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ADISINSIGHT REPORT

Recaticimab: First Approval

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Abstract
Recaticimab (艾心安®), a humanized monoclonal immunoglobulin G1 antibody that targets proprotein convertase subtilisin/kexin type 9 (PCSK9), is being developed by Suzhou Suncadia Biopharmaceutical for the treatment of hypercholesteremia and mixed dyslipidemia. Recaticimab received its first approval on 8 January 2025 in China, as an adjunct to diet, in combination with statins (with or without other lipid-lowering therapies) in adults with primary hypercholesterolemia (including heterozygous familial and non-familial hypercholesterolemia) and mixed dyslipidemia who have not achieved their low-density lipoprotein cholesterol (LDL-C) target despite receiving moderate or higher doses of statins, and for use as monotherapy in adults with non-familial hypercholesterolemia and mixed dyslipidemia to reduce LDL-C, total cholesterol, and apolipoprotein B levels. This article summarizes the milestones in the development of recaticimab leading to this first approval for hypercholesterolemia and mixed dyslipidemia.

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Recaticimab (艾心安®): Key Points

A PCSK9 monoclonal antibody is being developed by Suzhou Suncadia Biopharmaceutical for the treatment of hypercholesterolemia and mixed dyslipidemia

Received its first approval on 8 January 2025 in China

Approved for use in combination with statins (± other lipid-lowering therapies) in adults with primary hypercholesterolemia and mixed dyslipidemia who have not achieved their LDL-C target despite statin therapy, and as monotherapy in adults with non-familial hypercholesterolemia and mixed dyslipidemia

1 Introduction

Recaticimab (艾心安®) is a humanized monoclonal antibody against proprotein convertase subtilisin/kexin type 9 (PCSK9) being developed by Suzhou Suncadia Biopharmaceutical Co., Ltd (a subsidiary of Jiangsu Hengrui Pharmaceuticals Co., Ltd) for the treatment of hypercholesteremia and mixed dyslipidemia [1, 2]. Hypercholesterolemia is a major direct risk factor for atherosclerotic cardiovascular disease (ASCVD), which remains the leading cause of mortality worldwide [3]. Statins, which effectively reduce serum low-density lipoprotein cholesterol (LDL-C), are fundamental to cardiovascular risk reduction; however, many patients are statin intolerant, poorly adherent to statins, or unable to achieve their LDL-C goal despite high-intensity statin therapy [3–5]. In Asian populations, high-intensity statins are often poorly tolerated, with limited LDL-C reduction (only 6% more when the dose is doubled) [6]. The Chinese Guidelines for Lipid Management (2023) recommend starting with moderate-intensity statins, and if LDL-C remains uncontrolled, combination therapy is preferred over high-dose statins [7]. A more recent target for preventative treatment is the PCSK9, which contributes to cardiovascular events by promoting the degradation of the LDL receptor (LDLR) and reducing the clearance of LDL-C from the blood [3, 5]. While current anti-PCSK9 monoclonal antibodies potently lower LDL-C, the need for relatively frequent subcutaneous injections (i.e., every 2–4 weeks) may limit patient acceptability [8]. Recaticimab, a novel long-acting PCSK9 inhibitor, provides patients with a new lipid-lowering treatment option [2, 9].

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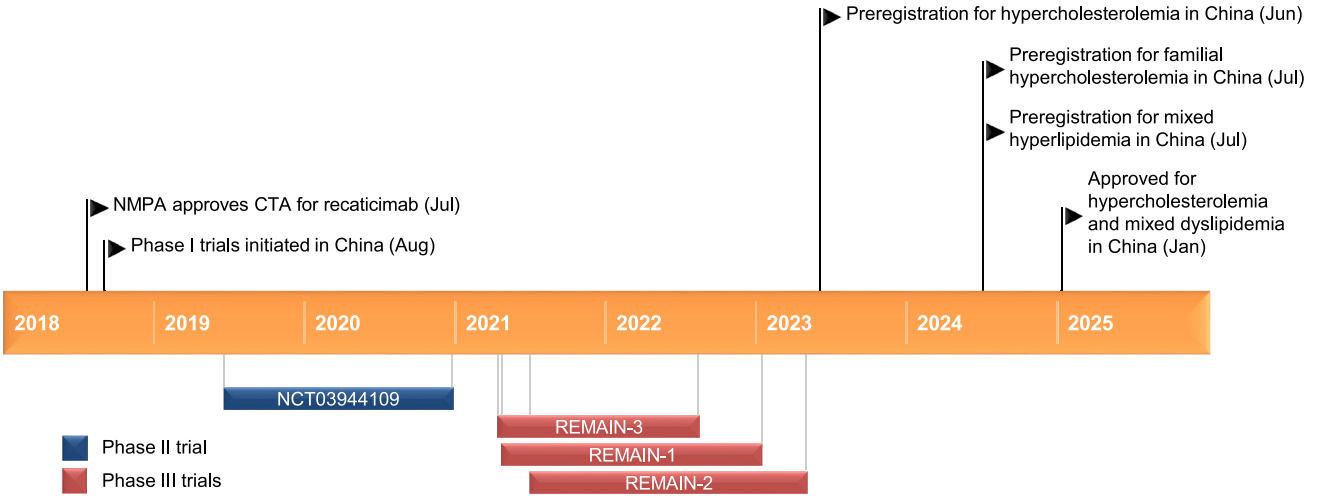

