Summary of Product Characteristics for Pharmaceutical Products

1.Name of the medicinal product

Imeg 1000mg tablets

2.Qualitative and quantitative composition

Each film coated contains: Imeglimin hydrochloride

1000mg tablets.

For the full list of excipients, see section 6.1.

3.Pharmaceutical form

Film Coated Tablet.

White capsule shaped film coated tablet scored on one side and plain on the other side.

4.Clinical particulars.

4.1 Therapeutic indications.

Imeglimin is a first in class drug with a unique dual mechanism of action for treatment of type 2 diabetes.

4.2 Posology and method of administration. Method of administration: Oral use.

Posology:

For adults, the recommended dosing range of imeglimin Hydrochloride is 1000mg – 2000mg in divided doses or as directed by the physician.

4.3 Contraindications.

Patients with a history of hypersensitivity to the ingredients of this drug

Patients with severe ketosis, diabetic coma or precoma, and type 1 diabetes mellitus [Prompt correction of hyperglycemia with transfusion and insulin is essential].

Patients with severe infections, pre- and post-surgery, and severe trauma [Because blood sugar control by insulin injection is desired, administration of this drug is not suitable].

4.4 Special warnings and precautions for use.

The use of this drug should be considered only when dietary therapy and exercise therapy, which are the basics of diabetes treatment, are adequately performed in advance and the effect is insufficient.

In patients with renal dysfunction, renal excretion is delayed and the blood concentration of this drug increases, depending on the degree of renal dysfunction. Efficacy and safety in patients with moderate or severe renal impairment (eGFR less than 45 mL/min/1.73 m2) have not been conducted and administration is not recommended.

If you have renal dysfunction, it is advisable to have your renal function checked regularly, as the excretion of this drug may be delayed and blood levels may increase.

Patients should be fully informed of hypoglycemic symptoms and how to deal with them when using this drug.

Since hypoglycemic symptoms may occur, care should be taken when administering to patients

who are engaged in work at heights, driving a car, etc.
When administering this drug, blood glucose should be checked regularly to confirm the drug's effect, and if the drug's effect is insufficient after 3 months of administration, a change to a more appropriate treatment should be considered.

The mechanism of action of this drug and biguanides may be partially shared, and the combination of both drugs may cause gastrointestinal symptoms compared to combination therapy with other antidiabetic drugs. Since many cases were observed, care should be taken when selecting concomitant drugs.

4.5 Interaction with other medicinal products and other forms of interaction.

This drug is mainly excreted through the kidneys as unchanged drug

Precautions for co-administration

Drug name, etc.	**Clinical symptoms/measures**	**Mechanism/risk factor**
Diabetes medicine • Insulin preparations Sulfonylureas Fast-acting insulin secretagogues α-glucosidase inhibitors Thiazolidines DPP-4 inhibitors GLP-1 receptor agonists SGLT2 inhibitors, etc.	Be alert for episodes of hypoglycemia. Especially when used concomitantly with insulin preparations, sulfonylureas or rapid-acting insulin secretagogues, the risk of hypoglycemia may be increased. Consider reducing the dose of insulin, sulfonylureas, or fast-acting insulin secretagogues to reduce the risk of hypoglycemia with these agents.	Hypoglycemic effect may be enhanced.
Biguanides	Pay attention to the occurrence of hypoglycemia and gastrointestinal symptoms.	For hypoglycemia, the hypoglycemic effect may be enhanced. Gastrointestinal symptoms tend to occur more frequently, especially in the initial period of concomitant use.
Drugs that enhance hypoglycemic action • β-blockers Salicylates Monoamine oxidase inhibitors, etc.	Patients should be carefully monitored for blood sugar levels and other conditions before administration	Hypoglycemic effect may be enhanced.
Drugs that attenuate hypoglycemic effects • Adrenaline Adrenal cortical hormone Thyroid hormone, etc.	Patients should be carefully monitored for blood sugar levels and other conditions before administration.	Hypoglycemic effect may be attenuated.

