

HIGHLIGHTS OF PRESCRIBING INFORMATION

These highlights do not include all the information needed to use KENGREAL safely and effectively. See full prescribing information for KENGREAL.

KENGREAL® (cangrelor) for injection, for intravenous use
Initial U.S. Approval: 2015

INDICATIONS AND USAGE

KENGREAL is a P2Y₁₂ platelet inhibitor indicated as an adjunct to percutaneous coronary intervention (PCI) for reducing the risk of periprocedural myocardial infarction (MI), repeat coronary revascularization, and stent thrombosis (ST) in patients in who have not been treated with a P2Y₁₂ platelet inhibitor and are not being given a glycoprotein IIb/IIIa inhibitor. (1)

DOSAGE AND ADMINISTRATION

- KENGREAL is intended for administration via a dedicated IV line, only after reconstitution and dilution. (2.3)
- Administer 30 mcg/kg intravenous (IV) bolus prior to PCI followed immediately by a 4 mcg/kg/min IV infusion for at least 2 hours or duration of procedure, whichever is longer. (2.1)
- To maintain platelet inhibition after discontinuation of KENGREAL infusion, an oral P2Y₁₂ platelet inhibitor should be administered. (2.2)

DOSAGE FORMS AND STRENGTHS

Single-use 10 mL vial containing 50 mg KENGREAL as a lyophilized powder for reconstitution (3.0)

CONTRAINDICATIONS

- Significant active bleeding (4.1)
- Hypersensitivity to KENGREAL or any component of the product (4.2)

WARNINGS AND PRECAUTIONS

- Bleeding: Like other drugs that inhibit platelet P2Y₁₂ function, KENGREAL can increase the risk of bleeding (5.1)

ADVERSE REACTIONS

The most common adverse reaction is bleeding. (5.1, 6.1)

To report SUSPECTED ADVERSE REACTIONS, contact Chiesi USA, Inc. at 1-888-661-9260 or FDA at 1-800-FDA-1088 or www.fda.gov/medwatch.

DRUG INTERACTIONS

- Clopidogrel: Do not administer during KENGREAL infusion. (7.1)
- Prasugrel: Do not administer during KENGREAL infusion. (7.1)

Revised: 09/2020

FULL PRESCRIBING INFORMATION: CONTENTS*

1	INDICATIONS AND USAGE	8.5	Geriatric Use
2	DOSAGE AND ADMINISTRATION	8.6	Renal Impairment
2.1	Recommended Dosing	8.7	Hepatic Impairment
2.2	Transitioning Patients to Oral P2Y ₁₂ Therapy	10	OVERDOSAGE
2.3	Preparation and Administration	11	DESCRIPTION
3	DOSAGE FORMS AND STRENGTHS	12	CLINICAL PHARMACOLOGY
4	CONTRAINDICATIONS	12.1	Mechanism of Action
4.1	Significant Active Bleeding	12.2	Pharmacodynamics
4.2	Hypersensitivity	12.3	Pharmacokinetics
5	WARNINGS AND PRECAUTIONS	13	NONCLINICAL TOXICOLOGY
5.1	Bleeding	13.1	Carcinogenesis, Mutagenesis, Impairment of Fertility
6	ADVERSE REACTIONS	14	CLINICAL STUDIES
6.1	Clinical Trials Experience	14.1	CHAMPION PHOENIX Trial
7	DRUG INTERACTIONS	14.2	CHAMPION PCI and PLATFORM Trials
7.1	Thienopyridines	16	HOW SUPPLIED/STORAGE AND HANDLING
8	USE IN SPECIFIC POPULATIONS		
8.1	Pregnancy		
8.3	Nursing Mothers		
8.4	Pediatric Use		

*Sections or subsections omitted from the full prescribing information are not listed.

FULL PRESCRIBING INFORMATION

1 INDICATIONS AND USAGE

KENGREAL is indicated as an adjunct to percutaneous coronary intervention (PCI) to reduce the risk of periprocedural myocardial infarction (MI), repeat coronary revascularization, and stent thrombosis (ST) in patients who have not been treated with a P2Y₁₂ platelet inhibitor and are not being given a glycoprotein IIb/IIIa inhibitor [see *Clinical Studies (14.1)*].

2 DOSAGE AND ADMINISTRATION

2.1 Recommended Dosing

The recommended dosage of KENGREAL is a 30 mcg/kg IV bolus followed immediately by a 4 mcg/kg/min IV infusion. Initiate the bolus infusion prior to PCI. The maintenance infusion should ordinarily be continued for at least 2 hours or for the duration of PCI, whichever is longer.

