Overview of Chugai Diabetes

CHUGAI PHARMACEUTICAL CO., LTD.
Department Manager
Project Management Dept.
Hisanori Takanashi
December 7, 2009
Forward-Looking Statements

This presentation may include forward-looking statements pertaining to the business and prospects of Chugai Pharmaceutical Co., Ltd. (the “Company”). These statements reflect the Company’s current analysis of existing information and trends. Actual results may differ from expectations based on risks and uncertainties that may affect the Company’s businesses.
Contents

1. Overview of diabetes treatment in Japan
2. Challenges with existing treatment
3. Chugai’s activities in diabetes
Diabetes Population

Million patients

5 EU states: UK, France, Germany, Italy and Spain

Epi Database (MattsonJack / Synix Co.)
Oral Agents for Type 2 Diabetes in Japan

- Decreased insulin sensitivity
- Decreased insulin release
- Elevated blood glucose

**Insulin sensitizer**
- Biguanide
- Thiazolidinedione

**Insulin secretagogue**
- Sulfonylurea

**Postprandial glucose controller**
- α-glucosidase inhibitor
- Glynide
Current Treatment Algorithm in Japan

Treatment guidelines in Japan:
• Oral treatment should start if a patient cannot achieve sufficient glucose control after lifestyle change (diet and exercise).
• In choosing drugs, consider clinical factors like disease status, complications and drug mechanism.
• Drug administration should start at low dose. Increase dosage depending on patients’ clinical status. Before starting SU, patients should be well informed of what should be done in case of hypoglycemia.
• Other treatment approach including combination therapy should be considered if a patient cannot achieve target glucose level 3 months after treatment.
Diabetes Market in Japan

NHI reimbursement price basos

The scope of the market is defined by Chugai.

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Contents

1. Overview of diabetes treatment in Japan
2. Challenges with existing treatment
3. Chugai’s activities in diabetes
Previously-untreated patients

Effect Duration (ADOPT)

Existing treatment cannot stop disease to progress over time

**Glucose Control and HbA1c**

**Glucose control**

Only 34% of patients with diabetes achieves desirable status

HbA1c (%)
- < 6.5
- 6.5 ≤ < 8.0
- 8.0 ≤

![Pie chart showing glucose control]

- 20% < 6.5
- 34% 6.5 ≤ < 8.0
- 46% 8.0 ≤

# of patients
- type 1: 793
- type 2: 16,141

**HbA1c and microvascular complications**

![Graph showing incidence per 1000 person years (%)]


# Profiles of Existing Treatments

## Treatment satisfaction

<table>
<thead>
<tr>
<th></th>
<th>Insulin sensitizer</th>
<th>Insulin secretagogue</th>
<th>Others</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>SU</td>
<td>GLI</td>
<td>BIG</td>
</tr>
<tr>
<td><strong>HbA1c reduction</strong></td>
<td>+++</td>
<td>+</td>
<td>++</td>
</tr>
<tr>
<td><strong>Duration of glucose control</strong></td>
<td>-</td>
<td>-</td>
<td>±</td>
</tr>
<tr>
<td><strong>Effect on weight</strong></td>
<td>↑</td>
<td>↑</td>
<td>~</td>
</tr>
<tr>
<td><strong>Hypoglycemia risks</strong></td>
<td>++</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td><strong>Edema</strong></td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td><strong>Gastrointestinal disorder</strong></td>
<td>-</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td><strong>Contraindication</strong></td>
<td>-</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td><strong>Ease of administration</strong></td>
<td>QD</td>
<td>TID</td>
<td>TID</td>
</tr>
</tbody>
</table>

QD: Once daily, TID: Three times daily
Contents

1. Overview of diabetes treatment in Japan
2. Challenges with existing treatment
3. Chugai’s activities in diabetes
Entry into Diabetes Area

- High unmet medical needs
  - Increasing number of patients
  - Challenges with existing treatments (glucose control sustainability)

- Enhanced R&D capabilities through the alliance with Roche
  - Sharing of research infrastructure
  - Global development through group network
  - Synergies with Roche Diagnostics

