ISC 2026:
Results from the OCEANIC-STROKE Phase III trial

Investor Webinar

Friday 6 February,
15:00 – 16:00 CET

Today's Agenda

Welcome

Alexander Siedler,
Investor Relations Officer at Bayer

Prepared Remarks

Christoph Koenen,
Global Head of Clinical Development & Operations at Bayer

Ashkan Shoamanesh,
Associate Professor of Medicine (Neurology), Marta and Owen Boris Chair in Stroke Research and Care at McMaster University

Jan F. Voss,
Senior Vice President, Global Head Asundexian at Bayer

Moderated Q&A

Cautionary Statements Regarding Forward-Looking Information

This presentation may contain forward-looking statements based on current assumptions and forecasts made by Bayer management.

Various known and unknown risks, uncertainties and other factors could lead to material differences between the actual future results, financial situation, development or performance of the company and the estimates given here. These factors include those discussed in Bayer's public reports which are available on the Bayer website at

https://www.bayer.com/

The company assumes no liability whatsoever to update these forward-looking statements or to conform them to future events or developments.

Bayer's Vision for Secondary Stroke Prevention

Christoph Koenen
Global Head of Clinical Development & Operations at Bayer

Each year, approximately 12 million people worldwide will experience a stroke[1]

Stroke is also the **third-leading** cause of death…

and **second-leading** cause of disability …[1]

Approximately **101 million** people worldwide are living with the consequences of stroke[2]

1. World Stroke Organization. Global Fact Sheet 2025. 2025. Available at: https://pmc.ncbi.nlm.nih.gov/articles/PMC11786524/ Accessed: December 2025.
2. Feigin VL, Owolabi MO, Abd-Allah F, Akinyemi RO, Bhattacharjee NV, Brainin M, Cao J, Caso V, Dalton B, Davis A, Dempsey R. Pragmatic solutions to reduce the global burden of stroke: a World Stroke Organization–Lancet Neurology Commission. The Lancet Neurology. 2023 Dec 1;22(12):1160-206.

The risk of secondary stroke remains unacceptably high, despite available secondary stroke prevention strategies

Secondary strokes are more devastating than first strokes, with greater risk of disability, long-term cognitive decline, mortality, and higher healthcare costs than first strokes.

20-30%

of strokes are recurrent[1]

Mortality rate increases with each recurrent stroke and there's higher **risk of dementia**[2,3,4]

~1 in 5

ischemic stroke survivors will have another stroke within 5 years

~1 in 10

ischemic stroke survivors experience another stroke in 1 year, even with available treatment options[2]

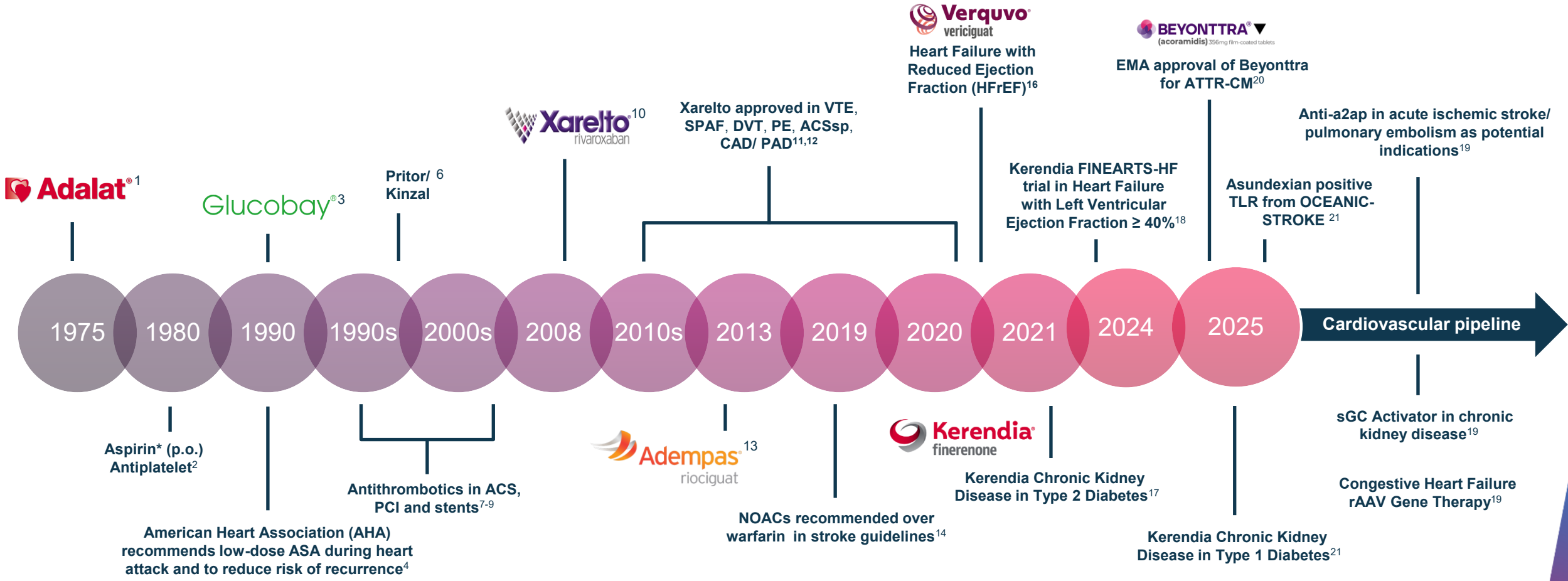
1. World Stroke Organization. Global Fact Sheet 2025. 2025. Available at: https://pmc.ncbi.nlm.nih.gov/articles/PMC11786524/ Accessed: December 2025.
2. Feigin VL, Owolabi MO, Abd-Allah F, Akinyemi RO, Bhattacharjee NV, Brainin M, Cao J, Caso V, Dalton B, Davis A, Dempsey R. Pragmatic solutions to reduce the global burden of stroke: a World Stroke Organization–Lancet Neurology Commission. The Lancet Neurology. 2023 Dec 1;22(12):1160-206.

Current Prevention Strategies Are Limited By Associated Bleeding Risk

Recurrence rate of stroke is unchanged over >20 years, despite available secondary stroke prevention strategies[1]

World Stroke Organization, Impact of Stroke. Available at: https://www.world-stroke.org/world-stroke-day-campaign/about-stroke/impact-of-stroke. Accessed: March 2024.

Bayer's Heritage in Cardiovascular Disease



*Year ASA approved for secondary stroke prevention. Acute coronary syndrome (ACS), acute coronary syndrome secondary prevention (ACSsp), coronary artery disease (CAD), deep vein thrombosis (DVT), novel oral anticoagulants (NOACs), peripheral artery disease (PAD), percutaneous coronary intervention (PCI), pulmonary embolism (PE), stroke prevention in atrial fibrillation (SPAF), venous thromboembolism pharmacoprophylaxis (VTEp).

1. Kvesic DZ. Journal of Medical Marketing. 2009;9(3):187-200. 2. Ugurlucan M et al. Recent Pat Cardiovasc Drug Discov 2012;7:71–76. 3. Asano N. Glycobiology. 2003;13(10):93R-104R. 4. Gunnar RM et al. Circulation. 1990 Aug;82(2):664-707. 5. Taylor AA et al. The Journal of Clinical Hypertension. 2011;13(9):677-86. 6. European Medicines Agency. Pritor. Available at: https://www.ema.europa.eu/en/medicines/human/EPAR/pritor. Accessed April 2024. 7. Watson RD et al. Bmj. 2002;325(7376):1348-51.). 8. Bode C and Huber K. European heart journal supplements. 2008 ;10(suppl_A):A13-20. 9. Leon MB et al. New England Journal of Medicine. 1998;339(23):1665-71. 10. Perzborn E et al. Nature Reviews Drug Discovery. 2011;10(1):61-75. 11. Singh R, Emmady PD. Rivaroxaban. InStatPearls [Internet] 2022. StatPearls Publishing. 12. Hospital Pharmacy Europe. Xarelto® approved for the treatment of PE and DVT. Available at: https://hospitalpharmacyeurope.com/news/editors-pick/xarelto-approved-for-the-treatment-of-pe-and-dvt/. Accessed April 2024. 13. Fierce Biotech. FDA approves Adempas to treat pulmonary hypertension. Available at: https://www.fiercebiotech.com/biotech/fda-approves-adempas-to-treat-pulmonary-hypertension. Accessed April 2024. Bayer. 14. January CT et al. Circulation. 2019;140(2):e125-51. 15. ClinicalTrials.gov. Efficacy and Safety of Finerenone in Subjects With Type 2 Diabetes Mellitus and Diabetic Kidney Disease (FIDELIO-DKD). Available at: https://www.clinicaltrials.gov/study/NCT02540993. Accessed April 2024. 16. Food and Drug Administration. VERQUVOTM (vericiguat) tablets, for oral use. Available at: https://www.accessdata.fda.gov/drugsatfda_docs/label/2021/214377s000lbl.pdf. Accessed April 2024. 17. Food and Drug Administration. KERENDIA (finerenone) tablets, for oral use. Available at: https://www.accessdata.fda.gov/drugsatfda_docs/label/2021/215341s000lbl.pdf. Accessed April 2024. 18. ClinicialTrials.gov. Study to Evaluate the Efficacy (Effect on Disease) and Safety of Finerenone on Morbidity (Events Indicating Disease Worsening) & Mortality (Death Rate) in Participants With Heart Failure and Left Ventricular Ejection Fraction (Proportion of Blood Expelled Per Heart Stroke) Greater or Equal to 40% (FINEARTS-HF). Available at: https://www.clinicaltrials.gov/study/NCT04435626. Accessed April 2024. 19. Bayer. Development Pipeline. Available at: https://www.bayer.com/en/pharma/development-pipeline. Accessed April 2024. 20. Bayer strengthens pharma portfolio with new cardiology drug acoramidis, Available at: https://www.bayer.com/media/en-us/bayer-strengthens-pharma-portfolio-with-new-cardiology-drug-acoramidis/. Accessed April 2024 21. Bayer. Bayer's Asundexian Met Primary Efficacy and Safety Endpoints in Landmark Phase III OCEANIC-STROKE Study in Secondary Stroke Prevention. Bayer Newsroom. Available at: https://www.bayer. com/en/us/news-stories/oceanic-stroke Accessed November 2025. 21. Bayer. KERENDIA® (finerenone) Meets Primary Endpoint in Phase III Clinical Trial for Adults with Type 1 Diabetes and Chronic Kidney Disease. https://www.bayer.com/en/us/news-stories/kerendia-for-type-1-diabetes-and-chronic-kidney-disease. Accessed December 2025.

