan, in addition to clinical trials conducted in New Zealand and Australia
  - Open U.S. FDA INDs for C3G and PNH

- Scientific advice has been obtained from regulatory agencies including:
  - Design of Phase 2 studies for C3G and PNH
  - Safety monitoring plan
  - Guidance on clinical pharmacology and nonclinical programs needed to support registration
  - Preliminary discussions and feedback on pivotal study endpoints
C3 GLOMERULOPATHY (C3G)  
A Rare Disease with No FDA-Approved Treatment

- C3G includes both **Dense Deposit Disease** (DDD) and **C3 glomerulonephritis** (C3GN)
- Estimated prevalence of **8–12 people affected per million in major markets**
  - Incidence rate of 1–2 per million patients diagnosed with C3G on an annual basis
- There are **no approved treatments** indicated for patients with C3G
  - Non-specific treatment approaches include blood pressure control and broad immunosuppression
- Significant unmet medical need as nearly half of **C3G patients progress to end-stage renal disease**
  - 30-50% progress to ESRD within 10 years
  - Greater than 50% of patients experience disease recurrence post renal transplant, with a 50% chance of graft loss

**DDD AND C3GN IMPACT ON RENAL SURVIVAL**

**Renal Survival (%)**

**Years from Diagnosis**


Barbour et al. (2015); NICE C3G Evidence Summary (2015);
Primary IC-MPGN is believed to be a renal disease associated with AP hyperactivity

Incidence and Prevalence
- Incidence rate similar to that of C3G: 1 to 2 per million in developed areas (e.g. U.S., Europe)
- Accounts for 4 – 10% of primary nephrotic syndrome in children

Age Distribution: May occur at any age
- Primary IC-MPGN occurs most commonly in older children and young adults (7 – 30 years)

Clinical Presentation
- Nephrotic syndrome, gross hematuria, and hypertension
- Variable rate of progression toward renal failure

Estimated 50% of patients progress to end-stage renal disease within 10 years

C3 GLOMERULOPATHY (C3G)
Phase 2 14-day Trial in Patients with C3G or IC-MPGN

**Clinical Trial Design**
- Group 1: 2 patients received ACH-4471 100mg TID x 14 days followed by 7-day taper
- Group 2: Up to 8 additional patients to receive ACH-4471 200mg TID x 14 days followed by 7-day taper

**Criteria**
- Must have clinical and pathologic diagnosis of C3G or IC-MPGN
- C3 must be <50% LLN with C4 >90% LLN
- Estimated glomerular filtration rate cannot be < 45 ml/min/1.73m²

**Outcome Measures**
- Changes in biomarkers of alternative pathway activity (AP) including:
  - C3 fragments and intact C3 levels, Bb, and Ba
- Proteinuria
- Pharmacokinetic profiles

**Clinical Trial Status**
- Group 1: Complete
- Group 2: Plans to enroll 8 additional patients ongoing
Normal Alternative Pathway and Kidney Histology

NORMAL KIDNEY HISTOLOGY

NORMAL GLOMERULAR FUNCTION
- No Proteinuria
- Normal GFR
Overactive Alternative Pathway and C3G Kidney Histology

ABNORMAL GLOMERULAR FUNCTION

- Proteinuria
- Reduced GFR

C3G DISEASE HISTOLOGY
Phase 2 14-day Trial in Patients with C3G or IC-MPGN

Patient A: Baseline Characteristics

Adult male with C3G; diagnosed in March 2017

- Key concomitant medications
  - Prednisolone
  - Mycophenolate
  - Enalapril
  - Spironolactone

- Disease characteristics at baseline prior to first dose:
  - Proteinuria: Albumin to Creatinine ratio (ACR) 259.3 mg/mmol (ref range: 0 – 2.5)
  - Urinalysis: 3+ protein, 1+ blood, 27 RBCs/HPF, 19 WBCs/HPF
  - BP: 126/72
  - eGFR: 91 ml/min/1.73m²
  - Fragment: Intact C3 ratio: 0.1692 (ref range: 0.0085 – 0.0949)
Phase 2 14-day Trial in Patients with C3G or IC-MPGN