- Strategies
  - Epogen and other renal franchise - increase in chronic renal failure in patients with underlying diabetes
  - Presence in GP market
  - Euglucon business
Target Organs for New Treatments

Pancreas:
- glucose-dependent insulin secretion ↑
- glucose-dependent glucagon secretion ↓
- enhance β-cell regeneration
  - GLP-1 agonist
  - DPP-4 inhibitor
  - Other secretagogue
  - Glucagon receptor antagonist

Central nervous system:
- food intake ↓
  - CB1 antagonist

Fat:
- glucose uptake ↑
- insulin sensitivity ↑
  - GLP-1 agonist
  - DPP-4 inhibitor
  - Other secretagogue

Skeletal muscles:
- glucose uptake ↑
- insulin sensitivity ↑
  - 11β-HSD-I

Liver:
- gluconeogenesis ↓
- glucose uptake ↑
  - Glucokinase activator

Kidney:
- glucose uptake ↓
  - SGLT2 inhibitor
# Profiles of New Treatments

## Treatment satisfaction

<table>
<thead>
<tr>
<th></th>
<th>Insulin sensitizer</th>
<th>Insulin secretagogue</th>
<th>Others</th>
</tr>
</thead>
<tbody>
<tr>
<td>SU</td>
<td>+</td>
<td>++</td>
<td>+</td>
</tr>
<tr>
<td>GLI</td>
<td>++</td>
<td>+++</td>
<td>+++</td>
</tr>
<tr>
<td>DPP-4</td>
<td>++</td>
<td>++</td>
<td>+</td>
</tr>
<tr>
<td>GLP-1</td>
<td>++</td>
<td>+++</td>
<td>+++?</td>
</tr>
<tr>
<td>BIG</td>
<td>±</td>
<td>±</td>
<td>±</td>
</tr>
<tr>
<td>TZD</td>
<td>±</td>
<td>±</td>
<td>±</td>
</tr>
<tr>
<td>AGI</td>
<td>±</td>
<td>±</td>
<td>±</td>
</tr>
<tr>
<td>SGLT2</td>
<td>±</td>
<td>±</td>
<td>±</td>
</tr>
</tbody>
</table>

- **HbA1c reduction**: +++ for SU, + for GLI, ++ for DPP-4, ++++ for GLP-1, ++ for BIG, +++ for TZD, + for AGI, and +++? for SGLT2.
- **Duration of glucose control**: - for SU, - for GLI, ? for DPP-4, ? for GLP-1, ± for BIG, ± for TZD, ± for AGI, and ? for SGLT2.
- **Effect on weight**: ↑ for SU, ↑ for GLI, ~ for DPP-4, ↓ for GLP-1, ~ for BIG, ↑ for TZD, ~ for AGI, and ↓ for SGLT2.
- **Hypoglycemia risks**: ++ for SU, + for GLI, - for DPP-4, - for GLP-1, - for BIG, - for TZD, - for AGI, and - for SGLT2.
- **Edema**: - for SU, - for GLI, - for DPP-4, - for GLP-1, - for BIG, - for TZD, + for AGI, and + for SGLT2.
- **Gastrointestinal disorder**: - for SU, - for GLI, - for DPP-4, - for GLP-1, - for BIG, - for TZD, + for AGI, and + for SGLT2.
- **Contraindication**: - for SU, - for GLI, - for DPP-4, - for GLP-1, - for BIG, - for TZD, + for AGI, and + for SGLT2.
- **Ease of administration**: QD for SU, TID for GLI, QD for DPP-4, Injection for GLP-1, TID for BIG, QD for TZD, TID for AGI, and QD for SGLT2.