Key milestones in the development of recaticimab for the treatment of hypercholesterolemia and mixed dyslipidemia. *NMPA* National Medical Products Administration, *CTA* clinical trial application

Recaticimab received its first approval on 8 January 2025 in China, as an adjunct to diet, for use in combination with statins (with or without other lipid-lowering therapies) in adults with primary hypercholesterolemia (including heterozygous familial and non-familial hypercholesterolemia) and mixed dyslipidemia who have not achieved their LDL-C target despite receiving moderate or higher doses of statins, and for use as monotherapy in adults with non-familial hypercholesterolemia and mixed dyslipidemia to reduce LDL-C, total cholesterol (TC), and apolipoprotein B (ApoB) levels [1, 10]. Approval was based on the positive results of three phase III registrational clinical trials (REMAIN-1, REMAIN-2, and REMAIN-3) [2, 9].

Recaticimab is administered by subcutaneous injection into the abdomen, thigh, or upper arm [10]. Injection site rotation is recommended. In patients with non-familial hypercholesterolemia and mixed hyperlipidemia, the recommended dose is 150 mg once every 4 weeks (Q4W) or 300 mg once every 8 weeks (Q8W). The flexible regimen can be selected based on patient preference and clinician guidance. When switching, the new regimen may be administered on the next scheduled dosing date of the original regimen. In patients with heterozygous familial hypercholesterolemia, the recommended dose is 150 mg Q4W [10]. Recaticimab is the longest-acting anti-PCSK9 antibody globally, allowing dosing interval of up to 8 weeks (12 injections per year). This approach does not increase the total drug dosage and avoids imposing an additional economic burden on patients. Additionally, it is the only PCSK9 inhibitor approved in China for monotherapy, without restrictions to patients with statin intolerance or contraindications.

2 Scientific Summary

2.1 Pharmacodynamics

Recaticimab is a humanized immunoglobulin G1 (IgG1) monoclonal antibody that selectively binds to circulating PCSK9 with high affinity [10–12]. By binding to circulating PCSK9, recaticimab prevents PCSK9 from binding to LDLR and inhibits subsequent PCSK9-mediated LDLR degradation [10]. This results in more LDLRs being available on the surface of hepatocytes to clear low-density lipoproteins from circulation and, in turn, reduced serum LDL-C levels [10, 13]. Following administration of a single subcutaneous dose of recaticimab (150 mg or 300 mg), the level of free PCSK9 in the body continued to decrease for ≈ 48 h before gradually returning to baseline level [10]. A single dose of recaticimab (51–450 mg) in healthy volunteers produced a 50–65% reduction in serum LDL-C levels in a phase I trial [11]. Recaticimab contains a strategically introduced YTE mutation within its Fc region which enhances its affinity for the neonatal Fc receptor (FcRn), protecting the drug from FcRn-mediated antibody catabolism and allowing for durable effects [12, 14]. The YTE-mutated recaticimab shows an affinity for FcRn that is 10 times higher compared to both the wild-type and other anti-PCSK9 antibodies [15].

Across phase I to III clinical trials of recaticimab ($n = 1{,}046$ recipients analyzed for immunogenicity), the anti-drug antibody (ADA) incidence during treatment was 14.1% and the neutralizing antibody incidence was 3.2% [10]. Immunogenicity did not impact recaticimab pharmacokinetics, efficacy, or safety [10]. The serum exposure of recaticimab was similar between ADA-positive and ADA-negative patients across various dose regimens [11]. At week 24, there was no significant difference

in LDL-C reduction between ADA-positive and ADA-negative patients in both 150 mg Q4W and 300 mg Q8W regimens, with generally similar trends through week 48 [14]. The overall incidence of treatment-related adverse events was similar between ADA-positive and ADA-negative patients [14].

