Kyowa Kirin R&D Day

December 10, 2020

Yoshifumi Torii, Ph.D., Executive Officer, Vice President, Head of R&D Division
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These uncertain factors include, but are not limited to, potential risks of the business activities in the pharmaceutical industry in Japan and overseas, intellectual property risks, risk of side effects, regulatory risks, product defect risks, risks of changes to the prices for raw materials, risks of changes to market prices, as well as risks of changes to foreign exchange rates and financial markets.

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Agenda

■ Introduction

■ Topics
  - Projects aiming to launch products within 10-20 years
    Axcelead / InveniAI
  - Projects aiming to launch products within 10 years
    Drug discovery using bispecific antibody technology developed in-house / Collaboration-leveraged drug discovery
  - Projects aiming to launch products within several years
    ME-401 / KHK7791 / RTA 402

■ Summary

■ Q&A Session
Introduction: Challenging the creation of new value in R&D

Initiatives for expanding the opportunities to create value unique to Kyowa Kirin

- **Initiatives and innovation through internal activities + collaborations**
  - Axcelead, InveniAI, SBI Biotech
  - Next generation antibody technology, Nucleic acid therapeutics, Small molecule drug discovery, Regenerative medicine

- **Foster next generation product candidates (global products, etc.)**
  - KHK4083, KW-6356, ME-401, RTA 402, KHK7791...

**Technology strategy**
Utilize next generation antibody technology and diverse modalities to build a platform that will support revolutionary new pharmaceuticals

**Disease strategy**
Utilize the accumulated knowledge and technology to change the lives of patients by providing unique value for UMN
Projects aiming to launch products within 10-20 years
Development of revolutionary small molecule drug discovery technology (collaboration with Axcelead)

- Analysis of potential target drugs
  - Screening technology that imitates the pathology, and molecule design technology
- Diverse drug discovery modality research
  - Fusion with biopharmaceutical research
- R&D experience in the priority categories
  - Appropriately grasp the treatment needs

Small molecule drug discovery infrastructure
- Leading capability in Japan

Vast experience in drug discovery
- Diverse expertise and know-how

Huge volume of drug discovery data
- Huge volume of data and analytical capabilities that enable efficient drug discovery

Strengthen technology-driven drug discovery and strive to create innovative new pharmaceuticals
Promotion of data driven drug discovery (collaboration with InveniAI)

- **InveniAI:**
  - Possesses drug discovery technology that uses AI and machine learning and has experience working with multiple pharmaceutical companies
  - Started collaborating with InveniAI in 2018 aimed at maximizing the value of Kyowa Kirin’s assets

- Expand the collaboration with InveniAI, and initiate efforts to identify new indications and potential target drugs suited to the next generation antibody technology independently developed by Kyowa Kirin

Diverse drug discovery modality research centered on antibody technology

Drug discovery technology that utilizes AI and machine learning

Utilize the strengths of each company, and accelerate “data driven drug discovery” based on data science
Projects aiming to launch products within 10 years
Drug discovery using in-house developed bispecific antibody technology
**In-House Development of Bispecific-Antibody Technology**

**Key points**
- Apply Kyowa Kirin’s unique bispecific-antibody technology for new therapeutic antibodies
- Pursue the development of “first in class” drugs by blending technology with biology

**An Example of Kyowa Kirin’s unique bispecific-antibody technology**

- Selection of linkers derived from Immunoglobulin (Ig) and the common sequence of L Chains
  - Committed to the native sequences*
  - Application of human antibody producing animals*
- Versatility equivalent to wild type IgG
  - High productivity
  - High stability
  - Low antigenicity
- Unique biology based on bivalent x bivalent binding

* Kyowa Kirin’s commitment to the native sequences derived from the research for POTELLIGENT®, COMPLEGENT®, and human antibody producing animals.
Flagship Project

Ideas Based on Research in Biology × Unique Bispecific Antibody Technologies = ‘First-in-Class’ Drugs That Combine Efficacy and Safety

If targeted by conventional antibodies...