Patient A: Significant Reduction in Proteinuria Observed

- Time-dependent decrease observed in proteinuria as measured by ACR
- Greater than 50% reduction achieved during 14 days of treatment
- ACH-4471 demonstrated potential early signs of clinical benefit
Phase 2 14-day Trial in Patients with C3G or IC-MPGN
Patient A: Biomarker Improvements

- ACH-4471 provided significant improvement fragment:intact C3 ratio
- Decrease in C3 fragments is potentially beneficial in C3G
- Mechanistic approach facilitates ability to address root cause of AP-mediated diseases
**Phase 2 14-day Trial in Patients with C3G or IC-MPGN**

**Patient A: Biomarker Improvements**

**EX VIVO Ba FORMATION RELATIVE TO NORMAL HUMAN SERUM**

- **ACH-4471** significantly decreased Ba levels, resulting from cleavage of factor B by factor D.

- Lower levels of Ba suggest lower levels of C3 convertase, resulting in lower levels of C3 fragments.

**Graph:**
- X-axis: Day
- Y-axis: Ex vivo Ba Formation (%)
- Data points showing significant decreases during the dosing phase and increases during the taper phase, followed by a decrease in follow-up.

**Key Points:**
- **Biomarker Improvements**
- **Ex vivo Ba Formation**
- **Dosing**, **Taper**, **Follow-up** phases.
Phase 2 14-day Trial in Patients with C3G or IC-MPGN

Patient A: Additional Biomarker Improvements

- Correction in complement biomarker protein Bb
  - 30% reduction in Bb level as compared to baseline

- To explore local changes in complement proteins in the kidney after dosing with ACH-4471
  - Ratio of urinary Ba:creatinine monitored
  - Observed an 4.4-fold improvement in Ba:creatinine ratio as compared to baseline
  - Ba production is a surrogate for C3 convertase formation

Improvements in multiple complement biomarkers demonstrated inhibition of complement factor D by ACH-4471
Patient B: Baseline Characteristics

Adult male with nephrotic syndrome; diagnosed with C3G in November 2016

Key Concomitant medications:
- Irbesartan
- Spironolactone

Disease characteristics on Day 1 prior to first dose:
- Proteinuria: Albumin to Creatinine ratio (ACR) 580.3 mg/mmol (ref range: 0 – 2.5)
- Urinalysis: 3+ protein, 1+ blood, 42 RBCs/HPF, and 10 WBCs/HPF
- BP: 123/80
- eGFR: 73 ml/min/1.73m²
- Fragment: Intact C3 Ratio: 0.1775 (ref range: 0.0085 – 0.0949)
Phase 2 14-day Trial in Patients with C3G or IC-MPGN

Patient B: Significant Reduction in Proteinuria Observed

ALBUMIN TO CREATININE RATIO OVER TIME

- Time-dependent decrease observed in proteinuria as measured by ACR
  - Greater than 50% reduction achieved during 14 days of treatment
  - ACH-4471 demonstrated potential early signs of clinical benefit
Phase 2 14-day Trial in Patients with C3G or IC-MPGN

Patient B: Biomarker Improvements

- ACH-4471 provided significant improvement fragment:intact C3 ratio
- Decrease in C3 fragments is potentially beneficial in C3G
- Mechanistic approach facilitates ability to address underlying cause of AP-mediated diseases
Phase 2 14-day Trial in Patients with C3G or IC-MPGN

Patient B: Biomarker Improvements

Ex vivo Ba FORMATION RELATIVE TO NORMAL HUMAN SERUM

- ACH-4471 significantly decreased Ba levels, resulting from cleavage of factor B by factor D
- Lower levels of Ba suggest lower levels of C3 convertase, resulting in lower levels of C3 fragments
Patient B: Biomarker Improvements

- Correction in complement biomarker protein Bb
  - Approximately 50% reduction in complement protein Bb as compared to baseline

- To explore local changes in complements in the kidney after dosing with ACH-4471
  - Ratio of urinary Ba:creatinine monitored
  - Observed an 18.6-fold improvement in Ba:creatinine ratio over baseline
  - Ba production is a surrogate for C3 convertase formation