QD: Once daily, TID: Three times daily.
Chugai’s Approach

Challenges with existing therapies

● Unsustainable efficacy
● Dose limiting side effects (hypoglycemia, weight gain, edema, GI disorder)

Expectations for new therapies

● Sustained control of blood glucose
● No hypoglycemic risks
● Weight neutral/weight loss
● Disease modifying effect (β-cell protection)

Aim at first-in-class and best-in-class with focus on: insulin secretagogues and disease-modifying candidates
To provide new treatment option

CSG452
Chugai

GLP-1 agonist
11 beta HSD inhibitor
11 beta HSD inhibitor (2)
Y2R pept. agonist
BHT-3021 (T1DM)

Roche

Innovation
to meet
unmet medical needs

multiple research projects

We accelerate our R&D efforts

to better serve stakeholders.
Overview of taspoglutide

CHUGAI PHARMACEUTICAL CO., LTD.
Project Management Dept.
Takaki Koga

December 7, 2009
Contents

1. Biological actions of GLP-1 and its agonists under clinical developments

2. Profile of taspoglutide and its development status
Biological Actions of GLP-1 and its Application to Therapeutic Agents

Blood glucose lowering effect through glucose dependent stimulation of insulin secretion (incretin effect)

- Glucose conc. dependent
  - Insulin secretion stimulation
  - Glucagon secretion inhibition
- Gastric emptying delay
- Appetite suppression
- Body weight reduction
- Beta cell protection/regeneration

**Diadvantage: Short half life**

T1/2 = 2 min.

Drucker, Cell Metabolism 2006;3:153-165

Long acting analogues
- Exenatide
- Liraglutide
- Taspoglutide

Degradation Enzyme inhibitors
- Sitagliptin
- Vildagliptin
- Alogliptin
# GLP-1 Agonists vs. DPP-4 Inhibitors

**GLP-1: stronger blood glucose control and weight loss**

<table>
<thead>
<tr>
<th></th>
<th>GLP-1 agonists</th>
<th>DPP-4 inhibitors</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>MOA</strong></td>
<td>Pharmacological GLP-1 receptor potentiation</td>
<td>Enhancement of intrinsic incretin actions</td>
</tr>
<tr>
<td><strong>Mode of administration</strong></td>
<td>Injection</td>
<td>Oral</td>
</tr>
<tr>
<td><strong>Glucose lowering</strong></td>
<td>HbA1c reduction &gt;1%</td>
<td>HbA1c reduction &lt;1%</td>
</tr>
<tr>
<td><strong>Body weight</strong></td>
<td>Reduction</td>
<td>Neutral</td>
</tr>
<tr>
<td><strong>Adverse events</strong></td>
<td>Nausea, Vomiting</td>
<td>Highly tolerable</td>
</tr>
<tr>
<td><strong>Hypoglycemic events</strong></td>
<td>Noted when administered with SU</td>
<td>None</td>
</tr>
</tbody>
</table>
# GLP-1 Agonists under Development in Japan

<table>
<thead>
<tr>
<th>Company</th>
<th>description</th>
<th>Administration</th>
<th>Status</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exenatide</td>
<td>Amylin/Lilly / Exendin-4 / solution</td>
<td>BID</td>
<td>NDA</td>
</tr>
<tr>
<td>Liraglutide</td>
<td>Novo Nordisk / GLP-1 analogue</td>
<td>QD</td>
<td>NDA</td>
</tr>
<tr>
<td>Lixenatide</td>
<td>Sanofi-Aventis / GLP-1 and Exendin-4 analogue</td>
<td>QD</td>
<td>P3</td>
</tr>
<tr>
<td>Exenatide LAR</td>
<td>Amylin / Lilly / Alkermes / Exendin-4 / polymer-based microspheres</td>
<td>QW</td>
<td>P3</td>
</tr>
<tr>
<td>Taspoglutide/ITM-077</td>
<td>Roche/Ipsen/Chugai/Teijin / GLP-1 analogue / low pH solution containing Zn</td>
<td>QW</td>
<td>P2</td>
</tr>
<tr>
<td>Albigultide</td>
<td>GSK / GLP-1-albumin fusion</td>
<td>QW</td>
<td>P1/2</td>
</tr>
<tr>
<td>LY2189265</td>
<td>Lilly / Fc-fusion protein GLP-1 analog</td>
<td>QW</td>
<td>P2</td>
</tr>
</tbody>
</table>

QD: Once daily, BID: Twice daily, QW: Once weekly
Contents

1. Biological actions of GLP-1 and its agonists under clinical developments
2. The profile of Taspoglutide and development status
Converting Native GLP-1 into Taspoglutide