Issues related to the limitations of conventional modalities

- Ideal efficacy, if the issue of systemic side effects (safety) is resolved.
- Ideal efficacy, if there is tissue selectivity.
- Ideal pharmacological function, if there is cell selectivity.
Ideas Based on Research in Biology × Unique Bispecific-Antibody Technology = ‘First-in-Class’ Drugs That Combine Efficacy and Safety

Realizing Ideal Efficacy with Safety Through Bispecific-Antibody Technologies

Flagship Project

Vehicle
Chemotherapy
Bispecific-Antibody

Tumor volume [mm³]

PDX* model
N=5
Mean ± SE
*Patient derived xenograft

Administration of Drug
Expansion of the Bispecific-Antibody Project

Type I

Combination of the targets
\((A, B \ldots) \times (X, Y \ldots)\)

Selection of the types
(Type I, II)

Challenges exceeding
the limitations of
conventional modalities

Type II
Collaboration-leveraged drug discovery
In 2016 Kyowa Kirin concluded an exclusive licensing agreement with SBI Biotech Co., Ltd. covering development, manufacturing and sales worldwide for SBI-9674, an antibody drug for autoimmune diseases.

* pDC: Plasmacytoid dendritic cell

All three pathways can be managed by targeting pDCs

New antibody that targets pDCs (in-licensed from SBI Biotech)

- pDCs are known to be the main source of type 1 interferons (Type 1 IFN), which are the cause of various autoimmune diseases
- Type 1 IFN produced from pDCs coordinate immune functions through a variety of mechanisms (see the illustration below)

Source: Nat Rev Rheumatol. 2010
New antibody that targets pDCs (in-licensed from SBI Biotech)

- At this year’s ACR (American College of Rheumatology) convergence, the results of the Phase 2 study of the developed compound that targets pDCs (Anti-BDCA2 mAb) in patients with SLE/CLE were reported.

**ABSTRACT NUMBER: 0935 • ACR Convergence 2020**

**Efficacy and Safety Results from a Phase 2, Randomized, Double-Blind Trial of BIIB059, an Anti-Blood Dendritic Cell Antigen 2 Antibody, in SLE**

Richard Furie1, Ronald van Vollenhoven1, Kenneth Kalunian2, Sandra Navarro1, Juanita Romero-Diaz1, Victoria Werth3, Xiaoli Huang2, Hux Carroll1, Adam Meyers4, Cristina Musselli5, Catherine Barby6 and Nathalie Franchimont.7, 1Zucker School of Medicine at Hofstra/Northwell, Great Neck, NY, 2Department of Rheumatology, Amsterdam Rheumatology and Immunology Center, Amsterdam, Netherlands, 3University of California San Diego, La Jolla, CA, 4University of Santo Tomas, Manila, Philippines, 5Instituto Nacional de Ciencias Medicas y Nutricion Salvador Zubiran, Mexico City, Mexico, 6University of Pennsylvania and the Michael J. Crescenz VA Medical Center, Philadelphia, PA, 7Biogen, Cambridge, MA, 8Biogen, Cambridge, MA, Biogen, Baar, Switzerland

**Background/Purpose:** Type I interferons (IFN-I), inflammatory mediators principally produced by plasmacytoid dendritic cells (pDCs), components of the innate immune system, have been implicated in the...

**ABSTRACT NUMBER: 0986 • ACR Convergence 2020**

**BIIB059, a Humanized Monoclonal Antibody Targeting Blood Dendritic Cell Antigen 2 on Plasmacytoid Dendritic Cells, Shows Dose-Related Efficacy in a Phase 2 Study in Participants with Active Cutaneous Lupus Erythematosus**