Improvements in multiple complement biomarkers demonstrated inhibition of complement factor D by ACH-4471
Phase 2 14-day Trial in Patients with C3G or IC-MPGN

Summary of Interim Proof-of-Concept Data

- **ACH-4471** was well-tolerated by both patients
  - No SAEs, discontinuations due to AEs, or fevers

- PoC established with 100 mg TID
  - Preliminary POC established with 50% improvement in proteinuria
  - AP inhibition confirmed based on changes in complement biomarkers

- Next steps
  - Evaluating 200 mg TID ACH-4471 in Group 2
  - Actively screening to enroll up to eight patients with C3G or IC-MPGN
Achillion was lead sponsor of externally-led PFDD meeting focused on C3G in August 2017

- First PFDD meeting focused on a renal disease

Led by the National Kidney Foundation and the FDA

Goal is to understand the patient perspective

- PFDD meetings provide an important opportunity to hear directly from patients and caregivers
- Understand the impact of the disease on patients’ daily lives
- Input may inform FDA’s decisions throughout the drug development process

Patient experiences shared at the meeting highlight the unmet need and the urgency to develop transformative therapies
ACH-4471
Phase 2 PNH Three-month / Long-term Extension Trials
Interim Results and Next Steps
PAROXYSMAL NOCTURNAL HEMOGLOBINURIA (PNH)

Factor D and Potential Protection from Intra- / Extra-vascular Hemolysis

PNH RBCs treated with a fD inhibitor may be protected from both intra- and extra-vascular hemolysis.

Adapted from Luzzatto L, Risitano AM, Notaro R. Haematologica 2010;95(4):523–526.
PAROXYSMAL NOCTURNAL HEMOGLOBINURIA (PNH)
A Rare Disease with Limited FDA-Approved Treatment

GOALS FOR INITIAL CLINICAL DEVELOPMENT

- Demonstrate proof-of-mechanism with a highly innovative approach
  - Limits C3 fragment deposition on PNH red blood cells
  - Reduction in plasma Bb levels

- Demonstrate proof-of-concept by showing clinical efficacy
  - Reduction in LDH
  - Increase in hemoglobin
  - Improvement in fatigue score (FACIT score)
  - Increase in PNH RBC clone size

- Elucidate PK/PD
  - Understand plasma concentrations of ACH-4471 necessary for potential efficacy

- Acceptable safety and tolerability profile
Study Status and Interim Results
Phase 2 Trial of ACH-4471 in Untreated PNH Patients

Enrollment: 4 to 12 pts

KEY INCLUSION / EXCLUSION CRITERIA
- PNH clone size ≥ 10%
- Anemia (Hgb < 12 g/dL)
- LDH ≥ 1.5X ULN
- ANC ≥ 1,000/ mm$^3$
- Platelets ≥ 50,000 μL
- Normal ALT
- Alk Phos ≤ 1.5X ULN

Objectives
- Reduction in LDH from baseline
- Improvements in Hgb, FACIT
- Increase PNH RBC clone size

**Part 1**
- **PATIENT C**
  - Classic PNH
  - Total days on therapy: 44
  - Days on 200mg TID: 14

**Part 2**
- **PATIENT D**
  - Classic PNH
  - Total days on therapy: 9
  - Days on 200mg TID: --

**Long-term Extension**
- **PATIENT B**
  - Aplastic Anemia / PNH
  - Total days on therapy: 126
  - Days on 200mg TID: 33
- **PATIENT A**
  - Classic PNH
  - Total days on therapy: 132
  - Days on 200mg TID: 40

**Three-month Dose Finding**
- **PATIENT C**
  - Classic PNH
  - Total days on therapy: 44
  - Days on 200mg TID: 14

Investigator determines clinical response to guide entry into Part 2
Investigator assessment of benefit determines entry into extension trial

Initial dose 100 mg TID. Protocol subsequently amended to allow:
- Newly enrolled patients to start at 150 mg TID
- Intra-patient dose escalation throughout both studies