Asundexian: A Potential New Treatment Option in Secondary Stroke Prevention

Asundexian is the first FXIa inhibitor with a successfully completed phase III trial in secondary stroke prevention.

OCEANIC STROKE

- Pragmatic study design well representative of clinical practice
- Study demographics & stroke subtypes generalizable to global stroke population
- Met primary efficacy and safety endpoints
  - Asundexian significantly reduced ischemic stroke
  - No increase in ISTH major bleeding
- **Addressing a high unmet need**

Engagement with Health Authorities has been initiated

Potential to become new treatment option in SSP

Blockbuster potential

OCEANIC-STROKE Study Results

Ashkan Shoamanesh
Associate Professor of Medicine (Neurology), Marta and Owen Boris Chair in Stroke Research and Care at McMaster University

McMaster University

Population Health Research Institute
HEALTH THROUGH KNOWLEDGE

International Stroke Conference

FACTOR XIa INHIBITION WITH ASUNDEXIAN IN ACUTE NON-CARDIOEMBOLIC STROKE OR HIGH-RISK TRANSIENT ISCHEMIC ATTACK: PRIMARY RESULTS OF THE OCEANIC-STROKE TRIAL

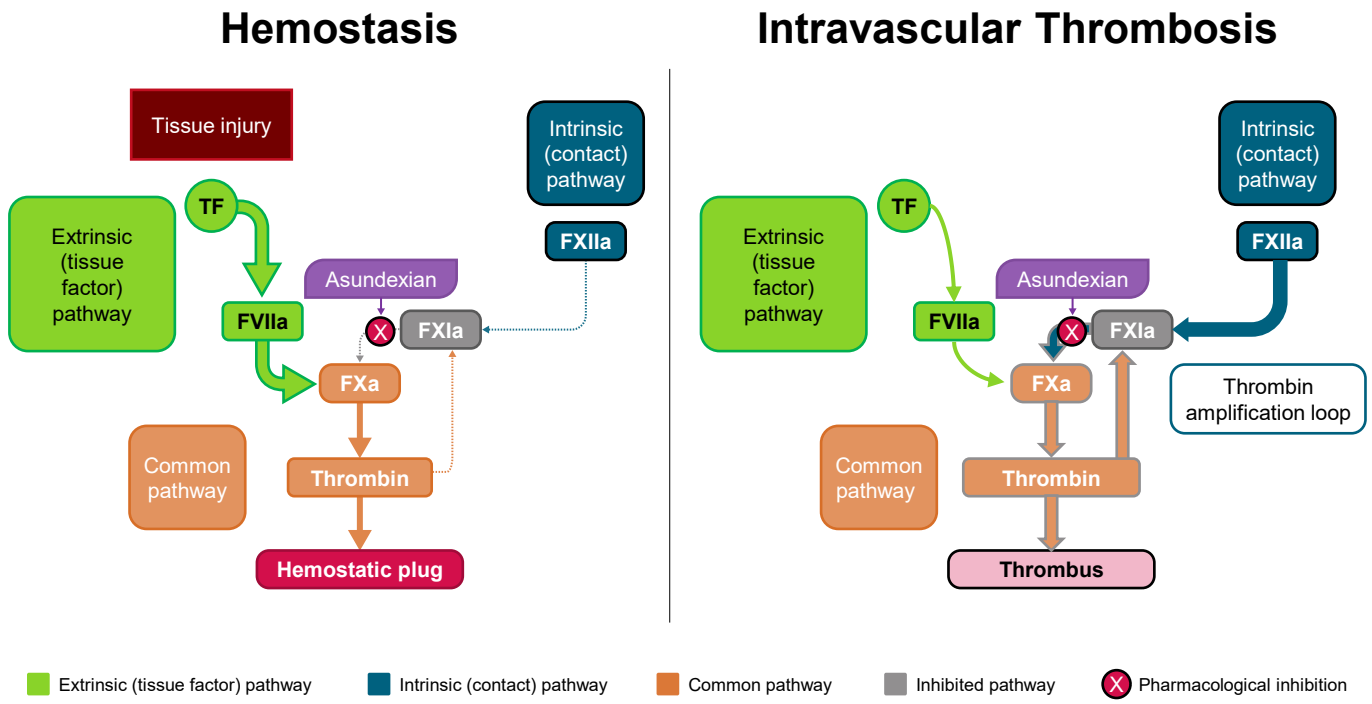
M. Sharma, Q. Dong, T. Hirano, S. Kasner, J. Saver, J. Masjuan, A. Demchuk, C. Cordonnier, D. Bereczki, G. Tsivgoulis, R. Veltkamp, I. Staikov, H-J. Bae, B. Campbell, A. Zini, I-H. Lee, S. Ameriso, M. Kovar, R. Mikulik, R. Lemmens, J. Ferro, T. Robinson, H. Christensen, S. Ozturk, R. Leker, P. Turcani, A. Slowik, P. Amaya, F.K. Hoo, G.M. De Marchis, M. Knoflach, P.N. Sylaja, J. Putaala, J.M. Coutinho, H.B. van der Worp, E. Miglane, V. Matijosaitis, A.G. Lindgren, G. Sampaio Silva, E. Sandset, S. Turuspekova, P. Amarenco, K. Sheth, E.E. Smith, J. Eikelboom, R. Joundi, K. Schulze, L. Xu, L. Heenan, C. Neumann, J. Gilbride, E. Muehlhofer, P. Colorado, L. Keller, H. Mundl, A. Shoamanesh for the OCEANIC-STROKE Steering Committee and Investigators

American Stroke Association.
A division of the American Heart Association.

#ISC26

BACKGROUND

- Genetic FXI deficiency associated with:
  - reduced risk of ischemic stroke
  - without increased risk of ICH[1–3]
- FXI has a minor role in hemostasis but may increase pathologic thrombosis
- Potential to uncouple hemostasis from thrombosis makes FXIa an attractive therapeutic target



Dashed arrows indicate minimal involvement of FXIa in hemostasis.
Figure adapted from Sharma M, et al. *European Stroke Journal.* 2026;11(1):aakaf017.

FVIIa, activated Factor VII; FXa, activated Factor X; FXI, Factor XI; FXIa, activated Factor XI; FXIIa, activated Factor XII; ICH, intracerebral hemorrhage; TF, tissue factor.

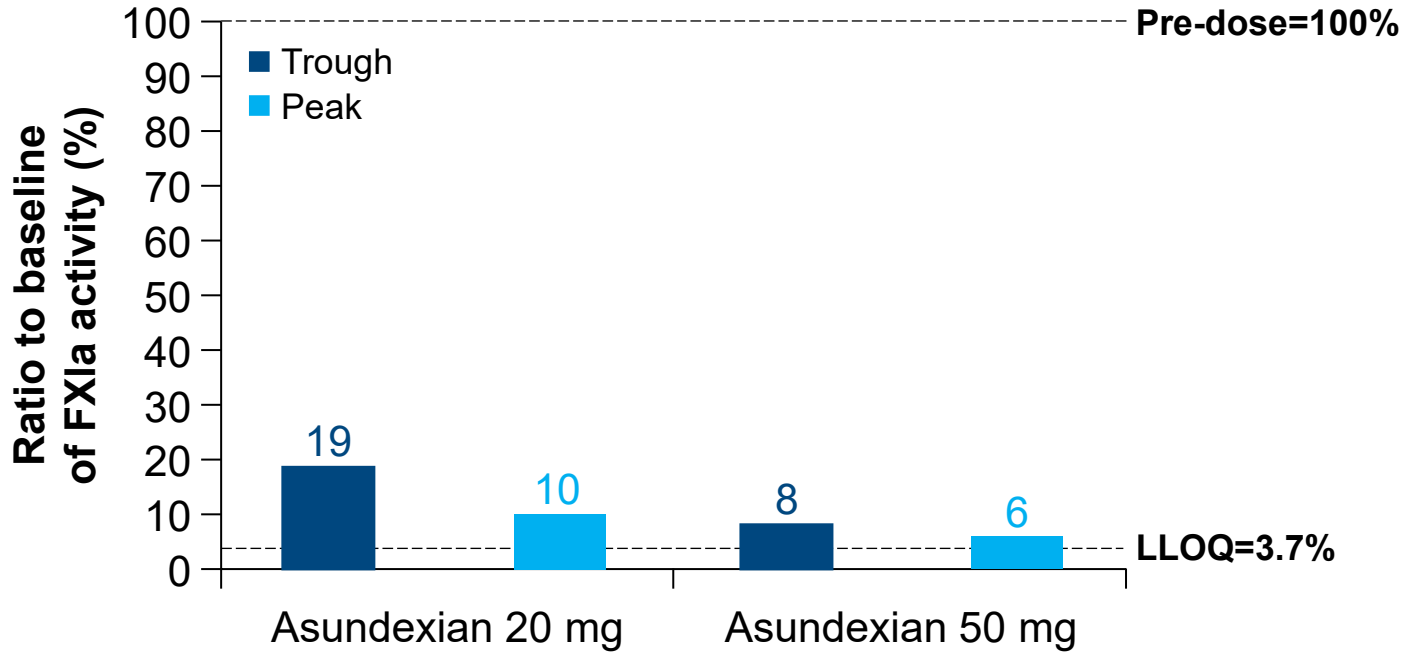
1. Gill D, et al. *Stroke*. 2018;49(11):2761–3; 2. Salomon O, et al. *Blood.* 2008;111(8):4113–7; 3. Georgi B, et al. *Stroke.* 2019;50(11):3004–12.