Victoria Werth1, Richard Furie1, Juanita Romero-Diaz1, Sandra Navarro1, Kenneth Kalunian2, Ronald van Vollenhoven1, Filipa Nyberg1, Benjamin Kaffenberger1, Seira Shekhi1, Goren Radunovic1, Xiaoli Huang2, Hux Carroll1, Francois Gaudreault1, Adam Meyers4, Catherine Barby6, Cristina Musselli5 and Nathalie Franchimont.7, 1University of Pennsylvania and the Michael J. Crescenz VA Medical Center, Philadelphia, PA, 2Zucker School of Medicine at Hofstra/Northwell, Great Neck, NY, 3Instituto Nacional de Ciencias Medicas y Nutricion Salvador Zubiran, Mexico City, Mexico, 4University of Santo Tomas, Manila, Philippines, 5University of California San Diego, La Jolla, CA, 6Department of Rheumatology, Amsterdam Rheumatology and Immunology Center, Amsterdam, Netherlands, 7Karolinska University Hospital, Stockholm, Sweden, 8Ohio State University, Columbus, OH, 9Division of Rheumatology, Allergy and Immunology, University of North Carolina, Chapel Hill, NC, 10Institute of Rheumatology, University of Belgrade, Belgrade, Serbia, 11Biogen, Cambridge, MA, 12Biogen, Cambridge, MA, 13Biogen, Baar, Switzerland

**Background/Purpose:** No approved targeted therapies have been developed for cutaneous lupus erythematosus (CLE), a disfiguring autoimmune disease that severely impairs quality of life.1 BIIB059 is...

Source: ACR convergence 2020 Abstract

** ✓ pDCs are involved in the pathology of autoimmune diseases incl. SLE/CLE ✓ Expectations are increasing for a therapeutic agent that targets pDCs**
Projects aiming to launch products within several years
ME-401: Results of a Phase 1b Study in B-cell Malignancies

- **Treatment:** ME-401 monotherapy or in combination with rituximab administered in IS

  - Daily dosing x2 cycles
  - Daily x1 week
  - No therapy x3 weeks

- **Overall Response Rate (ORR) and Adverse Events of Special Interest (AESI)**

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Evaluable Patients</th>
<th>ORR n (%)</th>
<th>Adverse Event of Special Interest (AESI)</th>
<th>Grade ≥ 3</th>
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</thead>
<tbody>
<tr>
<td>FL</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>By treatment group</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ME-401 monotherapy</td>
<td>17</td>
<td>13 (76%)</td>
<td>Diarrhea or colitis</td>
<td>2 (3.5%)</td>
</tr>
<tr>
<td>ME-401 + rituximab</td>
<td>19</td>
<td>17 (89%)</td>
<td>Diarrhea</td>
<td>2 (3.5%)</td>
</tr>
<tr>
<td></td>
<td>36</td>
<td>30 (83%)</td>
<td>Colitis</td>
<td></td>
</tr>
<tr>
<td>CLL/SLL</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>By treatment group</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ME-401 monotherapy</td>
<td>3</td>
<td>3 (100%)</td>
<td>Rash, all types</td>
<td>1 (1.8%)</td>
</tr>
<tr>
<td>ME-401 + rituximab</td>
<td>6</td>
<td>5 (83%)</td>
<td>ALT/AST elevation</td>
<td>1 (1.8%)</td>
</tr>
<tr>
<td></td>
<td>9</td>
<td>8 (89%)</td>
<td>Stomatitis</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Pneumonia/Infectious pneumonitis</td>
<td>0*</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Non-Infectious pneumonitis</td>
<td>1 (1.8%)</td>
</tr>
</tbody>
</table>

ME-401 administered on an intermittent schedule showed a high response rate and was generally well tolerated in r/r FL and CLL/SLL

* A patient with grade 5 COVID-19 pneumonia in Cycle 15

IS: Intermittent schedule, 1 cycle: 28 days, ORR: Overall response rate, FL: Follicular Lymphoma, CLL/SLL: Chronic Lymphocytic Leukemia/Small Lymphocytic Lymphoma, r/r: Relapsed/Refractory
ME-401: Global Phase 2 TIDAL* Study in r/r Follicular Lymphoma or Marginal Zone Lymphoma

- Subjects: Patients with r/r FL after failure of at least two systemic therapies
- Treatment: ME-401 monotherapy administered on an intermittent schedule