4.6 Fertility, pregnancy and lactation

Do not administer this drug to pregnant women or women who may be pregnant, but use insulin preparations. In animal experiments (rats), transfer to the fetus has been confirmed. In animal experiments in which this drug was administered during fetal organogenesis, rats were orally administered 1500 mg/kg/day (corresponding to an exposure level approximately 17 times higher than the maximum clinical dose of 2000 mg/day), low live fetal weight and delayed ossification2). After oral administration of 200 mg/kg/day (equivalent to approximately 1.4 times the maximum clinical dose of 2000 mg/day) to rabbits, there was a trend toward lower whole embryo resorption and the number of live fetuses after implantation.

A trend toward increased mortality and decreased live fetal weight has been observed

Lactating women: Consider the therapeutic benefit and the benefit of breastfeeding and consider continuing or discontinuing breastfeeding. In animal experiments (rats), migration into breast milk has been confirmed

4.7 Effects on ability to drive and use machines.

Patients should be warned of the potential hazards of driving or operating machinery if they experience side effects such as dizziness.

4.8 Undesirable effects

Hypoglycemia may occur. In particular, hypoglycemia may occur when used in combination with insulin preparations, sulfonylureas, or rapid-acting insulin secretagogues. If hypoglycemic symptoms (initial symptoms: weakness, severe hunger, sweating, etc.) are observed, take appropriate measures such as ingesting food containing carbohydrates.

However, if hypoglycemic symptoms are observed due to concomitant use with an α-glucosidase inhibitor, glucose should be administered.

Other side effects include

	1% to less than 5%	**Less than 1%**
Infectious diseases and parasitic diseases		cystitis
Metabolic and nutritional disorders		Loss of appetite
Eye disorder		Diabetic retinopathy, diabetic retinal edema/ macular edema
Gastrointestinal disorders	Nausea, Diarrhea, Constipation	Vomiting, Abdominal discomfort, Indigestion, Upper abdominal pain, Loose stools, Abdominal distension, Gastroesophageal reflux disease
Clinical examination		Blood lactate increase, lipase increase, weight loss

Reporting of suspected adverse reactions: Healthcare professionals are requested to report any suspected adverse reactions to report any suspected adverse reactions to the National Regulatory Authority.

4.9 Overdose: Clinically significant hypotension requires active cardiovascular support including frequent monitoring of cardiac and respiratory function, elevation of extremities and attention to circulating fluid volume and urine output.

5.Pharmacological properties

5.1 Pharmacodynamic of Imeglimin HCl. Mechanism of Action

Imeglimin is a drug that exerts a hypoglycemic effect through a glucose level-dependent insulin secretagogue effect and an insulin resistance-improving effect, and its mechanism of action is presumed to be via action on mitochondria.

Hypoglycemic effect: Imeglimin showed a hypoglycemic effect in Goto-Kakizaki (GK) rats, and

the effect lasted for 8 weeks from the start of administration.

Glucose Level-Dependent Insulin Secretagogue: Imeglimin stimulated insulin secretion in the presence of high glucose in an in vitro test using pancreatic islets derived from GK rats). In streptozotocin-induced diabetic model rats, GK rats, and high-fat diet-loaded rats, it increased blood insulin levels in glucose tolerance tests, and also in glucose clamp tests under hyperglycemic conditions. increased concentration). Patients with type 2 diabetes were orally administered 1500 mg of this drug Note) or placebo twice daily for 7 days, and a high glucose clamp test was performed 2 hours after the last dose. AUC 0-45min of post-treatment blood insulin concentration increased compared to the placebo group) (foreign subject data). Patients with type 2 diabetes were administered 1500 mg of this drug or placebo twice daily for 18 weeks, and an oral glucose tolerance test was performed 2 hours after the last dose. increased compared to the group) (foreign data).

Improving insulin resistance: Imeglimin showed gluconeogenesis-suppressing effects and glucose uptake-increasing effects) in in vitro tests using primary cultured hepatocytes and in vitro tests using muscle cell lines. It also increased the steady-state glucose infusion rate in a normoglycemic hyperinsulinum clamp test in a streptozotocin-induced diabetic model animal). Patients with type 2 diabetes were administered 1500 mg of this drug Note) or placebo twice daily for 18 weeks, and an oral glucose tolerance test was performed 2 hours after the last dose. The Stumvoll index, which is one of the subjects, improved compared to the placebo group) (foreign data). Note) The approved dosage and administration of this drug is 1000 mg twice daily.