2.2 Pharmacokinetics

Following the initial subcutaneous injection of recaticimab 150 mg or 300 mg, the median time to peak serum concentration is ≈ 7 to 9 days [10, 11]. When recaticimab is administered at a dose of 150 mg Q4W or 300 mg Q8W, steady state of serum recaticimab concentration is achieved after 12–16 weeks [10]. At steady state, the geometric mean of the apparent volume of distribution is 8.28–8.64 L. Drug accumulation based on serum exposure is about 2-fold with recaticimab 150 mg Q4W, while little accumulation is observed with 300 mg Q8W [10].

As a humanized IgG1 monoclonal antibody, recaticimab undergoes target-mediated clearance at low concentrations and is primarily eliminated via a non-saturable proteolytic pathway at high concentrations [12]. The enhanced binding of recaticimab to FcRn protects recaticimab from clearance via the proteolytic pathway, thereby extending its half-life [12]. The geometric mean apparent clearance of recaticimab was 0.254–0.271 L/day and the geometric mean elimination half-life was 22–27 days [10].

No dosage adjustments are required for patients with mild to moderate renal impairment or mild to moderate hepatic impairment [10]. Recaticimab should be used with caution in patients with severe renal or hepatic impairment, as no data are available [10].

Based on population pharmacokinetic analysis, neither background statins nor ezetimibe are expected to cause clinically significant reductions in recaticimab exposure [10].

2.3 Therapeutic Trials

2.3.1 Phase III

2.3.1.1 Monotherapy for Non-familial Hypercholesterolemia and Mixed Hyperlipidemia Recaticimab monotherapy was effective in reducing LDL-C in patients with non-familial hypercholesterolemia and mixed hyperlipidemia at low-to-moderate ASCVD risk in a multicenter, randomized, double-blind, placebo-controlled, phase III REMAIN-1 trial (NCT04849000) conducted in China [12]. REMAIN-1 enrolled adults with a fasting LDL-C level of ≥ 2.6 to < 4.9 mmol/L, fasting triglyceride level of ≤ 5.6 mmol/L, and a 10-year ASCVD risk score of < 10%. Patients were required to follow a lipid-lowering diet from the start of the 4- to 6-week run-in period and throughout the study. For the core treatment period, patients were randomized to receive subcutaneous injections of recaticimab at 150 mg Q4W ($n = 157$ treated), 300 mg Q8W ($n = 156$), or 450 mg every 12 weeks (Q12W; $n = 155$), or matching placebo ($n = 78$, 79, and 78, respectively). Randomization was stratified by LDL-C level (< 3.4 vs ≥ 3.4 mmol/L) [12].

Relative to placebo, recaticimab further reduced LDL-C by 49.6% (95% CI 44.2–54.9) at 150 mg Q4W, 52.8% (95% CI 48.3–57.2) at 300 mg Q8W, and 45.0% (95% CI 41.0–49.0) at 450 mg Q12W from baseline to the end of the double-blind core treatment period (defined as week 12 for the Q4W and Q12W groups and week 16 for the Q8W group) [$p < 0.0001$ for all comparisons; primary endpoint] [12]. With all dosing strategies, the effects of recaticimab on the primary endpoint were consistent across subgroups based on age (< 65 and ≥ 65 years), body mass index (< 24 and ≥ 24 kg/m^2), and baseline LDL-C (< 3.4 and ≥ 3.4 mmol/L). The least-squares mean (LSM) absolute changes in LDL-C relative to placebo were − 1.8 mmol/L, − 1.9 mmol/L, and

Features and properties of recaticimab

Alternative names	SHR-1209
Class	Antihyperlipidemics, monoclonal antibodies
Mechanism of action	PCSK9 protein inhibitor
Route of administration	Subcutaneous
Pharmacodynamics	Selectively binds to circulating PCSK9 with high affinity, preventing PCSK9 from binding to LDLR and inhibiting PCSK9-mediated LDLR degradation; produced a 50–65% reduction in serum LDL-C levels; strategic YTE mutation within its Fc region allows for durable effects
Pharmacokinetics	Peak serum concentration reached in ≈ 7 to 9 days following initial subcutaneous injection at the recommended dose; apparent clearance 0.254–0.271L/day; elimination half-life 22–27 days
Adverse reactions	Abnormal liver enzymes, injection site reactions, hyperglycemia, hyperbilirubinemia
ATC codes	
WHO ATC code	C10A-X (other lipid modifying agents)
EphMRA ATC code	C10A9 (all other cholesterol/triglyceride regulators)