Native GLP-1

DPP-IV cleavage site

Serine protease cleavage site

Taspoglutide

Aib<sup>8,35</sup> substitutions

The active form of native GLP-1 is rapidly degraded by peptidases
Aminoisobutylic acid (Aib) substitutions block enzymatic degradations
Agonist activity comparable with native form
Once weekly dosing supported by zinc-based formulation
Overseas Phase 2 study

- once weekly or once in two weeks injections, metformin combination -

<table>
<thead>
<tr>
<th>Screening (Max 3 weeks)</th>
<th>Randomization (Baseline)</th>
<th>Taspo/PBO treatment (8 weeks)</th>
<th>Follow-up (4 weeks)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metformin ≥1.5g/day</td>
<td>+ Taspoglutide 5 mg once/w</td>
<td>Metformin</td>
<td></td>
</tr>
<tr>
<td></td>
<td>+ Taspoglutide 10 mg once/w</td>
<td>Metformin</td>
<td></td>
</tr>
<tr>
<td></td>
<td>+ Taspoglutide 20 mg once/w</td>
<td>Metformin</td>
<td></td>
</tr>
<tr>
<td></td>
<td>+ Taspoglutide 10 mg once/2ws</td>
<td>Metformin</td>
<td></td>
</tr>
<tr>
<td></td>
<td>+ Taspoglutide 20 mg once/2ws</td>
<td>Metformin</td>
<td></td>
</tr>
<tr>
<td></td>
<td>+ Placebo once/w</td>
<td>Metformin</td>
<td></td>
</tr>
</tbody>
</table>

Day 1 (1st injection)  50 patients per treatment group
Changes in HbA1c:
Significant Reductions in Only Eight Weeks

**Once weekly**
- 5 mg
- 10 mg
- 20 mg

**Once in 2 weeks**
- 10 mg
- 20 mg

* Significant change from Placebo (%)

*\( p < 0.0001 \)
Changes in Body Weight: Significant Loss in Only Eight Weeks

* p = 0.0035, **p < 0.0001, *** p = 0.0083
# Adverse Events

<table>
<thead>
<tr>
<th></th>
<th>Once weekly</th>
<th>Once in two weeks</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Placebo n=49</td>
<td>5mg n=50</td>
</tr>
<tr>
<td>Nausea, n (%)</td>
<td>3 (6)</td>
<td>11 (22)</td>
</tr>
<tr>
<td>Diarrhea, n (%)</td>
<td>4 (8)</td>
<td>4 (8)</td>
</tr>
<tr>
<td>Vomiting, n (%)</td>
<td>2 (4)</td>
<td>2 (4)</td>
</tr>
<tr>
<td>Headache, n (%)</td>
<td>3 (6)</td>
<td>1 (2)</td>
</tr>
<tr>
<td>Decreased appetite, n (%)</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Dyspepsia, n (%)</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Abdominal distension, n (%)</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

_Gastrointestinal effects most frequent → titrated administration adopted in Phase III studies for reduction_
## Ongoing Phase 3 Studies by Roche