2.2 Transitioning Patients to Oral P2Y₁₂ Therapy

To maintain platelet inhibition after discontinuation of KENGREAL infusion, an oral P2Y₁₂ platelet inhibitor should be administered. Administer one as described below:

- Ticagrelor: 180 mg at any time during KENGREAL infusion or immediately after discontinuation [see *Clinical Pharmacology (12.2)*].
- Prasugrel: 60 mg immediately after discontinuation of KENGREAL. Do not administer prasugrel prior to discontinuation of KENGREAL [see *Drug Interactions (7.1)* and *Clinical Pharmacology (12.2)*].
- Clopidogrel: 600 mg immediately after discontinuation of KENGREAL. Do not administer clopidogrel prior to discontinuation of KENGREAL [see *Drug Interactions (7.1)* and *Clinical Pharmacology (12.2)*].

2.3 Preparation and Administration

KENGREAL is intended for IV administration, after reconstitution and dilution.

Preparation

For each 50 mg/vial, reconstitute by adding 5 mL of Sterile Water for Injection. Swirl gently until all material is dissolved. Avoid vigorous mixing. Allow any foam to settle. Ensure that the contents of the vial are fully dissolved and the reconstituted material is a clear, colorless to pale yellow solution. Reconstitute the vial prior to dilution in a bag. Parenteral drug products should be inspected visually for particulate matter after reconstitution.

Do not use without dilution. Before administration, each reconstituted vial must be diluted further with Normal Saline (Sodium Chloride Injection 0.9% USP) or 5% Dextrose Injection USP.

Withdraw the contents from one reconstituted vial and add to one 250 mL saline bag. Mix the bag thoroughly. This dilution will result in a concentration of 200 mcg/mL and should be sufficient for at least 2 hours of dosing. Patients 100 kg and over will require a minimum of 2 bags.

Reconstituted KENGREAL should be diluted immediately. Diluted KENGREAL is stable for up to 12 hours in 5% Dextrose Injection and 24 hours in Normal Saline at Room Temperature. Discard any unused portion of reconstituted solution remaining in the vial.

Administration

Administer KENGREAL via a dedicated IV line.

Administer the bolus volume rapidly (<1 minute), from the diluted bag via manual IV push or pump. Ensure the bolus is completely administered before the start of PCI. Start the infusion immediately after administration of the bolus [*see Dosage and Administration (2.1)*].

3 DOSAGE FORMS AND STRENGTHS

For Injection: 50 mg of KENGREAL lyophilized powder in a single-use 10 mL glass vial for reconstitution.

4 CONTRAINDICATIONS

4.1 Significant Active Bleeding

KENGREAL is contraindicated in patients with significant active bleeding [*see Warnings and Precautions (5.1) and Adverse Reactions (6.1)*].

4.2 Hypersensitivity

KENGREAL is contraindicated in patients with known hypersensitivity (e.g., anaphylaxis) to KENGREAL or any component of the product [*see Adverse Reactions (6.1)*].

5 WARNINGS AND PRECAUTIONS

5.1 Bleeding

Drugs that inhibit platelet P2Y₁₂ function, including KENGREAL, increase the risk of bleeding.

In CHAMPION PHOENIX bleeding events of all severities were more common with KENGREAL than with clopidogrel [*see Adverse Reactions (6.1)*]. Bleeding complications with KENGREAL were consistent across a variety of clinically important subgroups (see [Figure 1](#)).

Once KENGREAL is discontinued, there is no antiplatelet effect after an hour [*see Clinical Pharmacology (12.3)*].

6 ADVERSE REACTIONS

The following adverse reactions are also discussed elsewhere in the labeling:

- Bleeding [*see Warnings and Precautions (5.1)*]

6.1 Clinical Trials Experience

Because clinical trials are conducted under widely varying conditions, adverse reactions rates observed in the clinical trials of a drug cannot be directly compared to rates in the clinical trials of another drug and may not reflect the rates observed in practice.

The safety of KENGREAL has been evaluated in 13,301 subjects in controlled trials, in whom, 5,529 were in the CHAMPION PHOENIX trial.

Bleeding

There was a greater incidence of bleeding with KENGREAL than with clopidogrel. No baseline demographic factor altered the relative risk of bleeding with KENGREAL (see Table 1 and Figure 1).