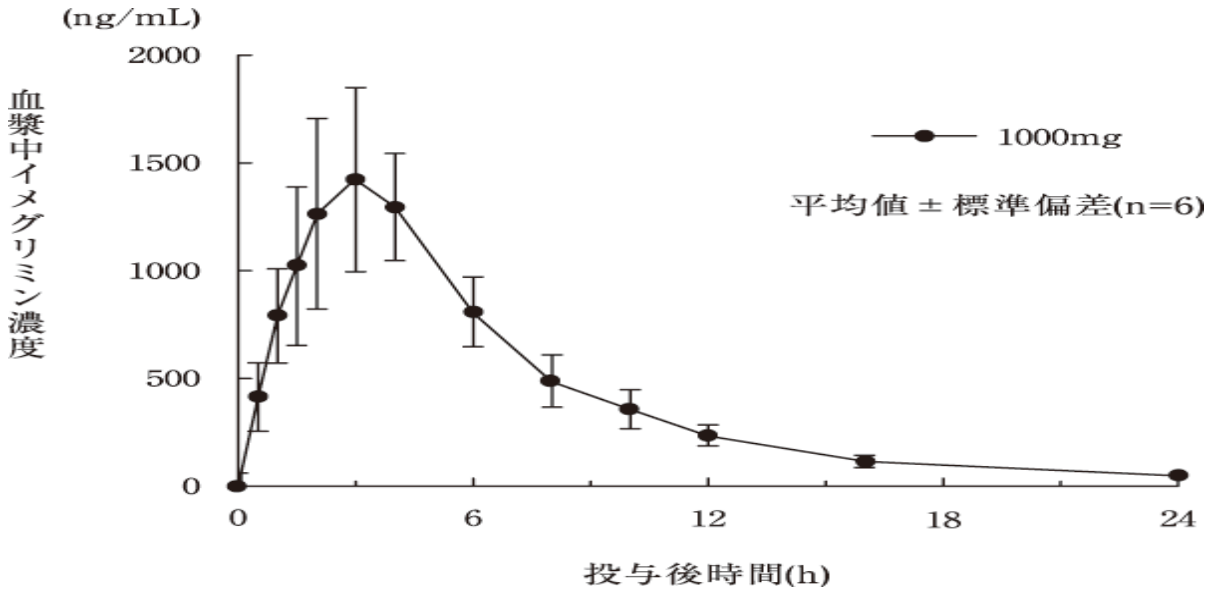
5.2 Pharmacokinetic properties

Following a single oral administration of 1000 mg of this drug to healthy adults under fasting conditions, the plasma concentration profile and pharmacokinetic parameters were as follows 5.

-Y axis =Plasma Imeglimin concentration Mg/ml

Mean+/_standard deviation(n=6)

X -axis= Administration per hour



Dose	t max (h)	C max (ng/mL)	AUC 0-24 (ng h/mL)	t 1/2 (h)
1000 mg (n=6)	2.5 (1.5–3.0)	1393 (40.3)	9780 (36.1)	12.0 (113.0)
Geometric mean (% geometric CV), t max : median (minimum value - maximum value)				

Repeated doses

When 1000 mg of this drug was orally administered twice daily to 6 healthy adults for 7 days, the plasma concentration reached a steady state on the 5th day of administration, and the Cmax and AUC 0-12 values on the 7th day. The accumulation ratios were 1.43 and 1.57, respectively. As a result of population PK analysis based on the plasma concentration obtained from 867 patients treated with this drug, type 2 diabetes patients enrolled in the domestic phase 3 study (monotherapy) (103 patients: mean eGFR 73.2 mL) /min/1.73m 2), the exposure (AUC 0-12,ss) was estimated to be 18.0 µg h/mL (geometric mean) when 1000 mg of this drug was orally administered twice daily.