- Target number of patients: 180
- Study location: US, Europe, Oceania, South Korea, Taiwan
- Primary endpoint: Overall response rate
- The results of TIDAL are intended to be submitted to the U.S. Food and Drug Administration (FDA) to support accelerated approval of the marketing application

* TIDAL: Trials of PI3K Delta in Non-Hodgkin’s Lymphoma
ME-401: Japanese Phase 2 K02 Study in r/r Indolent B-cell NHL

- **Subjects**: Patients with r/r indolent B-cell NHL* after failure of at least two systemic therapies
- **Treatment**: ME-401 monotherapy administered on an intermittent schedule

  - **Daily dosing x2 cycles**
  - **Cycles 1 and 2**
  - **Daily x1 week**
  - **No therapy x3 weeks**
  - **Intermittent Schedule on Cycles ≥3**

- **Target number of patients**: 60
- **Study location**: Japan
- **Primary endpoint**: Overall response rate
- **Plan to consider submission of the marketing application to the Japanese authorities after completion of the K02 study**

* Excluding Small lymphocytic lymphoma (SLL), lymphoplasmacytic lymphoma (LPL), and Waldenström’s macroglobulinemia (WM)
KHK7791 / tenapanor

**Mechanism of action**
- The primary uptake route of phosphorous is passive transport between cell membranes
- Tenapanor inhibits NHE3 and thereby inhibits the uptake of sodium (Na⁺)
- At the same time, the concentration of protons (H⁺) in the cells increases
- The increased intracellular proton concentration tightens the epithelial cell junctions, which regulates the uptake of phosphorous in the digestive tract, and thereby inhibits the uptake of phosphorous

**Features**
- Unlike existing phosphorous binders, the novel mechanism of action makes it a first-in-class phosphorous absorption inhibitor
Objectives
- Explain that hyperphosphatemia can be treated with a single agent, KHK7791
- Explain the validity of using KHK7791 for patients who are starting treatment for hyperphosphatemia

Objectives
- Satisfy the unmet needs that cannot be controlled with existing phosphate binders
- Confirm the efficacy by coadministration of KHK7791 with phosphate binders

Objectives
- Explain the benefit of switching from existing phosphate binders, taking advantage of the small pill size and quantity
- Quantitatively confirm the reduction in phosphate binder pill burden
KHK7791: PB switching study design

- Open-label, single-arm, phosphate binder (PB) switch study for hyperphosphatemia patients on hemodialysis

  - **Screening** 92 subjects
  - **Pre-enrollment** 83 subjects
  - **Enrollment** 67 subjects
  - **Phosphate binder (PB) + KHK7791**

**Enrollment criteria**
- Serum phosphorus level (s-P): 3.5-7.0 mg/dL

**Observation period** (3 weeks)

**Evaluation period** (26 weeks)

**Primary endpoint**: Achievement of 30% decrease in the mean total tablet number (PB+KHK7791) compared to baseline PB
KHK7791: PB switching study results

- **Primary endpoint**

<table>
<thead>
<tr>
<th>Achievement ratio</th>
<th>P value [95%CI]</th>
</tr>
</thead>
<tbody>
<tr>
<td>30% decrease</td>
<td>71.6% (48/67)</td>
</tr>
</tbody>
</table>

- **Mean change in total PB tablets over time**

- **Mean change in s-P over time**

More than 70% patients achieved a 30% decrease in the total number of PB tablets compared to the baseline.
A phase 2, randomized, double-blind, placebo-controlled, dose-finding study of
KHK7791 in hyperphosphatemia patients on hemodialysis

ClinicalTrials.gov Identifier: NCT03864458

Screening

Pre-enrollment

Enrollment Randomization

1:1:1:1:1

Washout of phosphate binders

1st washout period

Maximum 3 weeks

KHK7791 30mg BID

KHK7791 10mg BID

KHK7791 5mg BID

KHK7791 30mg BID dose-titration

Placebo BID

Treatment period

6 weeks

2nd washout period

3 weeks

Enrollment criteria

(Pre-enrollment) Serum phosphorus level : 3.5-6.0 mg/dL
(Enrollment) Serum phosphorus level : Increased by ≥1.0 mg/dL from screening to 6.1-9.9 mg/dL