ASUNDEXIAN

- Direct oral inhibitor of FXIa[1,2]
  - Once daily dosing
- No effect on bleeding time – alone or with DAPT
- Phase 2 studies >4000 participants showed[3–6]
  - >90% inhibition of FXIa at peak and trough
  - No significant increase in major bleeding over placebo with or without antiplatelets

Safety of the oral factor XIa inhibitor asundexian compared with apixaban in patients with atrial fibrillation (PACIFIC-AF): a multicentre, randomised, double-blind, double-dummy, dose-finding phase 2 study

Jonathan P Piccini, Valeria Caso, Stuart J Connolly, Keith A A Fox, Jonas Oldgren, W Schuyler Jones, Diana A Gorog, Václav Durdil, Thomas Viethen, Christoph Neumann, Hardi Mundl, Manesh R Patel, on behalf of the PACIFIC-AF Investigators*



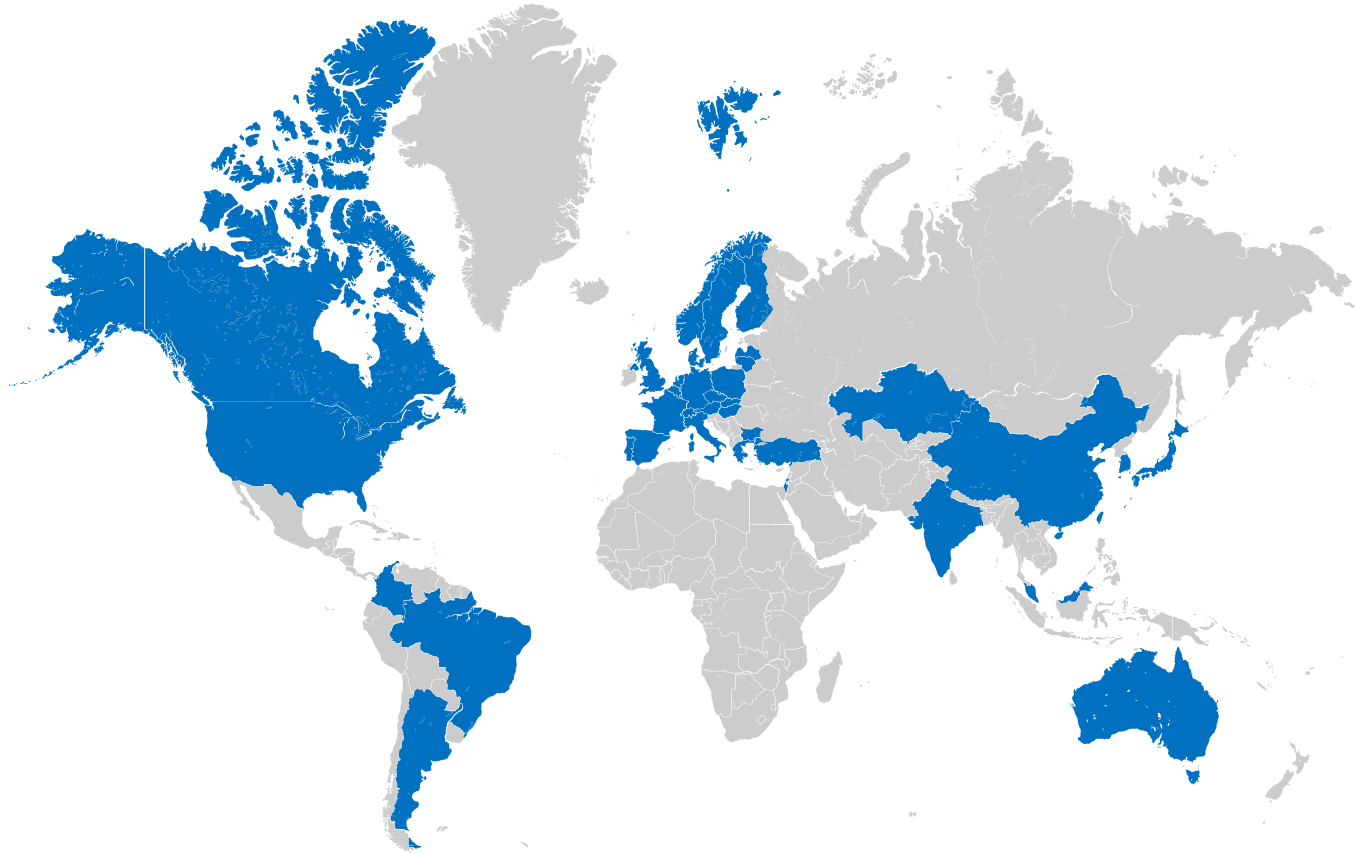
DAPT, dual antiplatelet therapy; FXIa, activated Factor XI.

1. Heitmeier S, et al. *J Thromb Haemost.* 2022;20(6):1400–11; 2. Piel I, et al. *Eur J Drug Metab Pharmacokinet.* 2023;48(4):411–25;
3. Rao SV, et al. *Circulation.* 2022;146(16):1196–206; 4. Piccini JP, et al. *Lancet.* 2022;399(10333):1383–90;
5. Shoamanesh A, et al. *Lancet.* 2022;400(10357):997–1007; 6. Eikelboom JW, et al. *J Am Coll Cardiol.* 2024;83(6):669–78.

OCEANIC-STROKE

Design

- OCEANIC-STROKE
  - Placebo-controlled
  - Double-blinded
  - Event-driven Phase 3 RCT
- Comparing asundexian 50 mg once daily and placebo
- Patients with non-cardioembolic stroke or high-risk TIA
  - Planned for antiplatelet therapy – single or aspirin + P2Y12 inhibitor (clopidogrel, ticagrelor, prasugrel)



37 Countries/Regions, 702 Sites

P2Y12, purinergic receptor Y12; RCT, randomized controlled trial; TIA, transient ischemic attack.

OCEANIC-STROKE: KEY ENDPOINTS

Endpoints (time to first occurrence)	
Primary efficacy*	**Primary safety**
Ischemic stroke	ISTH major bleeding
Secondary efficacy*	**Secondary safety**
• All strokes (ischemic and hemorrhagic) • Composite of CV death, MI or stroke • Composite of all-cause mortality, MI or stroke • Ischemic stroke in the first 90 days • Disabling stroke (mRS ≥3 at 90 days)	• Composite of ISTH major or CRNM bleeding • ISTH CRNM bleeding • Symptomatic intracranial hemorrhage • Hemorrhagic stroke • Fatal bleeding • Minor bleeding

*Hypothesis testing conducted using strict hierarchy order for efficacy endpoints.
CRNM, clinically relevant non-major; CV, cardiovascular; ISTH, International Society on Thrombosis and Haemostasis; MI, myocardial infarction; mRS, modified Rankin score.

KEY INCLUSION AND EXCLUSION CRITERIA

Key inclusion:

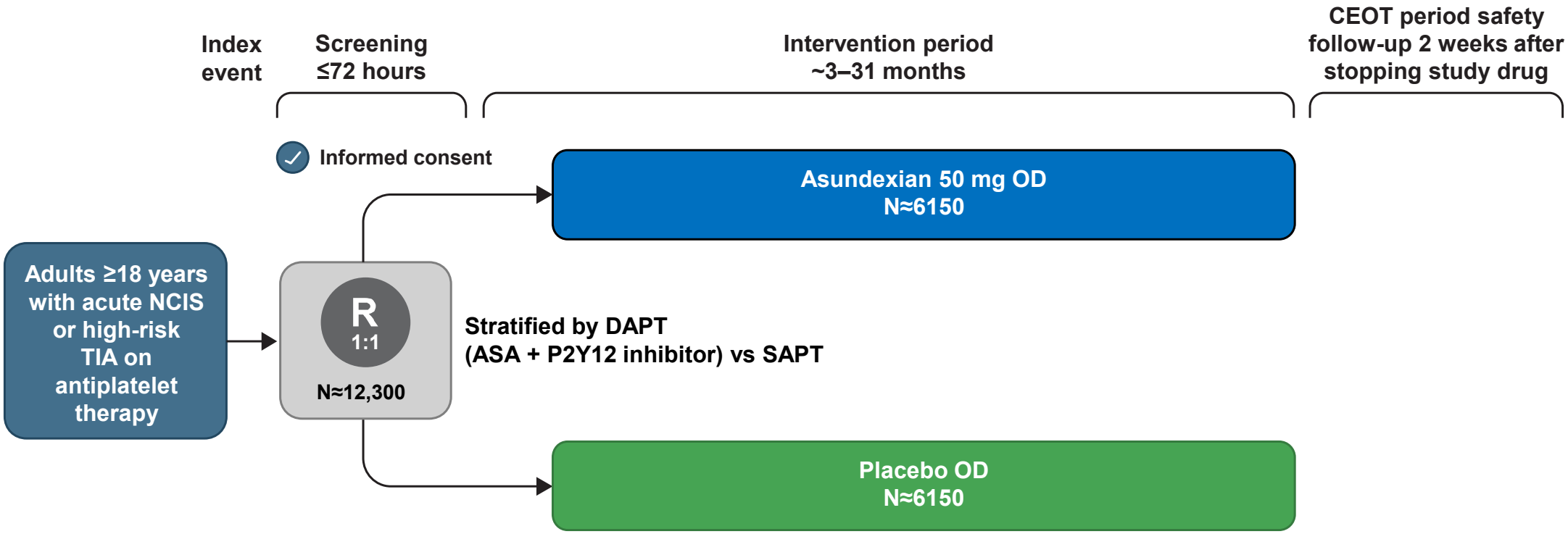
- Participants aged ≥18 years, within 72 hours of symptom onset:
  - Non-cardioembolic ischemic stroke (NIHSS ≤15) **or** high-risk TIA ($ABCD^2$ 6 or 7)
  - History of atherosclerosis **or** evidence of plaque on imaging **or** non-lacunar stroke on imaging
  - Plan for antiplatelet therapy, single or dual