LDL-C low-density lipoprotein cholesterol, *LDLR* LDL receptor, *PCSK9* proprotein convertase subtilisin/kexin type 9

− 1.6 mmol/L with recaticimab 150 mg Q4W, 300 mg Q8W, and 450 mg Q12W, respectively. More patients in the recaticimab groups achieved LDL-C < 2.6 mmol/L compared with placebo: 88.2 vs 7.5% (150 mg Q4W), 91.0 vs 8.2% (300 mg Q8W), and 86.9 vs 3.9% (450 mg Q12W). With respect to effects on other lipids, all recaticimab dosing strategies were associated with significant ($p < 0.0001$) reductions in non-HDL-C (percentage difference vs placebo: − 46.3, − 47.3, − 43.4% with 150 mg Q4W, 300 mg Q8W, and 450 mg Q12W, respectively), ApoB (− 43.6, − 46.0, − 40.3%), lipoprotein(a) (− 32.4, − 28.5, − 18.3%), TC/HDL-C (− 38.2, − 35.3, − 36.6%), ApoB/apolipoprotein A1 (ApoA1; − 45.7, − 47.0, − 43.8%). Recaticimab continued to be efficacious in recaticimab recipients who went on to receive open-label recaticimab until week 24 and also demonstrated efficacy in placebo recipients who switched to recaticimab for the open-label period [12].

2.3.1.2 Add-on to Statin for Non-familial Hypercholesterolemia and Mixed Hyperlipidemia Recaticimab as an add-on to statin therapy effectively reduced LDL-C levels in patients with non-familial hypercholesterolemia in the multicenter, randomized, double-blind, placebo-controlled, phase III REMAIN-2 trial (NCT04885218) [14]. After a run-in period during which patients received stable moderate- or high-intensity statin therapy (± ezetimibe or fenofibrate) for ≥ 4 weeks and were advised to follow a low-fat diet, eligible patients (those with an LDL-C of ≥ 1.8 mmol/L if ASCVD present or ≥ 2.6 mmol/L otherwise, a fasting triglyceride level of ≤ 5.6 mmol/L, and adequate treatment compliance) entered a randomized treatment period. Patients were assigned to subcutaneous recaticimab 150 mg Q4W ($n = 153$ treated), 300 mg Q8W ($n = 151$), or 450 mg Q12W ($n = 152$), or matching placebo ($n = 78$, 77, and 78, respectively), for 48 weeks. Randomization was stratified by concomitant use of ezetimibe (yes vs no) and LDL-C level at the end of the run-in period (≥ 2.6 vs < 2.6 mmol/L) [14].

Relative to placebo, all recaticimab dosing strategies significantly improved the LSM percentage change in LDL-C from baseline to week 24 ($p < 0.0001$ for all comparisons; primary endpoint); the LSM treatment difference was − 62.2% (95% CI − 67.0 to − 57.4) for the 150 mg Q4W regimen, − 59.7% (95% CI − 65.0 to − 54.4) for the 300 mg Q8W regimen, and − 53.4% (95% CI − 58.7 to − 48.2) for the 450 mg Q12W regimen [14]. The LSM absolute LDL-C level was reduced by a further 1.7, 1.7, and 1.5 mmol/L with recaticimab 150 mg Q4W, 300 mg Q8W, and 450 mg Q12W, respectively, relative to placebo. The LDL-C reduction with recaticimab was generally consistent across various patient subgroups, regardless of age (< 65 and ≥ 65 years), body mass index (< 24 and ≥ 24 kg/m^2), baseline LDL-C level (< 2.6 and ≥ 2.6 mmol/L), background lipid-lowering therapy (statins, ezetimibe), and the presence or absence of a history of ASCVD or type 2 diabetes mellitus (T2DM). For patients with a history of ASCVD, the treatment difference in LDL-C reductions ranged from − 54 to − 62%, compared with − 54 to − 65% for those without ASCVD across all recaticimab groups. For patients with T2DM, reductions ranged from − 54 to − 63%, compared with − 53 to − 62% for those without T2DM. At week 24, 85.8–94.5% of patients in the recaticimab groups achieved the LDL-C target, with similar rates in those with (84.4–94.3%) and without ASCVD (87.8–95.0%), compared with 13.9–15.9% in the placebo groups. Secondary lipid variables, including non-HDL-C, TC/HDL-C, ApoB, ApoB/ApoA1, and lipoprotein(a), were also significantly improved with recaticimab regimens relative to placebo ($p < 0.0001$ for all comparisons at week 24). The LSM treatment differences with recaticimab 150 mg Q4W, 300 mg Q8W, and 450 mg Q12W versus placebo were: − 56.6, − 53.4, and − 47.6% for non-HDL-C; − 53.3, − 50.5, and − 44.4% for ApoB; and − 36.1, − 28.1, and − 28.7% for lipoprotein(a) [14].