Table 1: Major Bleeding Results in the CHAMPION PHOENIX Study (Non-CABG related Bleeding)^a

CHAMPION PHOENIX	KENGREAL (N=5529)	Clopidogrel (N=5527)
Any GUSTO bleeding, n (%)	857 (15.5)	602 (10.9)
Severe/life-threatening ^b	11 (0.2)	6 (0.1)
Moderate ^c	21 (0.4)	14 (0.3)
Mild ^d	825 (14.9)	582 (10.5)
Any TIMI bleeding, n (%)	45 (0.8)	17 (0.3)
Major ^e	12 (0.2)	6 (0.1)
Minor ^f	33 (0.6)	11 (0.2)

Abbreviations: GUSTO: Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Arteries; TIMI: Thrombolysis in Myocardial Infarction

^a Safety population is all randomized subjects who received at least one dose of study drug

^b intracranial hemorrhage or bleeding resulting in substantial hemodynamic compromise requiring treatment

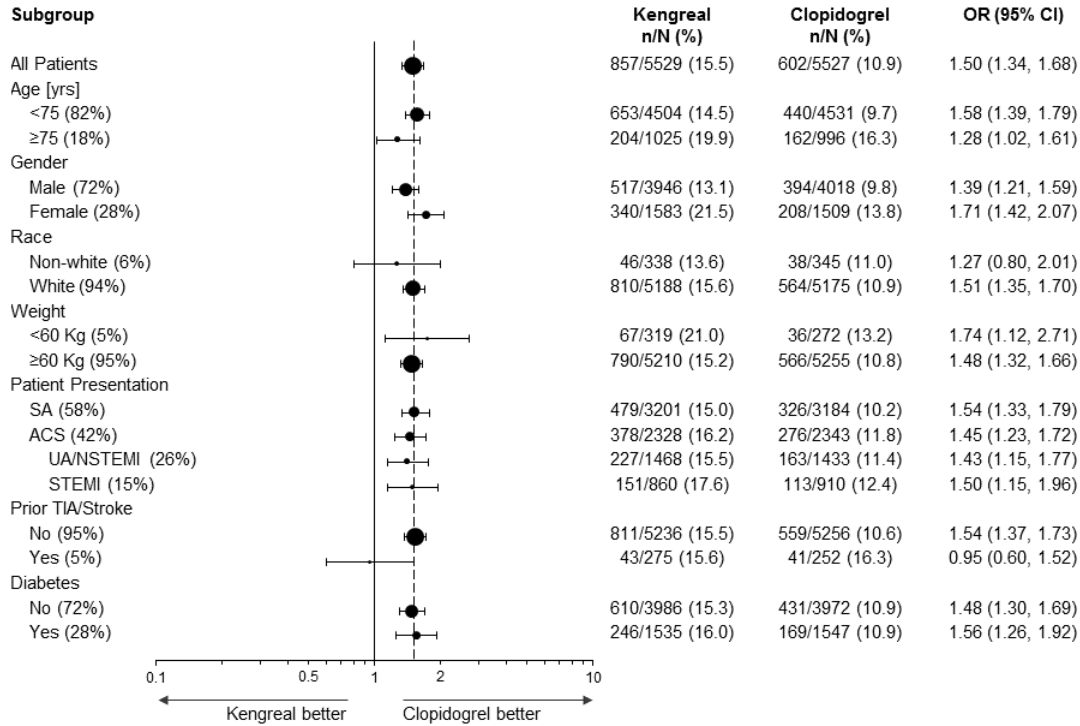
^c requiring blood transfusion but not resulting in hemodynamic compromise

^d all other bleeding not included in severe or moderate

^e any intracranial hemorrhage, or any overt bleeding associated with a reduction in hemoglobin of ≥ 5 g/dL (or, when hemoglobin is not available, an absolute reduction in hematocrit $\geq 15\%$)

^f any overt sign of bleeding (including observation by imaging techniques) that is associated with a reduction in hemoglobin of ≥ 3 g/dL and < 5 g/dL (or, when hemoglobin is not available, an absolute reduction in hematocrit of $\geq 9\%$ and $< 15\%$)

Figure 1: Bleeding Results in the CHAMPION PHOENIX Study^a (All Non-CABG related)



^a Safety population is all randomized subjects who received at least one dose of study drug

Note: The figure above presents effects in various subgroups most of which are baseline characteristics and most of which were pre-specified (patient presentation and weight were not pre-specified subgroups). The 95% confidence limits that are shown do not take into account how many comparisons were made, nor do they reflect the effect of a particular factor after adjustment for all other factors. Apparent homogeneity or heterogeneity among groups should not be over-interpreted.

Drug Discontinuation

In CHAMPION PHOENIX the rate of discontinuation for bleeding events was 0.3% for KENGREAL and 0.1% for clopidogrel. Discontinuation for non-bleeding adverse events was low and similar for KENGREAL (0.6%) and for clopidogrel (0.4%). Coronary artery dissection, coronary artery perforation, and dyspnea were the most frequent events leading to discontinuation in patients treated with KENGREAL.