Pharmacokinetic parameters following a single oral administration of 1000 mg of this drug to healthy adult men under fasting conditions and after meals were as follows. No clinically significant effect of diet was observed.

Dosing time	**t max (h)**	**C max (ng/mL)**	**AUC 0-48 (ng h/mL)**	**t 1/2 (h)**
Fasting (n=12)	3.0 (1.0–4.0)	1681 (27.5)	12970 (30.6)	7.2 (56.4)
Postprandial (n=12)	4.0 (3.0–4.0)	1424 (26.1)	11960 (29.9)	6.1 (59.9)
Geometric mean (% geometric CV), t max: median (minimum value - maximum value)				

Distribution: The protein binding rate of Imeglimin in human plasma was 1.2% to 6.4%) (in vitro).

Metabolism: When a single dose of 1000 mg of 14 C-labeled Imeglimin was orally administered to 6 healthy adult male subjects, Imeglimin was hardly metabolized, and the main radioactive component in plasma and urine was unchanged drug 9) Imeglimin did not inhibit CYP1A2, CYP2A6, CYP2B6, CYP2C8, CYP2C9, CYP2C19, CYP2D6, CYP2E1, CYP3A4/5 (IC 50 >100 µmol/L) , and at concentrations up to 120 µmol/L, CYP1A2, CYP2B6

, CYP2C9, CYP2C19 and CYP3A4/511) (in vitro).

Excretion: A single oral dose of 1000 mg of 14 C-labeled Imeglimin was administered to 6 healthy adult male subjects. The cumulative excretion rate of medium radioactivity was 54.8% of the administered radioactivity12) (data from foreigners). Imeglimin was a substrate for OCT1, OCT2, MATE1 and MATE2-K, but not for P-gp, BCRP, OAT1 and OAT (in vitro). Imeglimin showed no inhibitory effect on P-gp, BCRP, OATP1B1, OATP1B3, OAT1, OAT3 and MATE2-K (IC 50 >1000 μmol/L)),(in vitro). On the other hand, it showed an inhibitory effect on OCT1 (K i: 154 μmol/L), OCT2 (IC 50 : 146 μmol/L) and MATE1 (IC 50 : 19.24 μmol/L)) (in vitro), the possibility of clinically relevant drug interactions was considered low.

Pharmacokinetic in Patients with specific backgrounds

Persons with renal impairment:

The pharmacokinetics of a single oral dose of this drug in subjects with different degrees of renal impairment (classified based on eGFR measurements) was evaluated in subjects with normal renal function (eGFR ≥90 mL/min/1.73 m2). The results of a comparison with a single oral administration of 1000 mg of the drug were as follows

Renal function (eGFR (mL/min/1.73m 2))	**Dosage (mg)**	**Number of cases**	**Cmax _**	**AUC 0-last**
			Geometric mean ratio [90% confidence interval]	**Geometric mean ratio [90% confidence interval]**
Mild (60 ≤ eGFR < 90)	1000	6	1.42 [1.05, 1.91]	1.49 [1.03, 2.17]
Moderate (30 ≤ eGFR < 60)	1000	6	1.52 [1.13, 2.05]	1.81 [1.25, 2.63]
Severe (15 ≤ eGFR < 30)	500	6	1.50 [1.11, 2.02]	2.49 [1.71, 3.61]

Subjects with different degrees of renal impairment (classified based on measured values of CL cr (creatinine clearance)) were evaluated for the pharmacokinetics of repeated oral administration of this drug at 500 mg Note) twice daily in subjects with normal renal function. (CL cr >80 mL/min) was compared with repeated oral administration of 500 mg of this drug twice daily, and the results were as follows17) (non-Japanese data).