LDL-C reductions were sustained over longer-term treatment [14]. For LSM percentage change in LDL-C from baseline to week 48, the differences versus placebo were − 60.1% (95% CI − 67.7 to − 52.5) for 150 mg Q4W, − 64.0% (95% CI − 71.0 to − 57.1) for 300 mg Q8W, and − 48.4% (95% CI − 55.1 to − 41.7) for 450 mg Q12W [$p < 0.0001$ for all comparisons]. The proportion of patients achieving their LDL-C goal was sustained until week 48 [14].

2.3.1.3 Add-on to Stable Lipid-Lowering Therapy for Heterozygous Familial Hypercholesterolemia As an add-on to stable lipid-lowering therapy, recaticimab was effective in lowering LDL-C in adults with heterozygous familial hypercholesterolemia with poorly controlled LDL-C despite intensive lipid-lowering therapy in the multicenter, randomized, double-blind, placebo-controlled, phase III REMAIN-3 trial (NCT04844125) [16]. Patients enrolled in REMAIN-3 had been on stable lipid-lowering therapy for ≥ 28 days, had an LDL-C level of ≥ 2.6 mmol/L (≥ 1.8 mmol/L if they had a history of ASCVD), and fasting triglyceride level of ≤ 5.6 mmol/L. They were randomized to receive subcutaneous recaticimab 150 mg Q4W ($n = 95$) or matching placebo ($n = 48$) for 12 weeks. Randomization was stratified by concomitant use of ezetimibe (yes vs no) and LDL-C level (≥ 4.1 vs < 4.1 mmol/L) [16].

At week 12, the LSM percentage change from baseline in LDL-C was − 54.4% (95% CI − 57.9 to − 50.8) in recaticimab recipients versus − 4.5% (95% CI − 9.4 to 0.3) in placebo recipients, a treatment difference of − 49.8% (95% CI − 55.8 to − 43.9; $p < 0.0001$) [primary endpoint]. Recaticimab was also associated with significant reductions in non-HDL-C, ApoB, TC/HDL-C, ApoB/ApoA1,

and lipoprotein(a) relative to placebo ($p < 0.0001$ for all comparisons) [16].

2.3.2 Phase Ib/II

Recaticimab as an add-on to moderate-intensity statin therapy provided significant and substantial reductions in LDL-C level in patients with hypercholesterolemia in a multicenter, randomized, double-blind, placebo-controlled, phase Ib/II trial (NCT03944109) [11]. Patients in this trial were receiving stable dose of atorvastatin (10 or 20 mg/day) and had an LDL-C level ≥ 2.6 mmol/L, a fasting triglyceride level of ≤ 4.5 mmol/L, and a body mass index of 18–35 kg/m^2. Patients with homozygous familial hypercholesterolemia were not eligible. Patients were randomized to subcutaneous injections of recaticimab (75 mg Q4W, 150 mg Q4W, 150 mg Q8W, 300 mg Q8W, 300 mg Q12W, or 450 mg Q12W; overall, $n = 91$ treated) or placebo ($n = 19$) [11].

Relative to placebo, the LSM percentage reductions in LDL-C from baseline to end of treatment ranged from − 48.37 to − 59.51% across the recaticimab doses and schedules (all $p < 0.0001$ vs placebo; primary endpoint) [11]. End of treatment was defined as week 16 for the Q4W and Q8W groups and week 24 for the Q12W groups. Rapid LDL-C reductions were observed in all recaticimab groups after the initial administration, with a sharp decrease as early as day 7. During the first cycle, the maximum reduction occurred at week 4 in the 150 mg Q4W (− 56.74%) and 300 mg Q8W (− 59.27%) groups [11].