Non-Bleeding Adverse Reactions

Hypersensitivity

Serious cases of hypersensitivity were more frequent with KENGREAL (7/13301) than with control (2/12861). These included anaphylactic reactions, anaphylactic shock, bronchospasm, angioedema, and stridor.

Decreased renal function

Worsening renal function was reported in 3.2% of KENGREAL patients with severe renal impairment (creatinine clearance <30 mL/min) compared to 1.4% of clopidogrel patients with severe renal impairment.

Dyspnea

Dyspnea was reported more frequently in patients treated with KENGREAL (1.3%) than with control (0.4%).

7 DRUG INTERACTIONS

7.1 Thienopyridines

If clopidogrel or prasugrel are administered during KENGREAL infusion, they will have no antiplatelet effect until the next dose is administered. Clopidogrel and prasugrel, therefore, should not be administered until KENGREAL infusion is discontinued [*see Dosage and Administration (2.2) and Clinical Pharmacology (12.3)*].

8 USE IN SPECIFIC POPULATIONS

8.1 Pregnancy

Pregnancy Category C

There are no adequate and well-controlled studies of KENGREAL in pregnant women.

Cangrelor did not produce malformations in either the rat or rabbit reproductive studies, and is not considered to be a teratogen.

In embryo-fetal development studies in rats, cangrelor produced dose-related fetal growth retardation characterized by increased incidences of incomplete ossification and unossified hind limb metatarsals at plasma concentration of approximately 5 times lower than that achieved in the PCI setting at the maximum recommended human dose (MRHD). In rabbits, cangrelor was associated with increased incidences of abortion and intrauterine losses, as well as fetal growth retardation at plasma concentrations of approximately 12 times higher than the PCI setting at the MRHD.

8.3 Nursing Mothers

It is not known whether KENGREAL is excreted in human milk.

8.4 Pediatric Use

Safety and effectiveness in pediatric patients have not been established.

8.5 Geriatric Use

In CHAMPION PHOENIX, 18% of patients were ≥ 75 years. No overall differences in safety or effectiveness were observed between these patients and those patients < 75 years [see *Clinical Studies (14.1)*].

8.6 Renal Impairment

No dosage adjustment is required for patients with mild, moderate, or severe renal impairment [see *Clinical Pharmacology (12.3)*].

8.7 Hepatic Impairment

KENGREAL has not been studied in patients with hepatic impairment. However, the metabolism of KENGREAL is not dependent of hepatic function, so that dosage adjustment is not required for patients with hepatic impairment [see *Clinical Pharmacology (12.3)*].

10 OVERDOSAGE

There is no specific treatment to reverse the antiplatelet effect of KENGREAL but the effect is gone within one hour after the drug is discontinued.

In clinical trials, 36 patients received an overdose of KENGREAL, ranging from 36 to 300 mcg/kg (bolus dose) or 4.8 to 13.7 mcg/kg/min (infusion dose). The maximum overdose received was 10 times the PCI bolus dose or 3.5 times the PCI infusion dose in 4 patients. No clinical sequela were noted as a result of overdose following completion of KENGREAL therapy.

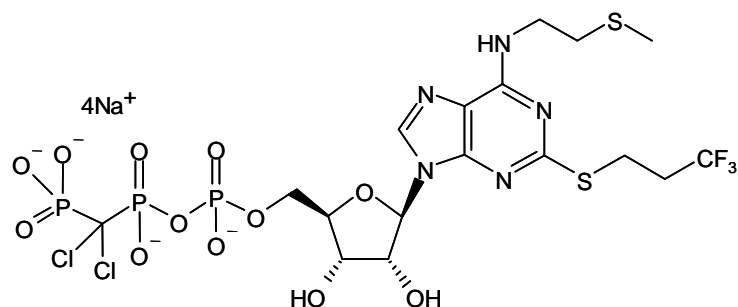
11 DESCRIPTION

KENGREAL is a direct-acting P2Y₁₂ platelet receptor inhibitor that blocks adenosine diphosphate (ADP)-induced platelet activation and aggregation. The chemical structure is similar to adenosine triphosphate (ATP).

The chemical name of KENGREAL is tetrasodium salt of N6-[2-(methylthio)ethyl]-2-[(3,3,3-trifluoropropyl)-5'-adenylic acid, monanhydride with (dichloromethylene) bisphosphonic acid.

The empirical formula of KENGREAL is C₁₇H₂₁N₅C₁₂F₃Na₄O₁₂P₃S₂ and the molecular weight is 864.3 g/mol.