Primary endpoint: Changes in serum phosphorus levels from baseline values at Week 6

Sites : 31
Target patients : 200

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**KHK7791: Dose-finding study results**

### Primary endpoint

<table>
<thead>
<tr>
<th>Week 6 Serum phosphorus level (mg/dL)</th>
<th>Placebo</th>
<th>KHK7791 5 mg</th>
<th>KHK7791 10 mg</th>
<th>KHK7791 30 mg</th>
<th>KHK7791 30 mg dose-titration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean±SD</td>
<td>0.6±1.6</td>
<td>-0.9±1.7</td>
<td>-1.4±1.5</td>
<td>-1.9±1.2</td>
<td>-2.0±1.1</td>
</tr>
<tr>
<td>Median</td>
<td>0.4</td>
<td>-1.0</td>
<td>-1.5</td>
<td>-2.1</td>
<td>-2.0</td>
</tr>
<tr>
<td>Difference</td>
<td>-</td>
<td>-1.6</td>
<td>-2.0</td>
<td>-2.6</td>
<td>-2.6</td>
</tr>
<tr>
<td>95% CI</td>
<td>-</td>
<td>[-2.3,-0.9]</td>
<td>[-2.7,-1.3]</td>
<td>[-3.2,-2.0]</td>
<td>[-3.2,-2.0]</td>
</tr>
<tr>
<td>p-value</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
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### Drug-related TEAEs, discontinued

<table>
<thead>
<tr>
<th>N=41</th>
<th>N=42</th>
<th>N=41</th>
<th>N=42</th>
<th>N=41</th>
<th>N=42</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drug-related TEAEs</td>
<td>7</td>
<td>(17.1)</td>
<td>22</td>
<td>(52.4)</td>
<td>28</td>
</tr>
<tr>
<td>[Gastrointestinal disorders]</td>
<td>5</td>
<td>(12.2)</td>
<td>22</td>
<td>(52.4)</td>
<td>28</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>4</td>
<td>(9.8)</td>
<td>21</td>
<td>(50.0)</td>
<td>27</td>
</tr>
</tbody>
</table>

**Severity of diarrhea**

<table>
<thead>
<tr>
<th>N=41</th>
<th>N=42</th>
<th>N=41</th>
<th>N=42</th>
<th>N=41</th>
<th>N=42</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>4</td>
<td>21</td>
<td>25</td>
<td>28</td>
<td>24</td>
</tr>
<tr>
<td>Moderate</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td>Severe</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

**KHK7791 significantly decreased serum phosphorus levels from baseline compared with the placebo and decreased serum phosphorus levels in a dose-dependent manner.**
KHK7791: Add-on study design

- A phase 2, randomized, double-blind, placebo-controlled, phosphate binder-combination study of KHK7791 in hyperphosphatemia patients on hemodialysis

ClinicalTrials.gov Identifier: NCT03864445

- Enrollment criteria
  
  (Pre-enrollment, Enrollment) Serum phosphorus level: 6.1-9.9 mg/dL

- Primary endpoint: Changes in serum phosphorus levels from baseline values at Week 6

- Treatment of phosphate binders
  
  Sites: 9
  Target patients: 40

- KHK7791 30 mg BID dose-titration + phosphate binder
  
  Placebo BID + phosphate binder
KHK7791: Add-on study results

- Primary endpoint and target achievement ratio of serum phosphorus level (3.5～6.0 mg/dL)