Renal function (CL cr *1)	**Number of cases**	**Cmax _**	**AUC 0-last**
		Geometric mean ratio [90% confidence interval]	Geometric mean ratio [90% confidence interval]
Mild (50 ≤ CL cr ≤ 80)	Four	1.28 [1.03, 1.59]	1.50 [1.16, 1.94]
Moderate (30 ≤ CLcr < 50)	6	1.95 [1.61, 2.35]	2.32 [1.85, 2.92]
Severe (CLcr < 30)	Five	2.86 [2.08, 3.94]	3.56 [2.51, 5.06]
Subjects with normal renal function were enrolled after considering the subject background by degree of renal impairment, and 8 subjects with normal renal function and severe renal impairment with mild and moderate renal impairment were included. compared 6 subjects with normal renal function, independent of 8 compared with subjects with mild and moderate renal impairment. *1: Creatinine clearance (mL/min)			

Pharmacokinetic in Persons with liver dysfunction

When a single oral dose of 1000 mg of this drug was administered to 7 subjects with moderate hepatic impairment (Child-Pugh classification B), the least square geometric mean ratio of Cmax and AUC 0-last of Imeglimin (hepatic impairment / healthy adults) and 90% confidence intervals were 1.29 [1.05, 1.60] and 1.47 [1.19, 1.82], respectively18) (non-Japanese data).

Elderly: In Japanese late phase 2 and 3 studies in patients with type 2 diabetes mellitus, the steady-state AUC (AUC 24, ss) of subjects who received multiple oral doses of 1000 mg of this drug twice daily was population- based. Estimated by PK analysis, the AUC24, ss of the elderly aged 65 years or older was 1.28 times higher than that of the elderly aged 65 years

5.3 Preclinical safety data

Domestic late phase 2 study

A placebo-controlled, double-blind, controlled trial was conducted in type 2 diabetes patients who were either treatment-naive or receiving other oral hypoglycemic monotherapy for at least 12 weeks. Patients who are being treated with other oral hypoglycemic drugs should discontinue administration of oral hypoglycemic drugs at screening, and after the washout period, receive 500 mg*1), 1000 mg, 1500 mg*1) or placebo once daily2. As a result of oral administration for 24 weeks, HbA1c significantly decreased in all dose groups compared to the placebo group, as shown in the table below.

Dosing group	**Number of cases *1**	**HbA1c (%)**		
		Mean value before administration *2	**Amount of change from before administration *3**	**Difference from placebo *3**
Placebo	75	7.89±0.676	0.43±0.092 [0.25, 0.61]	-
500mg	75	7.94±0.679	−0.09±0.091 [−0.27, 0.09]	−0.52±0.128 [−0.77, −0.27] p<0.0001
1000mg	73	7.85±0.650	−0.51±0.093 [−0.69, −0.32]	−0.94±0.129 [−1.19, −0.68] p<0.0001
1500mg	73	7.91±0.618	−0.57±0.094 [−0.76, −0.39]	−0.94±0.129 [−1.19, −0.68] p<0.0001

*1: FAS population
*2: Mean ± standard deviation
*3: Analysis by Mixed Model Repeated Measures, least squares mean ± standard error [95% confidence interval]

The incidence of side effects was 5.3% (4/75 cases) in the 500 mg group, 5.4% (4/74 cases) in the 1000 mg group, 24.0% (18/75 cases) in the 1500 mg group, and 8.0% (6/75 cases) in the placebo group. It was an example. Adverse reactions with an incidence of 2% or more were not observed in the placebo group, 500 mg group, or 1000 mg group. 3/75 cases) and vomiting 2.7%

(2/75 cases). Hypoglycemia (symptomatic hypoglycemia and/or blood glucose level <70 mg/dL, same below) was 1.3% (1/75 cases) in the 500 mg group, 1.4% (1/74 cases) in the 1000 mg group, and 1.4% (1/74 cases) in the 1500 mg group. 5.3% (4/75 cases) and 1.3% (1/75 cases) in the placebo group, but no severe hypoglycemia Note 2) was observed.