The chemical structure is represented below:



Cangrelor for Injection is a sterile white to off-white lyophilized powder for IV infusion. In addition to the active ingredient, cangrelor, each single use vial contains mannitol (158 mg), sorbitol (52 mg), and sodium hydroxide to adjust the pH.

12 CLINICAL PHARMACOLOGY

12.1 Mechanism of Action

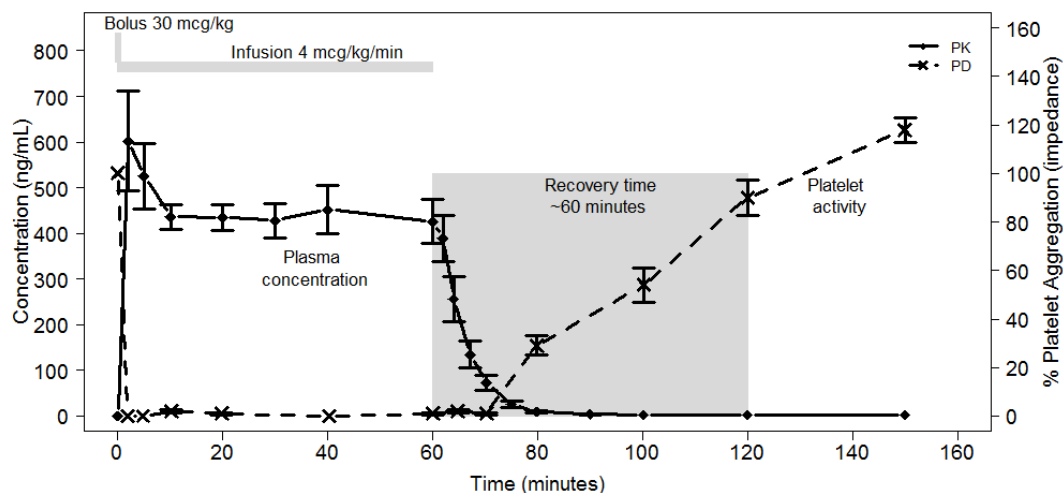
Cangrelor is a direct P2Y₁₂ platelet receptor inhibitor that blocks ADP-induced platelet activation and aggregation. Cangrelor binds selectively and reversibly to the P2Y₁₂ receptor to prevent further signaling and platelet activation.

12.2 Pharmacodynamics

Cangrelor inhibits activation and aggregation of platelets. After administration of a 30 mcg/kg IV bolus followed by a 4 mcg/kg/min IV infusion, platelet inhibition occurs within 2 minutes.

Figure 2 shows the effect on platelet activity, and its relation to cangrelor plasma concentration, of administering a 30 mcg/kg IV bolus, followed by a 1-hour 4 mcg/kg/min IV infusion, of cangrelor. The anti-platelet effect is maintained for the duration of the infusion. After discontinuation of the infusion, the anti-platelet effect decreases rapidly and platelet function returns to normal within 1 hour.

Figure 2: Cangrelor PD Characteristics



12.3 Pharmacokinetics

KENGREAL administered intravenously has linear pharmacokinetics in both healthy volunteers and patients. KENGREAL is rapidly distributed and metabolized, reaching C_{max} within 2 minutes after administration of an intravenous bolus followed by infusion.

Distribution

In a study in healthy volunteers, KENGREAL administration at a dose of 30 mcg/kg bolus plus 4 mcg/kg/min showed a volume of distribution of 3.9 L. Plasma protein binding of KENGREAL is about 97-98%.

Metabolism

KENGREAL is deactivated rapidly in the circulation by dephosphorylation to its primary metabolite, a nucleoside, which has negligible anti-platelet activity. KENGREAL's metabolism is independent of hepatic function and it does not interfere with other drugs metabolized by hepatic enzymes.

Elimination

Following IV administration of [3H] KENGREAL 58% of radioactivity was recovered in urine. The remaining 35% of radioactivity was in feces, presumably following biliary excretion. The average elimination half-life of KENGREAL is about 3-6 minutes.

Specific Populations

KENGREAL pharmacokinetics are not affected by sex, age, renal status or hepatic function. No dose adjustment is needed for these factors [see *Use in Specific Populations (8)*].

Weight

Although weight was a significant covariate for PK with higher clearance in heavier patients, the impact of weight on drug exposure is accounted by the use of weight-based dosing.