<table>
<thead>
<tr>
<th>Study name</th>
<th>Background medications</th>
<th>Comparators</th>
<th>N</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>T-emerge 1</td>
<td>Diet &amp; exercise</td>
<td>Placebo</td>
<td>330</td>
<td>Taspoglutide demonstrated superior HbA1c reduction versus placebo.</td>
</tr>
<tr>
<td>T-emerge 2</td>
<td>Metformin, TZD, Metformin + TZD</td>
<td>Exenatide</td>
<td>990</td>
<td>Taspoglutide demonstrated superior HbA1c reduction versus exenatide following 24 weeks of treatment.</td>
</tr>
<tr>
<td>T-emerge 3</td>
<td>Pioglitazone + metformin</td>
<td>Placebo</td>
<td>330</td>
<td></td>
</tr>
<tr>
<td>T-emerge 4</td>
<td>Metformin</td>
<td>Sitagliptin</td>
<td>630</td>
<td>Taspoglutide demonstrated superior HbA1c reduction versus sitagliptin.</td>
</tr>
<tr>
<td>T-emerge 5</td>
<td>Metformin + SU</td>
<td>Insulin glargin</td>
<td>990</td>
<td></td>
</tr>
<tr>
<td>T-emerge 6</td>
<td>SU ± metformin</td>
<td>Pioglitazone</td>
<td>650</td>
<td></td>
</tr>
<tr>
<td>T-emerge 7</td>
<td>Metformin (high BMI)</td>
<td>Placebo</td>
<td>260</td>
<td></td>
</tr>
<tr>
<td>T-emerge 8</td>
<td>History of cardiovascular event</td>
<td>Placebo</td>
<td>2000</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Taspoglutide</td>
<td>Exenatide</td>
<td>Liraglutide</td>
<td>Exenatide LAR</td>
</tr>
<tr>
<td>-------------</td>
<td>--------------</td>
<td>-----------</td>
<td>-------------</td>
<td>---------------</td>
</tr>
<tr>
<td><strong>Origin</strong></td>
<td>Human</td>
<td>Lizard</td>
<td>Human</td>
<td>Lizard</td>
</tr>
<tr>
<td><strong>Administration</strong></td>
<td>Once weekly</td>
<td>Twice daily</td>
<td>Once daily</td>
<td>Once weekly</td>
</tr>
<tr>
<td><strong>Efficacy (HbA1c)</strong></td>
<td>&gt; exenatide (T-emerge 2)</td>
<td>&gt; exenatide (LEAD6)</td>
<td>&gt; exenatide (DURATION1)</td>
<td></td>
</tr>
<tr>
<td><strong>Device</strong></td>
<td>Autoinjector (TBD)</td>
<td>Pen</td>
<td>Pen</td>
<td>Vial, prepared at each use</td>
</tr>
</tbody>
</table>
Development in Japan

Development code: ITM-077/RG1583
Originator: Ipsen/Roche
Partner: Co-development with Teijin Pharma
Chemical Structure: Human GLP-1 analogue
Formulation: Injection (sustained-release with zinc-based formulation)

< Ongoing study in Japan >

<table>
<thead>
<tr>
<th>Study name</th>
<th>Phase 2 clinical study of ITM-077/RG1583 in type 2 diabetes mellitus patients.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Conducters</td>
<td>Teijin Pharma / Chugai Pharmaceutical</td>
</tr>
<tr>
<td>Summary</td>
<td>To investigate the efficacy, safety and dose-response of ITM-077/RG1583 after 12 weeks treatment in comparison with placebo in type 2 diabetes mellitus patients.</td>
</tr>
<tr>
<td>Administration</td>
<td>Once weekly, sc at abdomen</td>
</tr>
<tr>
<td>Design</td>
<td>Multi-center, double-blind, randomized, placebo-controlled, parallel intergroup study</td>
</tr>
<tr>
<td>Primary endpoint</td>
<td>Efficacy (HbA1c), safety</td>
</tr>
</tbody>
</table>
Overview of CSG452

CHUGAI PHARMACEUTICAL CO., LTD.
Project Management Dept.
Global Project Leader
Sachiya Ikeda

December 7, 2009
Contents

1. Profile of CSG452
2. Profile of SGLT2 inhibitor and its development status
3. Development status of CSG452
The compound is;

- Selective SGLT2 inhibitor (C-glycoside) created at Fuji-Gotemba Laboratory, Chugai
- Co-development with Roche since 2007
- Ongoing global phase 2 (dose finding) study including participation from Japan
- FDD scheduled in 2010
Mode of Action of CSG452

- Blood glucose control through direct glucose excretion

- Sodium glucose co-transporter (SGLT) causes renal tubular reabsorption of blood glucose after glomerular filtration

- CSG452 prevents reabsorption of glucose by selectively inhibiting SGLT2, which carries larger transportation capacities between the two SGLTs in kidney. The blood glucose level is decreased as a result of glucose excretion in urine.