Key exclusion:

- History of AF or other cardioembolic source requiring anticoagulation
- Ischemic stroke within 7 days of index event
- Strokes following procedures (TAVI, CABG) or other specific cause (e.g. vasculitis)
- End-stage renal disease requiring dialysis
- Active non-trivial bleeding (e.g. PH1 or PH2); asymptomatic HT and CMB permitted
- History of non-traumatic ICH; significant GI bleeding within 6 months

$ABCD^2$, age, blood pressure, clinical features, duration of symptoms, and diabetes; AF, atrial fibrillation; CABG, coronary artery bypass grafting; CMB, cerebral microbleed; GI, gastrointestinal; HT, hemorrhagic transformation; ICH, intracerebral hemorrhage; NIHSS, National Institutes of Health Stroke Scale; TAVI, transcatheter aortic valve implantation; TIA, transient ischemic attack.

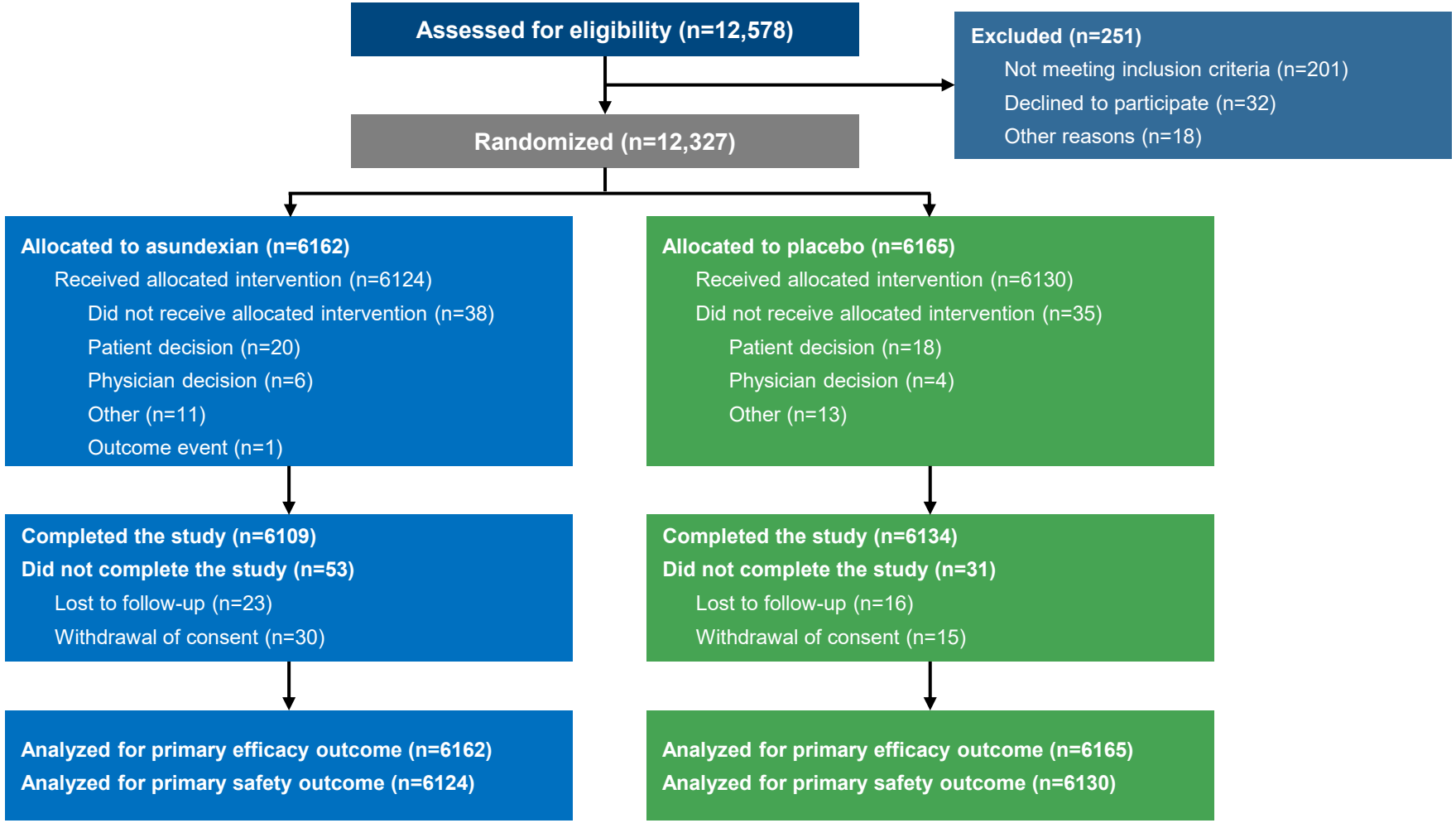
OCEANIC-STROKE: STUDY DESIGN



ASA, aspirin; CEOT, common end of treatment; DAPT, dual antiplatelet therapy; NCIS, non-cardioembolic ischemic stroke; OD, once daily; P2Y12, purinergic receptor Y12; R, randomization; SAPT, single antiplatelet therapy; TIA, transient ischemic attack.

Sharma M, et al. *Eur Stroke J.* 2026;11(1):aakaf017.

CONSORT DIAGRAM



BASELINE CHARACTERISTICS

Characteristics	Asundexian 50 mg	Placebo
Randomized, N	6162	6165
Age, years, mean (SD)	67.7 (10.8)	67.5 (10.9)
Female sex, n (%)	2063 (33.5)	2047 (33.2)
Medical history, n (%)		
Previous history of stroke or TIA	1310 (21.3)	1345 (21.8)
Coronary artery disease	949 (15.4)	1013 (16.4)
Hypertension	4937 (80.1)	4868 (79.0)
Diabetes mellitus	2134 (34.6)	2115 (34.3)
Current smoker	1644 (26.7)	1665 (27.0)
Race, n (%)		
White	4105 (66.6)	4078 (66.1)
Asian	1721 (27.9)	1742 (28.3)
Black	143 (2.3)	139 (2.3)
Other	193 (3.1)	206 (3.3)

SD, standard deviation; TIA, transient ischemic attack.

INDEX EVENT CHARACTERISTICS

Characteristics	Asundexian 50 mg	Placebo
Index event, n (%)		
Ischemic stroke	5839 (94.8)	5838 (94.7)
High-risk TIA	323 (5.2)	325 (5.3)
TOAST subtype of index event,† n (%)		
Large-artery atherosclerosis	2512 (43.0)	2484 (42.5)
Stroke of undetermined etiology	1786 (30.6)	1710 (29.3)
Small-vessel occlusion	1290 (22.1)	1349 (23.1)
Stroke of other etiology	161 (2.8)	188 (3.2)
Cardioembolic	89 (1.5)	107 (1.8)
NIHSS at randomization,† median (IQR)	2 (1, 4)	2 (1, 4)
NIHSS at randomization,† n (%)		
≤3	4087 (70.0)	4079 (69.9)
4–7	1385 (23.7)	1375 (23.6)
≥8	365 (6.3)	382 (6.5)
Dual antiplatelet therapy	3859 (62.6)	3853 (62.5)