Drug-Drug Interactions

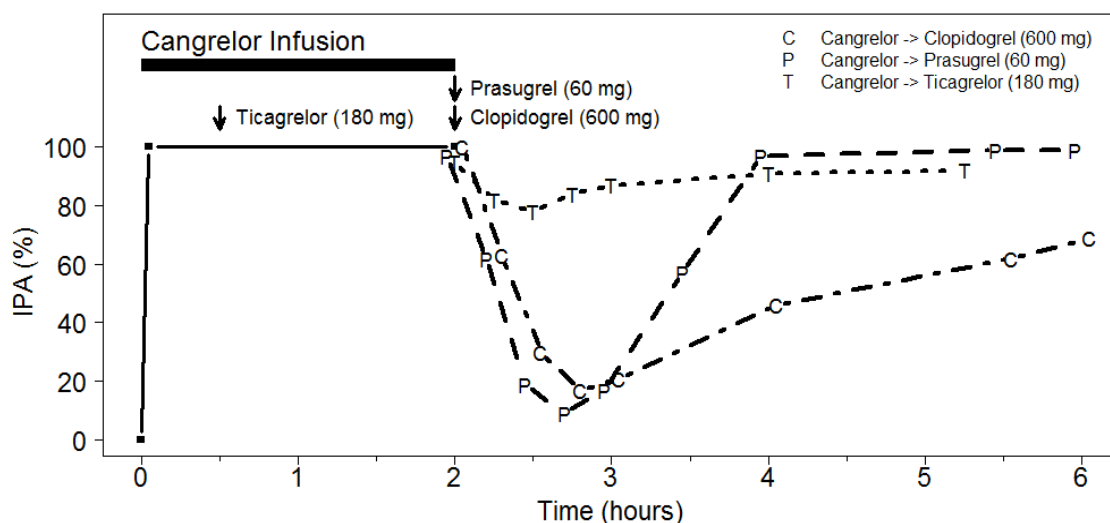
Co-administration of cangrelor with unfractionated heparin, aspirin, and nitroglycerin was formally studied in healthy subjects, with no evidence of an effect on the PK/PD of cangrelor.

In clinical trials cangrelor has been co-administered with bivalirudin, low molecular weight heparin, clopidogrel, prasugrel, and ticagrelor without clinically detectable interactions.

The expected antiplatelet effect of a 600 mg loading dose of clopidogrel or a 60 mg loading dose of prasugrel was blocked when clopidogrel or prasugrel was administered during a cangrelor infusion [see *Drug Interactions (7.1)*].

In contrast, the antiplatelet effect of a 180 mg ticagrelor loading dose was not altered significantly when ticagrelor was administered during cangrelor infusion [see *Drug Interactions (7.1)*].

Figure 3: Inhibition (Mean) of 20 μ M ADP-induced Platelet Aggregation (IPA) Measured by Light Transmission Aggregometry after Cangrelor 30 mcg/kg Bolus and 120-minute 4 mcg/kg Infusion with Transition to Other Oral P2Y₁₂ Platelet Inhibitors.



As shown in Figure 3, discontinuation of cangrelor infusion, followed by administration of the irreversible P2Y₁₂ platelet inhibitors clopidogrel and prasugrel led to a 1-hour decrease in IPA followed by an increase in inhibition of platelet aggregation beginning at about one hour. This time course of platelet inhibition reflects the pharmacokinetics of cangrelor (offset) followed by the absorption and metabolism of clopidogrel and prasugrel to active metabolites (onset).

Administration of ticagrelor, a reversible P2Y₁₂ platelet inhibitor, during the cangrelor infusion led to minimal decrease in platelet inhibition for approximately 0.5 hours following discontinuation of the cangrelor infusion. Administering ticagrelor during cangrelor infusion does not attenuate the anti-platelet effect of ticagrelor.

In vitro studies suggest that neither cangrelor nor its major metabolites inhibit the activity of the hepatic CYP isoenzymes at therapeutic concentrations. Therefore, cangrelor administration is not expected to interfere with the hepatic metabolism of other concomitantly administered therapeutic agents.

13 NONCLINICAL TOXICOLOGY

13.1 Carcinogenesis, Mutagenesis, Impairment of Fertility

Carcinogenesis

No carcinogenicity studies were conducted.

Mutagenesis

Cangrelor was non-mutagenic and non-clastogenic in genetic toxicology studies, including in vitro bacterial gene mutation assay, mouse lymphoma thymidine kinase assay, chromosome aberration assay in human peripheral lymphocytes, and in vivo bone marrow micronucleus assay in mice.

Impairment of Fertility

Cangrelor had no significant effect on male or female rats fertility treated for 28 days, or on early embryonic development at steady state plasma concentration (C_{ss}) of approximately the same as that achieved in the PCI setting at the MRHD.