Phase 3 study in Japan (monotherapy)

A placebo-controlled, double-blind controlled study in type 2 diabetes patients who had no prior treatment for type 2 diabetes other than diet and exercise or who had received other oral hypoglycemic monotherapy for 12 weeks or more. Carried out. Patients being treated with other oral hypoglycemic drugs were discontinued at screening, and after the washout period, 1000 mg of this drug or placebo was orally administered twice daily for 24 weeks. As shown, HbA1c was significantly decreased in the favipiravir group compared with the placebo group

Dosing group	**Number of cases *1**	**HbA1c (%)**		
		Mean value before administration *2	**Amount of change from before administration *3**	**Difference from placebo *3**
Placebo	106	7.93±0.684	0.15±0.07 [0.008, 0.286]	-
This drug	106	7.99±0.764	−0.72±0.07 [−0.856, −0.581]	−0.87±0.09 [−1.041, −0.691] p<0.0001

*1: FAS population
*2: Mean ± standard deviation
*3: Analysis by Mixed Model Repeated Measures, least squares mean ± standard error [95% confidence interval]

The incidence of adverse reactions was 4.7% (5/106 cases) in the drug group and 6.5% (7/107 cases) in the placebo group. Adverse reactions with an incidence of ≥2% were not observed in either the riociguat group or the placebo group. Hypoglycemia was observed in 1.9% (2/106 cases) of the drug group and 0.9% (1/107 cases) of the placebo group, but no severe hypoglycemia Note 2) was observed 25

Phase 3 long-term study in Japan (monotherapy and combination therapy)

An open-label study was conducted in type 2 diabetic patients whose blood sugar was inadequately controlled with diet/exercise therapy or monotherapy with hypoglycemic drugs for 12 weeks or longer. This drug was administered orally at a dose of 1000 mg twice daily for 52 weeks as monotherapy or as a combination therapy in which this drug was added to one other antihyperglycemic drug. HbA1c decreased and stable glycemic control was maintained for 52 weeks.

Dosing group	**Number of cases *1**	**HbA1c (%)**	
		Mean value before administration *2	**Amount of change from before administration *3**
Monotherapy of this drug	134	7.83±0.730	
Concomitant use of sulfonylureas	127	8.63±0.903	
Concomitant use with an α-glucosidase inhibitor	64	8.37±0.771	

Concomitant fast-acting insulin secretagogues	64	8.48±0.837	
Concomitant use of biguanides	64	8.16±0.607	
Concomitant use of thiazolidinediones	65	8.72±0.942	
Concomitant DPP-4 inhibitor	63	8.23±0.750	
SGLT2 inhibitor combination	63	8.50±0.748	
GLP-1 receptor agonist combination	70	8.66±0.848	

*1: FAS population
*2: Mean ± standard deviation
*3: Analysis by Mixed Model Repeated Measures, least squares mean ± standard error [95% confidence interval]

The incidence of side effects was 9.7% (13/134 cases) in the monotherapy group, 21.3% (27/127 cases) in the sulfonylurea combination group, and 9.4% (6/64 cases) in the α-glucosidase inhibitor combination group. , 15.6% (10/64) in the fast-acting insulin secretagogue group, 37.5% (24/64) in the biguanide group, 9.2% (6/65) in the thiazolidinedione group, and DPP 22.2% (14/63) in the -4 inhibitor group, 11.1% (7/63) in the SGLT2 inhibitor group, and 11.4% (8/70) in the GLP-1 receptor agonist group there were. Adverse reactions with an incidence of 2% or higher, excluding hypoglycemia, were nausea in 3.0% (4/134 cases) and constipation in 2.2% (3/134 cases) of the monotherapy group, and constipation in the combination therapy group (3.1% of the sulfonylurea group). 4/127 cases), diarrhea 4.7% (3/64 cases) in the fast-acting insulin secretagogue group, diarrhea 15.6% (10/64 cases) in the biguanide group, and nausea 10.9% (7/64 cases). , vomiting 4.7% (3/64 cases), upper abdominal pain 3.1% (2/64 cases), abdominal discomfort 3.1% (2/64 cases), decreased appetite 3.1% (2/64 cases), α-glucosidase inhibition Constipation 3.1% (2/64 cases) in the drug combination group, diarrhea 3.1% (2/65 cases), vomiting 3.1% (2/65 cases), diabetic retinopathy 3.1% (2/65 cases) in the thiazolidinedione group.), nausea 6.3% (4/63), decreased appetite 3.2% (2/63) in the DPP-4 inhibitor combination group, nausea 3.2% (2/63) in the SGLT2 inhibitor combination group, GLP-1 Concomitant use of receptor agonists resulted in nausea 4.3% (3/70), vomiting 2.9% (2/70), dyspepsia 2.9% (2/70), and anorexia 2.9% (2/70). Hypoglycemia was 1.5% (2/134) in the monotherapy group, 11.8% (15/127) in the sulfonylurea combination group, and 3.1% (2/64) in the α-glucosidase inhibitor combination group. 6.3% (4/64) in the fast-acting insulin secretagogue group and 7.0% in the biguanide group. Note 2) was not recognized. No hypoglycemia was observed in the thiazolidinedione drug combination group or the GLP-1 receptor agonist combination.