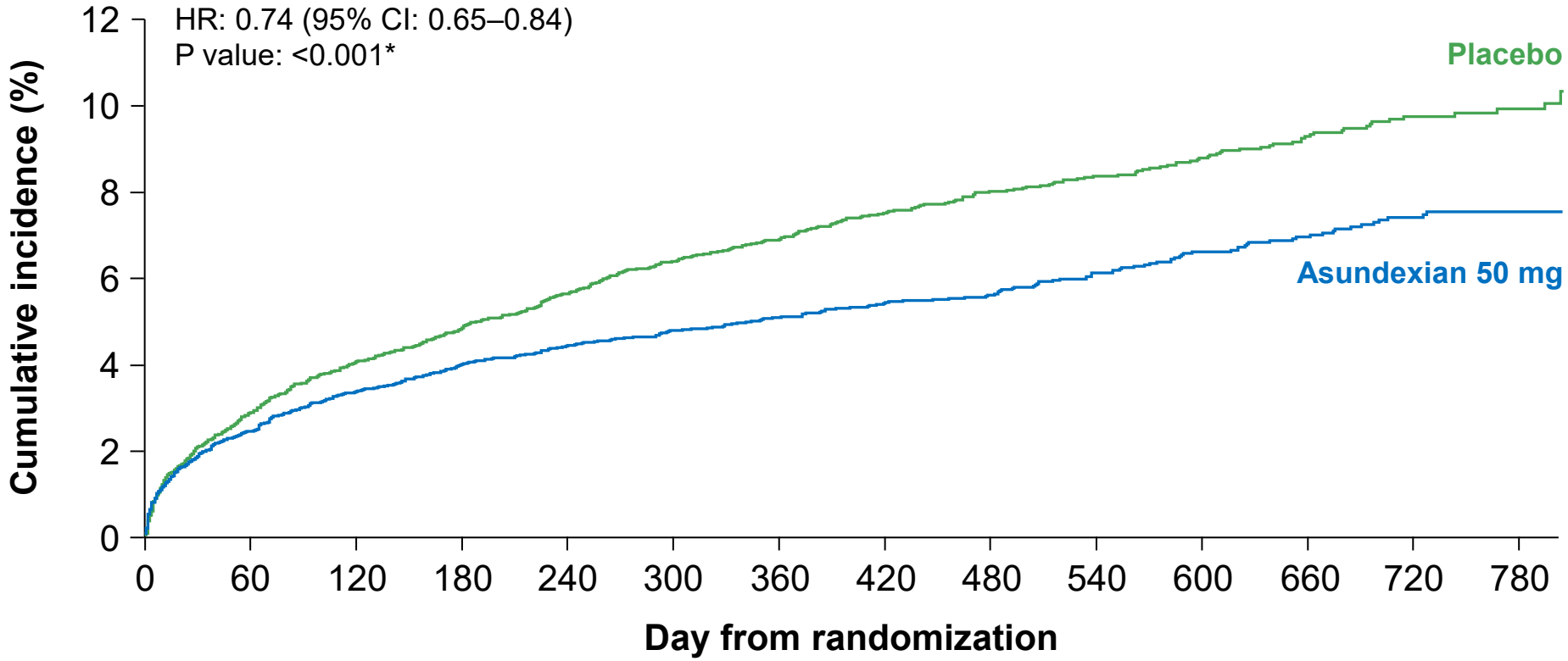
†Stroke index event only.
IQR, interquartile range; NIHSS, National Institutes of Health Stroke Scale; TIA, transient ischemic attack; TOAST, Trial of Org 10172 in Acute Stroke Treatment.

ACUTE TREATMENT OF INDEX STROKE

	Overall N=11677	Asundexian 50 mg N=5839	Placebo N=5838
Intravenous thrombolysis and/or endovascular therapy,† n (%)	3201 (27.4)	1608 (27.5)	1593 (27.3)
Intravenous thrombolysis only	2314 (19.8)	1146 (19.6)	1168 (20.0)
Endovascular therapy only	371 (3.2)	202 (3.5)	169 (2.9)
Intravenous thrombolysis and endovascular therapy	516 (4.4)	260 (4.5)	256 (4.4)

†Stroke index event only.

CUMULATIVE INCIDENCE OF ISCHEMIC STROKE



No. at risk

Placebo	6165	5949	5853	5754	5370	4840	4406	3990	3497	3070	2564	1961	1410	792
Asundexian 50 mg	6162	5958	5859	5763	5384	4876	4463	4033	3543	3101	2588	2004	1428	810

*P value is obtained from stratified log-rank test (stratified by baseline intention of DAPT). csHR and 95% CI are provided here.
Absolute risk reduction at 1 year was 1.9%, with a number needed to treat of 53.
Cumulative incidence curves are estimated by Aalen–Johansen method, truncated at Day 820.
CI, confidence interval; csHR, cause-specific hazard ratio; DAPT, dual antiplatelet therapy; HR, hazard ratio.

EFFICACY OUTCOMES

Outcome	Asundexian 50 mg (N=6162) n (%)	Placebo (N=6165) n (%)	csHR (95% CI)†	P value‡
Primary efficacy event				
Ischemic stroke	384 (6.2)	518 (8.4)	0.74 (0.65–0.84)	<0.001
Secondary efficacy events				
All strokes (ischemic, hemorrhagic)	404 (6.6)	545 (8.8)	0.74 (0.65–0.84)	<0.001
CV death, MI or stroke	568 (9.2)	685 (11.1)	0.83 (0.74–0.92)	<0.001
All-cause mortality, MI, or stroke	649 (10.5)	757 (12.3)	0.85 (0.77–0.95)	0.003
Ischemic stroke in the first 90 days	183 (3.0)	218 (3.5)	0.84 (0.69–1.02)	0.08
Disabling/fatal stroke¶	128 (2.1)	185 (3.0)	0.69 (0.55–0.87)	Not applicable

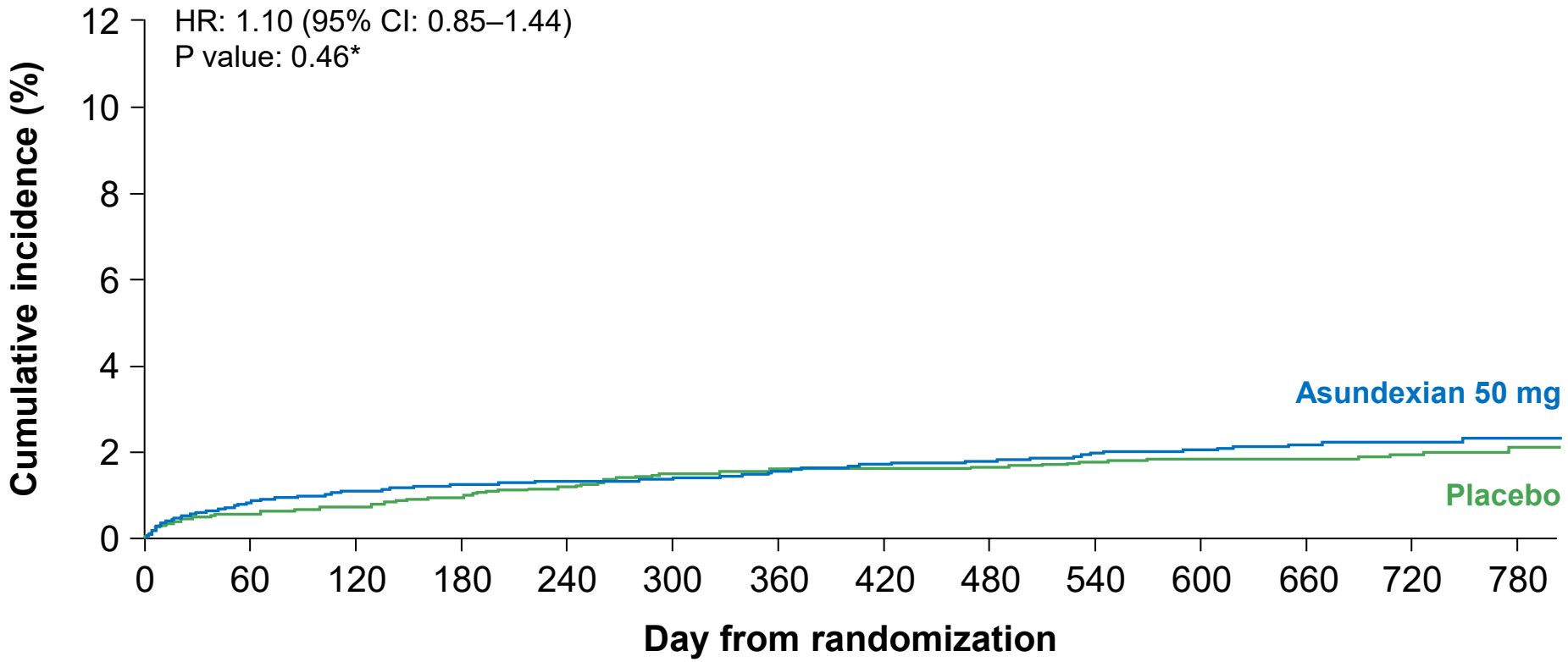
†csHRs are estimated from stratified cox proportional hazard model (stratified by baseline intention of T); ‡P values are obtained from stratified log-rank test (stratified by baseline intention of T); ¶A disabling stroke is defined as a stroke of any type during the trial associated with a modified Rankin Scale (mRS) of ≥3 at 90 days after the stroke or an increase of 1 point if the last available mRS before the recurrent stroke event was ≥3.
csHR, cause-specific hazard ratio; CI, confidence interval; CV, cardiovascular; MI, myocardial infarction; mRS, modified Rankin score.

SAFETY OUTCOMES

Outcome	Asundexian 50 mg (N=6162) n (%)	Placebo (N=6165) n (%)	csHR (95% CI)†
Primary safety event			
ISTH major bleeding	117 (1.9)	107 (1.7)	1.10 (0.85–1.44)
Secondary safety events			
ISTH major or clinically relevant non-major bleed	339 (5.5)	307 (5.0)	1.12 (0.96–1.30)
Clinically relevant non-major bleeding	231 (3.8)	210 (3.4)	1.11 (0.92–1.34)
Symptomatic intracranial hemorrhage (includes intracerebral hemorrhage)	41 (0.7)	36 (0.6)	1.15 (0.74–1.80)
Hemorrhagic stroke	13 (0.2)	20 (0.3)	0.66 (0.33–1.32)
Fatal bleeding	14 (0.2)	8 (0.1)	1.77 (0.74–4.23)
Minor bleeding	479 (7.8)	512 (8.4)	0.94 (0.83–1.07)

†csHRs are estimated from stratified Cox proportional hazards model (stratified by baseline intention of DAPT).
CI, confidence interval; csHR, cause-specific hazard ratio; DAPT, dual antiplatelet therapy; ISTH, International Society on Thrombosis and Haemostasis.