Interim data reported by Achillion August 8, 2017
Male patient; diagnosed with PNH in 2011 after presentation with dermal thrombosis and hemolytic anemia

- Otherwise healthy with active lifestyle; no transfusion requirements at baseline

### Phase 2 Trial of ACH-4471 in Untreated PNH Patients

#### Patient A: Classic PNH

**Status:**
- Patient remains on 200 mg TID

### Current Value

<table>
<thead>
<tr>
<th>HgB (g/dL)</th>
<th>LDH (U/L)</th>
<th>FACIT</th>
<th>PNH clone size (%)</th>
<th>C3 fragment deposition</th>
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<tbody>
<tr>
<td>14.1</td>
<td>272</td>
<td>49</td>
<td>43</td>
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### Baseline

<table>
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<tr>
<th>Days on therapy</th>
<th>TID</th>
<th>Days</th>
<th>TID</th>
<th>Days</th>
<th>TID</th>
<th>Days</th>
<th>TID</th>
<th>Days</th>
<th>TID</th>
<th>Days</th>
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<tr>
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<td></td>
<td></td>
<td></td>
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<tr>
<td>1848</td>
<td>100mg</td>
<td>13 days</td>
<td>175mg</td>
<td>17 days</td>
<td>200mg</td>
<td>40 days</td>
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<tr>
<td>272</td>
<td>150mg</td>
<td>62 days</td>
<td>TID*</td>
<td></td>
<td></td>
<td>(last labs @ day 20)</td>
<td></td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>

**Interim data reported by Achillion August 8, 2017**

- Median values for LDH and HgB shown.
- Individual data points shown through day 20 for 200 mg TID group.
Phase 2 Trial of ACH-4471 in Untreated PNH Patients

Patient C: Classic PNH

Male patient; diagnosed with PNH in 2003
- Otherwise healthy with active lifestyle; no transfusion requirements at baseline (BL)

<table>
<thead>
<tr>
<th>Days on therapy</th>
<th>150mg TID*</th>
<th>175mg TID**</th>
<th>200mg TID**†</th>
</tr>
</thead>
<tbody>
<tr>
<td>13 days</td>
<td>1272</td>
<td>175 mg</td>
<td>200 mg</td>
</tr>
<tr>
<td>14 days</td>
<td>200 mg</td>
<td>200 mg</td>
<td>200 mg</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Current Value</th>
<th>HgB (g/dL)</th>
<th>LDH (U/L)</th>
<th>FACIT</th>
<th>PNH clone size (%)</th>
<th>C3 fragment deposition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>12.8</td>
<td>551</td>
<td>36</td>
<td>43</td>
<td>Negative</td>
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<tr>
<td>Baseline</td>
<td>11.7</td>
<td>1272</td>
<td>23</td>
<td>24</td>
<td>Negative</td>
</tr>
</tbody>
</table>

Status:
- Patient reported non-compliance after initiation of 200 mg TID dose
- Patient voluntarily withdrew consent for reasons unrelated to safety on day 41

Hgb: hemoglobin  | LDH: lactose dehydrogenase  | TID: three times daily
† Patient began taper on day 41 following withdrawn consent.
** Individual data points shown through day 14 for 200 mg TID group.
* Median values for LDH and HgB shown.
### Phase 2 Trial of ACH-4471 in Untreated PNH Patients

**Patient D: Classic PNH**

- **No history of transfusion-dependence**

**Male patient; diagnosed in 2012 with PNH**

#### Status:
- Patient recently enrolled
- Currently receiving 150 mg TID dose and will be evaluated for intra-patient dose escalation

#### Current Data:

<table>
<thead>
<tr>
<th></th>
<th>HgB (g/dL)</th>
<th>LDH (U/L)</th>
<th>FACIT</th>
<th>PNH clone size (%)</th>
<th>C3 fragment deposition</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Current Value</strong></td>
<td>12.4</td>
<td>504</td>
<td>n/a</td>
<td>n/a</td>
<td>n/a</td>
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<tr>
<td><strong>Baseline</strong></td>
<td>12.0</td>
<td>899</td>
<td>n/a</td>
<td>36</td>
<td>Negative</td>
</tr>
</tbody>
</table>

#### Days on therapy:

- **Baseline**
  - **Days on therapy**
    - 150mg TID:
      - 9 days
    - 175mg TID:
      - 200mg TID:

#### Graph:

- **Hgb: hemoglobin**
- **LDH: lactose dehydrogenase**
- **TID: three times daily**

* Individual data points shown through day 6 for 150 mg TID group.