Phase 3 study in Japan (insulin combination therapy).

A double-blind, placebo-controlled study was conducted in patients with type 2 diabetes who had insufficient glycemic control with insulin therapy (either monotherapy or combination therapy with insulin and one oral hypoglycemic agent). Oral administration of 1000 mg of this drug or placebo twice daily for 16 weeks in combination with an insulin preparation (either basal insulin or mixed/combined dissolved insulin, daily dose of 8 units or more and 40 units

or less) As a result of administration, as shown in the table below, HbA1c at week 16 of administration was significantly lower in this drug group than in the placebo group.

Dosing group	**Number of cases *1**	**HbA1c (%)**		
		Mean value before administration *2	Amount of change from before administration *3	Difference from placebo *3
Placebo	106	8.82±0.813	−0.03±0.07 [−0.177, 0.115]	-
This drug	108	8.74±0.721	−0.63±0.07 [−0.778, −0.489]	−0.60±0.10 [−0.802, −0.404] p<0.0001
*1: FAS population *2: Mean ± standard deviation *3: Analysis by Mixed Model Repeated Measures, least squares mean ± standard error [95% confidence interval].				

In addition, subjects who had been in the riociguat group during the 16-week double-blind period continued to receive riociguat for 36 weeks (total of 52 weeks) during the open-label period. The amount of change (least squares mean ± standard error) was -0.64 ± 0.09%. The incidence of adverse reactions up to Week 16 of administration was 14.8% (16/108 subjects) in this drug group and 12.1% (13/107 subjects) in the placebo group. Adverse reactions with an incidence of 2% or more were hypoglycemia, which accounted for 12.0% (13/108 cases) of the drug group and 6.5% (7/107 cases) of the placebo group. The incidence of adverse reactions up to 52 weeks of administration of this drug was 25.9% (28/108 cases), and adverse reactions with an incidence of 2% or more were hypoglycemia in 21.3% (23/108 cases) and constipation in 2.8% (3/108 cases).) Met. Severe hypoglycemia Note 2) was not observed. Note 1) The approved dosage and administration of this drug is 1000 mg twice daily. Note 2) Severe hypoglycemia: Hypoglycemia requiring third party assistance or other treatment for carbohydrate or glucagon administration

6.Pharmaceutical particulars

6.1 List of excipients

Microcrystalline cellulose pH
102 BP Croscarmellose
sodium BP
Aerosil 200 BP
Hydroxypropyl cellulose
BP Magnesium stearate
BP Tabcoat TC 580118
White.

6.2 Incompatibilities

Not applicable.

6.3 Shelf life

2 years

6.4 Special precautions for storage.

Do not store above 30°C. Protect from direct sunlight. Keep all medicines out of reach of

children.

6.5 Nature and contents of container

PVC /Aluminium foil blister packs of 3 x10's in unit box with literature insert.

6.6 Special precautions for disposal and other handling

Any unused medicinal product or waste material should be disposed of in accordance with local requirements.

7. Marketing authorization holder.

Dawa Limited,

Plot No 7879/8, Baba Dogo Road –Ruaraka

P.O Box 16630-6200-Nairobi - Kenya.

8. Marketing authorization number(s)

Under registration.

9. Date of first authorization/renewal of the authorization

September 17, 2025.

10. Date of revision of the text

February 2022