CUMULATIVE INCIDENCE OF ISTH MAJOR BLEEDING

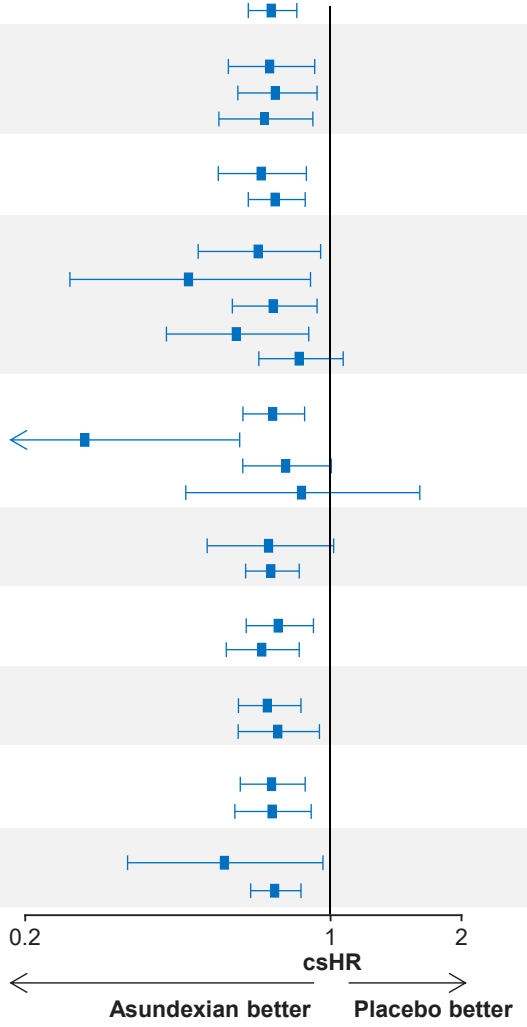


No. at risk														
Placebo	6130	5391	5021	4833	4415	3944	3572	3165	2775	2441	2026	1549	1121	618
Asundexian 50 mg	6124	5354	4968	4807	4366	3900	3547	3104	2699	2374	1943	1508	1082	613

*P value is obtained from stratified log-rank test (stratified by baseline intention of DAPT). csHR and 95% CI are provided here.
Cumulative incidence curves are estimated by Aalen–Johansen method, truncated at Day 820.
CI, confidence interval; csHR, cause-specific hazard ratio; DAPT, dual antiplatelet therapy; HR, hazard ratio.

SUBGROUP ANALYSES FOR ISCHEMIC STROKE

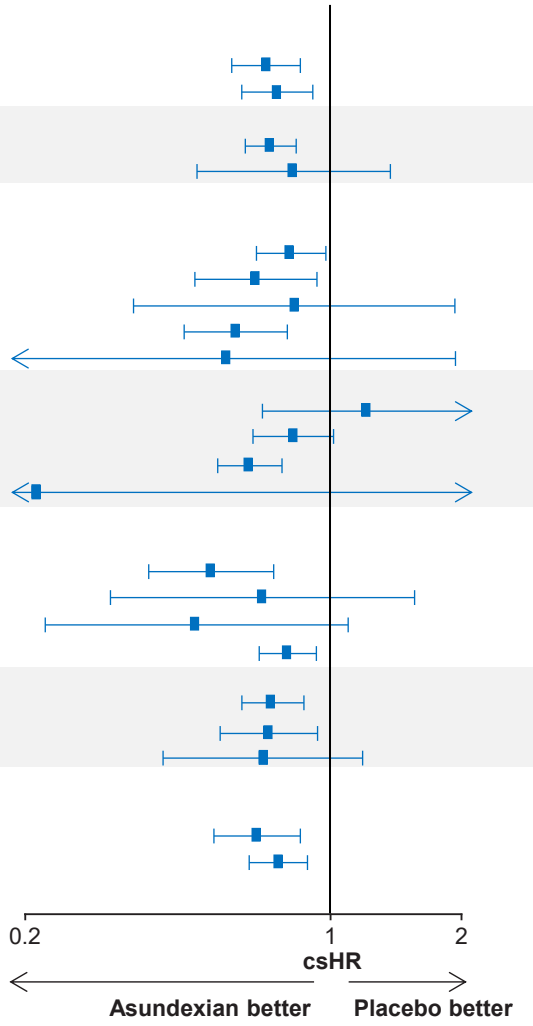
	Asundexian 50 mg n/N (%)	Placebo n/N (%)	csHR (95% CI)
Overall	**384/6162 (6.2%)**	**518/6165 (8.4%)**	0.74 (0.65–0.84)
Age			
<65 yr	128/2313 (5.5%)	176/2308 (7.6%)	0.73 (0.58–0.92)
65–75 yr	152/2318 (6.6%)	197/2318 (8.5%)	0.76 (0.61–0.94)
>75 yr	104/1531 (6.8%)	145/1539 (9.4%)	0.72 (0.56–0.92)
Sex			
Female	114/2063 (5.5%)	159/2047 (7.8%)	0.70 (0.55–0.89)
Male	270/4099 (6.6%)	359/4118 (8.7%)	0.75 (0.64–0.88)
Region			
North America	60/783 (7.7%)	86/782 (11.0%)	0.69 (0.50–0.96)
South America	14/281 (5.0%)	29/286 (10.1%)	0.48 (0.25–0.91)
Western Europe, Australia, and Israel	132/2425 (5.4%)	175/2419 (7.2%)	0.75 (0.60–0.94)
Eastern Europe	42/913 (4.6%)	68/910 (7.5%)	0.62 (0.42–0.91)
Asia-Pacific	136/1760 (7.7%)	160/1768 (9.0%)	0.86 (0.68–1.08)
Race			
White	235/4105 (5.7%)	314/4078 (7.7%)	0.74 (0.63–0.88)
Black	8/143 (5.6%)	25/139 (18.0%)	0.28 (0.13–0.62)
Asian	123/1721 (7.1%)	156/1742 (9.0%)	0.79 (0.63–1.01)
Other	18/193 (9.3%)	23/206 (11.2%)	0.86 (0.47–1.60)
Hypertension prior to randomization			
No	58/1225 (4.7%)	84/1297 (6.5%)	0.73 (0.52–1.02)
Yes	326/4937 (6.6%)	434/4868 (8.9%)	0.74 (0.64–0.85)
Diabetes prior to randomization			
No	214/4028 (5.3%)	281/4050 (6.9%)	0.77 (0.64–0.92)
Yes	170/2134 (8.0%)	237/2115 (11.2%)	0.70 (0.58–0.86)
History of stroke or TIA prior to index event			
No	238/4852 (4.9%)	325/4820 (6.7%)	0.73 (0.61–0.86)
Yes	146/1310 (11.1%)	193/1345 (14.3%)	0.77 (0.62–0.95)
Medical history of atherosclerosis			
No	224/4172 (5.4%)	300/4165 (7.2%)	0.74 (0.62–0.88)
Yes	160/1990 (8.0%)	218/2000 (10.9%)	0.74 (0.60–0.91)
Vascular imaging evidence of atherosclerosis			
No	23/528 (4.4%)	40/550 (7.3%)	0.58 (0.35–0.97)
Yes	361/5634 (6.4%)	478/5615 (8.5%)	0.75 (0.66–0.86)



CIs are unadjusted for multiplicity and may not be used for inference.
CI, confidence interval; csHR, cause-specific hazard ratio; TIA, transient ischemic attack.

SUBGROUP ANALYSES FOR ISCHEMIC STROKE

	Asundexian 50 mg n/N (%)	Placebo n/N (%)	csHR (95% CI)
Brain imaging demonstrating an acute non-lacunar infarct			
No	193/3173 (6.1%)	266/3158 (8.4%)	0.72 (0.60–0.86)
Yes	191/2989 (6.4%)	252/3007 (8.4%)	0.76 (0.63–0.92)
Type of index event			
Ischemic stroke	358/5839 (6.1%)	485/5838 (8.3%)	0.73 (0.64–0.84)
TIA	26/323 (8.0%)	33/325 (10.2%)	0.83 (0.49–1.38)
Stroke subtype (TOAST) classification*			
Large-artery atherosclerosis	203/2512 (8.1%)	247/2484 (9.9%)	0.82 (0.68–0.98)
Small-vessel occlusion (lacune)	59/1290 (4.6%)	90/1349 (6.7%)	0.68 (0.49–0.94)
Stroke of other determined etiology	9/161 (5.6%)	13/188 (6.9%)	0.84 (0.36–1.96)
Stroke of undetermined etiology	83/1786 (4.6%)	127/1710 (7.4%)	0.61 (0.46–0.81)
Cardioembolism	4/89 (4.5%)	8/107 (7.5%)	0.58 (0.17–1.95)
Time from index event to randomization			
≤24 hours	27/335 (8.1%)	24/356 (6.7%)	1.22 (0.70–2.11)
>24–48 hours	152/2190 (6.9%)	181/2150 (8.4%)	0.83 (0.67–1.03)
>48–72 hours	204/3592 (5.7%)	310/3630 (8.5%)	0.66 (0.55–0.78)
>72 hours	1/45 (2.2%)	3/29 (10.3%)	0.22 (0.02–2.08)
Intravenous thrombolysis and/or endovascular therapy for index stroke*			
Intravenous thrombolysis only	54/1146 (4.7%)	99/1168 (8.5%)	0.54 (0.39–0.75)
Endovascular therapy only	11/202 (5.4%)	13/169 (7.7%)	0.71 (0.32–1.57)
Intravenous thrombolysis and endovascular therapy	9/260 (3.5%)	18/256 (7.0%)	0.50 (0.22–1.11)
Neither intravenous thrombolysis nor endovascular therapy	284/4231 (6.7%)	355/4245 (8.4%)	0.80 (0.69–0.94)
NIHSS score of index stroke at randomization*			
≤3	240/4087 (5.9%)	321/4079 (7.9%)	0.74 (0.63–0.87)
4–7	95/1385 (6.9%)	130/1375 (9.5%)	0.73 (0.56–0.95)
≥8	23/365 (6.3%)	33/382 (8.6%)	0.71 (0.41–1.20)
Planned dual antiplatelet therapy with aspirin and a P2Y12 inhibitor			
No	119/2303 (5.2%)	174/2312 (7.5%)	0.68 (0.54–0.86)
Yes	265/3859 (6.9%)	344/3853 (8.9%)	0.77 (0.65–0.90)