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**Interim data reported by Achillion August 8, 2017**
Phases 2 Trial of ACH-4471 in Untreated PNH Patients

Patient B: Aplastic Anemia PNH

Male patient diagnosed with AA in 2008; subsequently diagnosed with PNH in 2016
- Treated with ATG, oral prednisone and cyclosporine; ending in 2012
- Baseline marrow function: platelets range 30-60K, ANC 0.7-1.5 and requires Q3-4 weekly RBC transfusions to maintain Hgb ≥ 8 g/dL

Baseline marrow function:

<table>
<thead>
<tr>
<th>Current Value</th>
<th>LDH (U/L)</th>
<th>FACIT</th>
<th>PNH clone size (%)</th>
<th>C3 fragment deposition</th>
</tr>
</thead>
<tbody>
<tr>
<td>9.0</td>
<td>461</td>
<td>31</td>
<td>36</td>
<td>Negative</td>
</tr>
<tr>
<td>7.5</td>
<td>941</td>
<td>22</td>
<td>20</td>
<td>Negative</td>
</tr>
</tbody>
</table>

**Baseline**

- 100mg TID* 13 days
- 150mg TID* 58 days
- 175mg TID* 22 days
- 200mg TID** 33 days (last labs @ day 18)

**Status:**

- Patient remains on 200 mg TID
- Patient continued to receive RBC transfusions during therapy

Interim data reported by Achillion August 8, 2017
Measures of Clinical Efficacy & Safety in PNH

<table>
<thead>
<tr>
<th>Measure</th>
<th>Goal</th>
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<tbody>
<tr>
<td>Lactose dehydrogenase (LDH)</td>
<td>Clinically meaningful reduction in LDH</td>
</tr>
<tr>
<td>Hemoglobin (Hgb)</td>
<td>Stabilize / increase hemoglobin</td>
</tr>
<tr>
<td>C3 fragment deposition</td>
<td>Observe no C3 fragment deposition on PNH RBCs</td>
</tr>
<tr>
<td>Fatigue (FACIT scale)</td>
<td>Improvement over time in objective measures of patient fatigue</td>
</tr>
<tr>
<td>PNH RBC Clone Size</td>
<td>Increase percentage of PNH RBC clones from baseline</td>
</tr>
<tr>
<td>Safety</td>
<td>Favorable tolerability profile</td>
</tr>
</tbody>
</table>

Clinical data generated to date highlight the potential role of factor D inhibition in PNH.
**Objective** Develop an extended release tablet formulation to allow for:
- Optimized trough exposures
- Reduced dosing frequency

ACH-4471 has demonstrated high permeability, with animal and modeling data reporting absorption throughout the GI tract

Human bioavailability study for extended release tablet is planned by year-end 2017
### Near-term ACH-4471 Clinical Development Plan

<table>
<thead>
<tr>
<th>Compound</th>
<th>Indication</th>
<th>Anticipated Next steps</th>
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<tbody>
<tr>
<td>ACH-4471</td>
<td>PNH</td>
<td>Phase 2: Monotherapy</td>
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<tr>
<td></td>
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<td>Ongoing enrollment</td>
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<tr>
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<td>Phase 2: 1H18</td>
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<td>Add-on trial to support “switch strategy” for suboptimal responders</td>
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<td>C3G &amp; IC-MPGN</td>
<td>Phase 2: Ongoing 14-day dosing</td>
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<td>Phase 2: 1H18 12-mon open trial</td>
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<td>Phase 2: 1H18 6-mon randomized (C3G only)</td>
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<tr>
<td>Extended Release Tablet</td>
<td>Phase 1: YE17 Bioavailability study</td>
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