14 CLINICAL STUDIES

14.1 CHAMPION PHOENIX Trial

The CHAMPION PHOENIX trial was intended to test whether faster platelet inhibition with cangrelor at the time of PCI would reduce the rate of periprocedural thrombotic events compared to a drug with a slower antiplatelet effect, clopidogrel, given at about the time of PCI. It was a randomized, double-blind study in which patients with coronary artery disease (stable angina, UA/NSTEMI, STEMI) requiring PCI and receiving standard therapy including aspirin and heparin or bivalirudin were randomized 1:1 to KENGREAL (n=5472) or to clopidogrel 300 or 600 mg (n=5470). Patients who had already taken an oral P2Y₁₂ platelet inhibitor were not eligible to enroll. Patients administered glycoprotein IIb/IIIa inhibitors (GPI) or for whom GPI use was planned were also not eligible to enroll. PHOENIX was thus a study of people undergoing PCI who had not been previously treated with anti-platelet therapy other than aspirin.

The primary outcome measure was the first occurrence of any one of the composite endpoint of all-cause mortality, myocardial infarction (MI), ischemia-driven revascularization (IDR), and stent thrombosis (ST) within 48 hours after randomization.

KENGREAL was administered as 30 mcg/kg bolus followed by 4 mcg/kg/min infusion for 2 to 4 hours. Clopidogrel 600 mg was administered immediately at the end of the KENGREAL infusion in patients randomized to KENGREAL. Clopidogrel 300 mg or 600 mg was administered shortly before PCI or shortly afterward, in patients randomized to clopidogrel.

KENGREAL significantly reduced the occurrence of primary composite endpoint events compared to clopidogrel (relative risk reduction [RRR] 22%). Most of the effect was a reduction in post-procedural MIs detected solely by elevations in CK-MB (type 4a MI). KENGREAL did not reduce the risk of death. [Table 2](#) shows the study results for the primary composite endpoint and the contribution of each component to the primary endpoint.

Table 2: Primary Endpoint and Its Component Events at 48 Hours in CHAMPION PHOENIX (mITT population^a)

	KENGREAL (N=5470) n (%)	Clopidogrel (N=5469) n (%)	KENGREAL vs. clopidogrel	
			OR (95% CI)	p-value
<u>Primary Endpoint</u> Death/MI/IDR/ST	257 (4.7)	322 (5.9)	0.78 (0.66,0.93) ^b	0.005
Death	18 (0.3)	18 (0.3)		
MI	202 (3.7)	254 (4.6)		
IDR	10 (0.2)	14 (0.3)		
ST	27 (0.5)	36 (0.7)		

Note: if a subject had more than one event at 48 hours, then worst outcome counted (death >MI >IDR >ST)

^aThe mITT population is all randomized subjects who received at least one dose of study drug and underwent the index PCI procedure

^b Based on logistic model adjusted for loading dose and baseline patient status for primary endpoint

A supplementary analysis was also performed omitting two subcomponent events of the primary endpoint that were of lesser clinical significance: intraprocedural stent thrombosis (defined as a new or increasing thrombus within or adjacent to a deployed stent occurring during the index PCI procedure), and myocardial infarction with less than a 10-fold increase in CK-MB, or with less than a 5-fold increase in CK-MB in the presence of new Q waves or new left bundle branch block (LBBB). These results are shown in Table 3.

Table 3: Supplementary Endpoint and Its Component Events at 48 Hours in CHAMPION PHOENIX (mITT population)

n (%)	KENGREAL (N=5470)	Clopidogrel (N=5469)	KENGREAL vs. clopidogrel OR (95% CI)
<u>Supplementary Endpoint</u> Death/SCAI-MI/IDR/ARC-ST	79 (1.4)	114 (2.1)	0.69 (0.52,0.92)
Death	18 (0.3)	18 (0.3)	
SCAI-MI ^a	48 (0.9)	80 (1.5)	
IDR	13 (0.2)	16 (0.3)	
ARC-ST ^b	0 (0.0)	0 (0.0)	