2.4 Adverse Events

Recaticimab had a safety profile generally comparable to that of placebo across clinical trials in patients with primary hypercholesterolemia and mixed hyperlipidemia [11, 12, 14, 16]. Pooled safety data are available from across five clinical studies (four of which were placebo controlled) in which a total of 1,092 patients with primary hypercholesterolemia and mixed hyperlipidemia received recaticimab ($n = 437$, 327, and 328 at 150 mg Q4W, 300 mg Q8W, and 450 mg Q12W, respectively; $n = 426$ exposed for ≥ 24 weeks and 345 exposed for ≥ 48 weeks) and 535 received placebo [10]. The most common adverse reactions (incidence ≥ 1% with recaticimab and at a higher frequency than with placebo) were liver enzyme abnormalities (4.4% with recaticimab vs 3.4% with placebo), injection site reactions (3.8 vs 1.3%), hyperglycemia (2.4 vs 0.7%; includes increased glycosylated hemoglobin, decreased glucose tolerance, increased blood glucose, and abnormal blood glucose), and hyperbilirubinemia (1.1 vs 0.6%). Adverse reactions led to permanent treatment discontinuation in 0.5% of recaticimab recipients versus 0.2% of placebo recipients, with injection site reactions being the predominant cause in recaticimab recipients [10].

In the pooled analysis, injection site reactions were reported in 3.8% of recaticimab recipients versus 1.3% of placebo recipients [10]. The most common were injection site rash/redness (2.0% of recaticimab recipients vs 0.8% of placebo recipients) and injection site pruritus (2.0 vs 0.0%). In recaticimab recipients, injection site reactions were typically transient and of mild severity; there were no severe injection site reactions. Allergic reactions were relatively infrequent (1.2% of recaticimab recipients vs

Key clinical trials of recaticimab (Suzhou Suncadia Biopharmaceutical)

Drug(s)	Indication	Phase	Status	Location(s)	Identifier
Recaticimab, PL	Non-familial hypercholesterolemia and mixed hyperlipidemia	III	Completed	China	REMAIN-1; NCT04849000; SHR-1209-301
Recaticimab, PL	Non-familial hypercholesterolemia (pts receiving stable statin therapy)	III	Completed	China	REMAIN-2; NCT04885218; SHR-1209-302
Recaticimab, PL	Heterozygous familial hypercholesterolemia (pts receiving stable lipid-lowering therapy)	III	Completed	China	REMAIN-3; NCT04844125; SHR-1209-303
Recaticimab	Homozygous familial hypercholesterolemia	II	Completed	China	NCT04455581; SHR-1209-201
Recaticimab, PL	Hypercholesterolemia (pts receiving stable statin therapy)	Ib/II	Completed	China	NCT03944109; SHR-1209-102

PL placebo, *pts* patients

0.4% of placebo recipients) and the most common of these was rash (0.5 vs 0.2%). Myalgia, which occurred in 0.3% of recaticimab recipients and in no placebo recipients, was mild and transient [10].

2.5 Ongoing Clinical Trials

A real-world multicenter cohort study (ELITE-ACS; NCT06738758) is evaluating the impact of early intensive lipid-lowering therapy with PCSK9 inhibitors, including recaticimab, on the prognosis of Chinese patients with acute coronary syndrome. A randomized, open-label, multicenter phase 4 trial (PISTIAS-2; NCT06902740) is evaluating the efficacy of intensive lipid-lowering therapy with recaticimab plus statin versus statin alone in reversing asymptomatic intracranial atherosclerotic plaques in Chinese patients. An open-label, multicenter phase 4 trial (ChiCTR2500099939) is evaluating the long-term efficacy and safety of recaticimab in Chinese patients with ASCVD or at high risk for ASCVD who have not achieved their lipid goals.

3 Current Status

Recaticimab received its first approval on 8 January 2025 in China, for use in combination with statins (with or without other lipid-lowering therapies) in adults with primary hypercholesterolemia and mixed dyslipidemia who have not achieved their LDL-C target despite receiving moderate or higher doses of statins, and as monotherapy in adults with non-familial hypercholesterolemia and mixed dyslipidemia to reduce LDL-C, TC, and ApoB levels [1].

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Declarations

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Ethics approval, Consent to participate, Consent to publish, Availability of data and material, Code availability Not applicable.

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