<table>
<thead>
<tr>
<th>Week 6</th>
<th>Serum phosphorus level (mg/dL)</th>
<th>Mean±SD</th>
<th>Placebo N=24</th>
<th>KHK7791 N=23</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median</td>
<td>-0.2</td>
<td>-2.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Difference</td>
<td>-</td>
<td>-2.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>95% CI</td>
<td>-</td>
<td>[-2.9,-1.3]</td>
<td></td>
<td></td>
</tr>
<tr>
<td>p-value</td>
<td>-</td>
<td>&lt;0.001</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Week 6</th>
<th>Target achievement proportion</th>
<th>%</th>
<th>(n/N)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Placebo N=24</td>
<td>37.5</td>
<td>(9/24)</td>
</tr>
<tr>
<td></td>
<td>KHK7791 N=23</td>
<td>87.0</td>
<td>(20/23)</td>
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</table>

- Drug-related TEAEs, discontinued

<table>
<thead>
<tr>
<th>Drug-related TEAEs</th>
<th>Placebo N=24</th>
<th>%</th>
<th>KHK7791 N=23</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drug-related TEAEs</td>
<td>N=24</td>
<td></td>
<td>N=23</td>
<td></td>
</tr>
<tr>
<td>[Gastrointestinal disorders]</td>
<td>2</td>
<td>(8.3)</td>
<td>16</td>
<td>(69.6)</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>2</td>
<td>(8.3)</td>
<td>16</td>
<td>(69.6)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Severity of diarrhea</th>
<th>Placebo</th>
<th></th>
<th>KHK7791</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>2</td>
<td></td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>Moderate</td>
<td>0</td>
<td></td>
<td>7</td>
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</tr>
<tr>
<td>Severe</td>
<td>0</td>
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</tbody>
</table>

Adding on KHK7791 resulted in a significant decrease in serum phosphorus levels compared with the placebo.
RTA 402
RTA 402 / Bardoxolone methyl

RTA 402 (Bardoxolone methyl) activates the Keap1-Nrf2 pathway, which is an oxidative stress response system.

RTA 402: CARDINAL Phase 3 for Alport Syndrome patients

What is Alport Syndrome?
Alport Syndrome is a kidney disease caused by genetic mutations in either the type IV collagen α3, α4 or α5 chain. In Japan, Alport Syndrome is certified as designated intractable diseases and specific pediatric chronic diseases. Severe cases have been reported to progress to end-stage kidney disease in the late teens and early 20s. Currently there is no approved treatment for Alport Syndrome as an indication.

- Number of subjects: 157 (Placebo: 80, RTA 402: 77)
- Subjects: 12-70 years, eGFR: 30-90 mL/min/1.73m²
- Primary efficacy endpoint: Change in eGFR from baseline at Week 48 and Week 100
- Key secondary endpoint: Change in eGFR from baseline at Week 52 and Week 104 (4 weeks after withdrawal of drug)
- Region: US, JP, EU, AU

The 2-year study period was completed, and Reata announced the year 2 results on Nov 9th.

[Diagram of study design]

1-year results were published in Reata’s 3Q financial results presentation last November. Both primary/key secondary endpoints have been met.
Both primary/key secondary endpoints were met. The results also show a favorable safety profile.
Based on the current results, KKC also plans to submit a marketing approval application for the indication of Alport Syndrome in Japan.
Summary
Summary (1): Challenging the creation of new value in R&D

Initiatives for expanding the opportunities to create value unique to Kyowa Kirin

- **Initiatives and innovation through internal activities + collaborations**
  - Axcelead, InveniAI, SBI Biotech
  - **Next generation antibody technology**, Nucleic acid therapeutics, Small molecule drug discovery, Regenerative medicine

- **Foster next generation product candidates (global products, etc.)**
  - KHK4083, KW-6356, ME-401, RTA 402, KHK7791...

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**Technology strategy**

Utilize next generation antibody technology and diverse modalities to build a platform that will support revolutionary new pharmaceuticals

**Disease strategy**

Utilize the accumulated knowledge and technology to change the lives of patients by providing unique value for UMN
Summary (2): Challenging the creation of new value in R&D

R&D spirits

- Be the Best in Science (サイエンスで負けない)
- Passion for Innovation (イノベーションへの情熱)
- Every Challenge, a Step to Success (挑戦なくして成功なし)
- This is me. This is our team. (多様な個性が輝くチームになろう)
Q&A Session
Thank you.