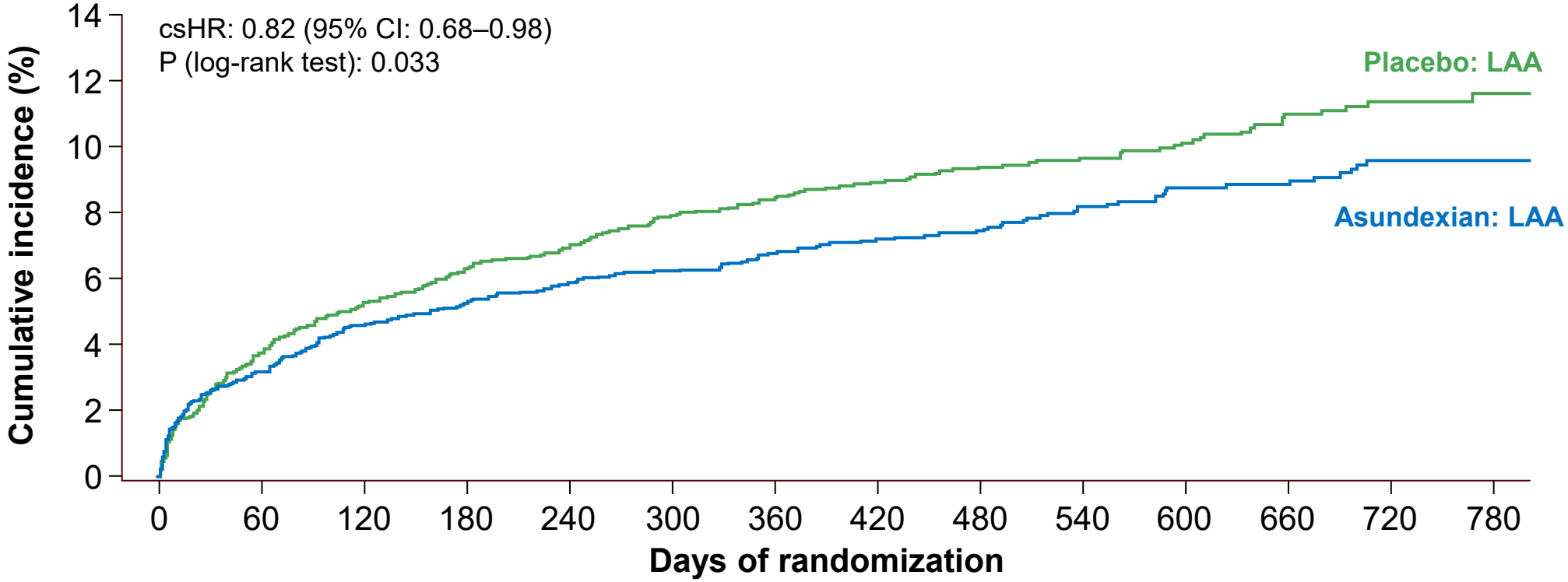
*For index event of ischemic stroke.
CIs are unadjusted for multiplicity and may not be used for inference.
CI, confidence interval; csHR, cause-specific hazard ratio; NIHSS, National Institutes of Health Stroke Scale; P2Y12, purinergic receptor Y12; TIA, transient ischemic attack; TOAST, Trial of Org 10172 in Acute Stroke Treatment.

NO TREATMENT INTERACTION FOR EFFICACY OUTCOMES ACROSS ISCHEMIC STROKE SUBTYPES

Efficacy endpoints	Large-artery atherosclerosis			Small-vessel occlusion			Stroke of undetermined etiology			
	Asundexian n n (%) (N=2512)	Placebo n (%) (N=2484)	Asundexian vs Placebo HR (95% CI)†	Asundexian n n (%) (N=1290)	Placebo n (%) (N=1349)	Asundexian vs Placebo HR (95% CI)†	Asundexian n n (%) (N=1786)	Placebo n (%) (N=1710)	Asundexian vs Placebo HR (95% CI)†	Inter. P-value
Primary efficacy endpoint (ischemic stroke)§	203 (8.08)	247 (9.94)	0.82 (0.68–0.98)	59 (4.57)	90 (6.67)	0.68 (0.49–0.94)	83 (4.65)	127 (7.43)	0.61 (0.46–0.81)	0.21
All strokes (ischemic and hemorrhagic)	211 (8.40)	257 (10.3)	0.82 (0.68–0.98)	65 (5.04)	99 (7.34)	0.68 (0.50–0.93)	87 (4.87)	132 (7.72)	0.62 (0.47–0.81)	0.21
Disabling or a fatal stroke*	72 (2.87)	86 (3.46)	0.83 (0.61–1.14)	19 (1.47)	25 (1.85)	0.80 (0.44–1.46)	26 (1.46)	51 (2.98)	0.48 (0.30–0.77)	0.15

†Cause-specific hazard ratios are estimated from stratified cox proportional hazards model (stratified by baseline intention of T). §Ischemic stroke here includes undetermined stroke. *Stroke of any type during the trial associated with a mRS of ≥3 at 90 days after the recurrent stroke or an increase of 1 point if the last available mRS before the recurrent stroke event was ≥3.
CI, confidence interval; HR, hazard ratio; mRS, modified Rankin Scale.

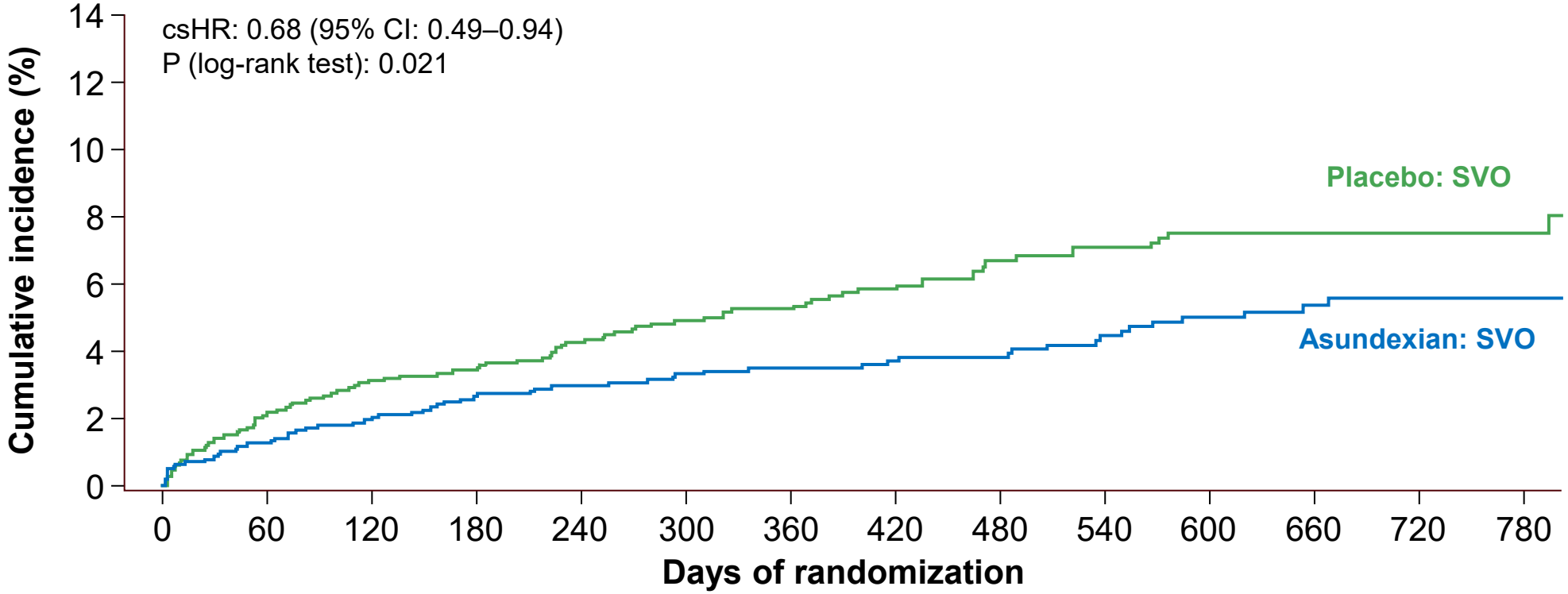
CUMULATIVE INCIDENCE OF THE PRIMARY EFFICACY ENDPOINT OF ISCHEMIC STROKE IN THE LARGE-ARTERY ATHEROSCLEROSIS SUBGROUP



No. at Risk														
Placebo: LAA	2484	2373	2327	2279	2152	1942	1778	1621	1413	1243	1035	797	574	313
Asundexian: LAA	2512	2406	2349	2310	2161	1954	1783	1598	1402	1215	1016	796	584	329

Cumulative incidence curves (Aalen-Johansen) are truncated at Day 820.
CI, confidence interval; csHR, cause-specific hazard ratio; LAA, large-artery atherosclerosis.