Insulin-independent blood glucose control is achieved
Contents

1. Profile of CSG452
2. Profile of SGLT2 inhibitor and its development status
3. Development status of CSG452
Profile of SGLT2 inhibitor

**Indication**
- Type 2 diabetes (possible Type 1 diabetes and pre-diabetes)

**Differentiation from existing drugs**
- Sustained blood glucose control irrespective of patient background
- Body weight /Blood pressure reduction
- Anti-diabetic effects by cancellation of glucotoxicity
- Low hypoglycemic risk
- High safety and tolerability (no GI disturbance, no edema)
- Combined with all anti-diabetics theoretically

**Safety issues to be evaluated in long term studies**
- Incidence of urinary tract infection and/or genital infection
- Water imbalance (polyuria, nocturia, dehydration, hypovolemia, increased haematocrit)
- Renal toxicity
SGLT2 inhibitor opens new insight into the diabetes pathophysiology “glucotoxicity”

- Sustained hyperglycemia impairs insulin secretion and inhibits insulin signaling (insulin resistance). This leads further hyperglycemia and it worsens impaired insulin secretion and insulin resistance then finally diabetes is developed.

- This vicious cycle is called *GLUCOTOXICITY* and is thought to play a major role for onset and progression of diabetes mellitus.
SGLT2 inhibitor opens new insight into the diabetes pathophysiology “glucotoxicity”

- Sustained hyperglycemia impairs insulin secretion and inhibits insulin signaling (insulin resistance). This leads further hyperglycemia and it worsens impaired insulin secretion and insulin resistance then finally diabetes is developed.

- This vicious cycle is called **GLUCOTOXICITY** and is thought to play a major role for onset and progression of diabetes mellitus.
## Competitive landscape of selective SGLT2 inhibitor (as of Nov/2009)

<table>
<thead>
<tr>
<th>Drug</th>
<th>Company</th>
<th>Dev. stage</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Overseas</td>
</tr>
<tr>
<td>Dapagliflozin</td>
<td>BMS/ AZ</td>
<td>P3</td>
</tr>
<tr>
<td>Canagliflozin (TA-7284)</td>
<td>J&amp;J/ Mitsubishi-Tanabe</td>
<td>P3</td>
</tr>
<tr>
<td>CSG452 (R7201)</td>
<td>Chugai/ Roche</td>
<td>P2</td>
</tr>
<tr>
<td>BI 10773</td>
<td>Boehringer Ingelheim</td>
<td>P2</td>
</tr>
<tr>
<td>ASP-1941</td>
<td>Astellas</td>
<td>P2</td>
</tr>
<tr>
<td>LX4211</td>
<td>Lexicon</td>
<td>P2</td>
</tr>
<tr>
<td>ISIS 388626</td>
<td>Isis</td>
<td>P1</td>
</tr>
</tbody>
</table>
Contents

1. Profile of CSG452
2. Profile of SGLT2 inhibitor and its development status
3. Development status of CSG452
## Ongoing Clinical Trial

### < P2 Multinational Study >

<table>
<thead>
<tr>
<th>Title of the study</th>
<th>Investigate Glycemic Parameters of Efficacy, Safety/ Tolerability and Pharmacokinetics of Five Dose Levels of R7201/CSG452 in Patients With Type 2 Diabetes Mellitus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sponsor</td>
<td>Chugai Pharmaceutical</td>
</tr>
<tr>
<td>Summary</td>
<td>12-week Study will evaluate the efficacy, safety and pharmacokinetics of 5 doses of R7201 compared to placebo in patients with type 2 diabetes mellitus.</td>
</tr>
<tr>
<td>Dose</td>
<td>Once daily, Oral</td>
</tr>
<tr>
<td>Study design</td>
<td>Treatment, Randomized, Double Blind (Subject, Investigator), Parallel Assignment, Safety/Efficacy Study 6 arm study (2.5mg, 5mg, 10mg, 20mg, 40mg, Placebo)</td>
</tr>
<tr>
<td>Inclusion Criteria</td>
<td>Either treated with diet, exercise and stable metformin, or with diet and exercise alone.</td>
</tr>
<tr>
<td>Primary Outcome</td>
<td>Absolute change in HbA1c</td>
</tr>
</tbody>
</table>
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