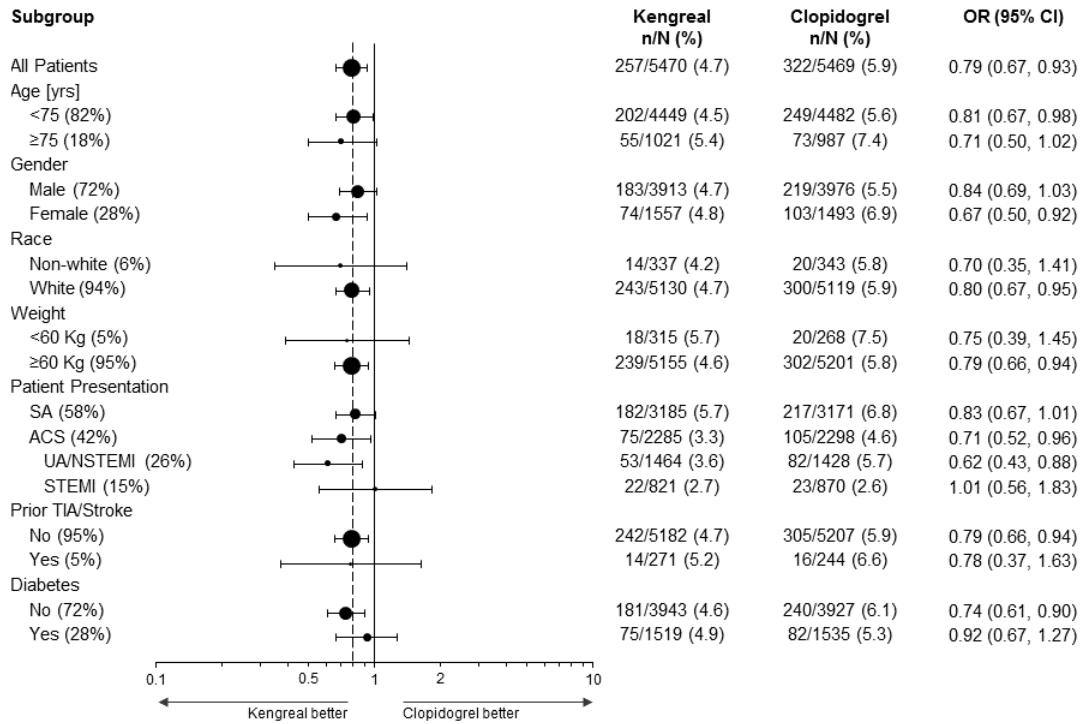
Note: if a subject had more than one event at 48 hours, then worst outcome counted (death >SCAI-MI >IDR >ARC-ST)

^a SCAI MI: CK-MB $\geq 10X$ ULN, or CK-MB $\geq 5X$ ULN with new Q waves or new LBBB

^b ARC-ST defined according to the ARC definition [Cutlip et al. 2007]

The effect of KENGREAL appeared to be consistent in a variety of pre-specified and other clinically important subgroups (see Figure 4).

Figure 4: CHAMPION PHOENIX Study: Primary Efficacy Endpoint by Subgroup (mITT Population^a)



^a The mITT population is all randomized subjects who received at least one dose of study drug and underwent the index PCI procedure.

Note: The figure above presents effects in various subgroups most of which are baseline characteristics and most of which were pre-specified (patient presentation and weight were not pre-specified subgroups). The 95% confidence limits that are shown do not take into account how many comparisons were made, nor do they reflect the effect of a particular factor after adjustment for all other factors. Apparent homogeneity or heterogeneity among groups should not be over-interpreted.

14.2 CHAMPION PCI and PLATFORM Trials

Two additional concurrent clinical trials of the effect of KENGREAL in patients undergoing percutaneous coronary intervention, CHAMPION PCI and CHAMPION PLATFORM were conducted and terminated early for futility. They were completed prior to the design and conduct of CHAMPION PHOENIX. The comparative characteristics and outcomes of each trial are shown in [Table 4](#).

Table 4: Summary of the CHAMPION Trials

		PCI	PLATFORM	PHOENIX
Subjects Randomized (% of planned enrollment)		8,846 (99%)	5,346 (84%)	11,145 (100%)
Primary Endpoint at 48 hours		Death, MI, or IDR	Death, MI, or IDR	Death, MI, IDR, or ST
Outcome of primary analysis, OR (95% CI)		1.05 (0.88, 1.24)	0.87 (0.71, 1.07)	0.78 (0.66, 0.93)
Clopidogrel dose and time in clopidogrel arm		600 mg immediately before PCI	600 mg immediately after PCI	300 or 600 mg shortly before or shortly after PCI
Population enrolled (%)	Stable angina	15%	5%	58%
	UA/NSTEMI	74%	95%	26%
	STEMI	11%	Excluded	16%

16 HOW SUPPLIED/STORAGE AND HANDLING

KENGREAL is supplied as a sterile lyophilized powder in single-use 10 mL vials.

- NDC # 10122-620-01: 10 mL vial containing 50 mg cangrelor
- NDC # 10122-620-10: 10 count of 10 mL vials containing 50 mg cangrelor

Vials of KENGREAL should be stored at USP Controlled Room Temperature, [20°C to 25°C (68°F to 77°F) with excursions between 15°C and 30°C (59°F and 86°F) permitted].

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