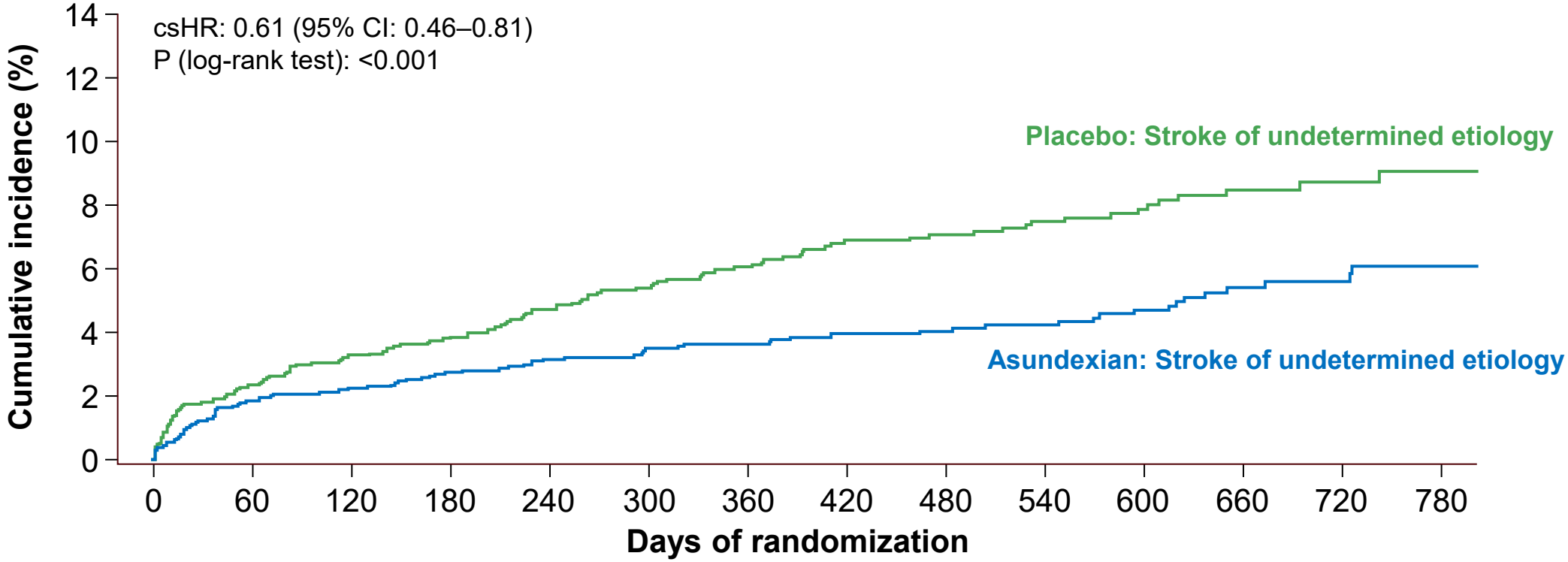
CUMULATIVE INCIDENCE OF THE PRIMARY EFFICACY ENDPOINT OF ISCHEMIC STROKE IN THE SMALL-VESSEL OCCLUSION SUBGROUP



No. at Risk													
Placebo: SVO	1349	1311	1295	1286	1208	1104	992	901	798	706	613	474	351	200
Asundexian: SVO	1290	1266	1250	1231	1167	1065	974	896	785	705	617	467	319	164

Cumulative incidence curves (Aalen-Johansen) are truncated at Day 820.
CI, confidence interval; csHR, cause-specific hazard ratio, SVO: small-vessel occlusion

CUMULATIVE INCIDENCE OF THE PRIMARY EFFICACY ENDPOINT OF ISCHEMIC STROKE IN THE STROKE OF UNDETERMINED ETIOLOGY SUBGROUP



No. at Risk														
Placebo: SUE	1710	1661	1638	1611	1472	1297	1169	1038	907	789	636	476	332	190
Asundexian: SUE	1786	1737	1719	1690	1562	1407	1290	1156	1018	894	714	547	385	225

Cumulative incidence curves (Aalen-Johansen) are truncated at Day 820.
CI, confidence interval; csHR, cause-specific hazard ratio, SUE, stroke of undetermined etiology

NO TREATMENT INTERACTION FOR SAFETY OUTCOMES ACROSS ISCHEMIC STROKE SUBTYPES

Safety endpoints	Large-artery atherosclerosis			Small-vessel occlusion			Stroke of undetermined etiology			
	Asundexian n (%) (N=2496)	Placebo n (%) (N=2468)	Asundexian vs Placebo HR (95% CI)†	Asundexian n (%) (N=1280)	Placebo n (%) (N=1342)	Asundexian vs Placebo HR (95% CI)†	Asundexian n (%) N=1777	Placebo n (%) N=1705	Asundexian vs Placebo HR (95% CI)†	Inter. P-value
Primary safety endpoint: ISTH major bleeding	53 (2.1)	44 (1.8)	1.20 (0.80–1.79)	23 (1.8)	30 (2.2)	0.82 (0.48–1.42)	29 (1.6)	25 (1.5)	1.11 (0.65–1.90)	0.52
Symptomatic intracranial hemorrhage (including intracerebral hemorrhage)	14 (0.6)	12 (0.5)	1.17 (0.54–2.54)	13 (1.0)	12 (0.9)	1.19 (0.54–2.60)	9 (0.5)	9 (0.5)	0.95 (0.38–2.40)	0.93
Hemorrhagic stroke	4 (0.2)	5 (0.2)	0.80 (0.21–2.98)	6 (0.5)	8 (0.6)	0.82 (0.28–2.37)	2 (0.1)	5 (0.3)	0.38 (0.07–1.98)	0.72

†Cause-specific hazard ratios are estimated from stratified cox proportional hazards model (stratified by baseline intention of T).
CI, confidence interval; HR, hazard ratio; ISTH, International Society on Thrombosis and Haemostasis.

CONSISTENT RESULTS IN PATIENTS WITH EMBOLIC STROKE OF UNDETERMINED SOURCE

Endpoints	ESUS			No ESUS			
	Asundexian n (%)	Placebo n (%)	Asundexian vs Placebo HR (95% CI)†	Asundexian n (%)	Placebo n (%)	Asundexian vs Placebo HR (95% CI)†	Inter. P-value
Primary efficacy endpoint (ischemic stroke)§	N=924 42 (4.5)	N=920 76 (8.3)	– 0.53 (0.37–0.78)	N=4914 316 (6.4)	N=4918 409 (8.3)	– 0.77 (0.67–0.89)	0.07
ISTH major bleeding	N=923 16 (1.7)	N=918 14 (1.5)	– 1.11 (0.54–2.27)	N=4878 90 (1.8)	N=4885 89 (1.8)	– 1.02 (0.76–1.37)	0.80

§Ischemic stroke here includes undetermined stroke.
†Cause-specific hazard ratios are estimated from stratified cox proportional hazards model (stratified by baseline intention of DAPT).
CI, confidence interval; DAPT, dual antiplatelet therapy; ESUS, embolic stroke of undetermined source; HR, hazard ratio; ISTH, International Society on Thrombosis and Haemostasis.

CONCLUSION

- OCEANIC-STROKE enrolled a large representative sample of non-cardioembolic ischemic stroke subtypes.
- Asundexian reduced ischemic and disabling stroke, without increasing ISTH major bleeding or intracranial hemorrhage across all qualifying ischemic stroke subtypes.
- These findings were consistent in patients with qualifying ESUS.
- Asundexian provides a novel efficacious and safe secondary stroke prevention treatment for patients with non-cardioembolic ischemic stroke, irrespective of underlying etiology.

ESUS, embolic stroke of undetermined source; ISTH, International Society on Thrombosis and Haemostasis.

Asundexian's Potential in Secondary Stroke Prevention

Jan F. Voss
Senior Vice President, Global Head Asundexian at Bayer

Stroke presents an overwhelming financial burden for both healthcare systems and patients

Preventing recurrent strokes could alleviate economic burden, free up healthcare resources, and allow for more stroke survivors to remain productive.

The economic burden of stroke

- The global financial impact of stroke has reached **$891 billion,** projected to increase to **$1 trillion by 2030**[1,2]
- Direct costs (treatment, rehab, social care, and informal caregiving) account for **$393 billion**[1]
- Production losses due to workforce reductions and caregiving responsibilities amount to **$58 billion**[1]

The humanistic burden

- Recurrent ischemic stroke is a strong predictor of **reduced short-term HRQoL**[3]
- The fear of stroke recurrence was commonly reported as **anxiety-provoking in patients**[4]
- Other psychological factors such **as post-stroke fatigue and depression** also significantly impact the HRQoL of stroke survivors and their caregivers[5,6,7]

HRQoL, health related quality of life. **1** Owolabi MO, et al. The Lancet Public Health. 2022 Jan 1;7(1):e74-85.; **2** World Stroke Organization. Impact of Stroke. Available at: https://www.world-stroke.org/world-stroke-day-campaign/about-stroke/impact-of-stroke. Accessed January 2026.; **3** Segerdahl M, et al. BMC Geriatr. 2023;23(1):584; **4** Chun HY, et al. Pilot and feasibility studies. 2018 Aug 14;4(1):139.; **5** Feigin VL, et al. The Lancet Neurology. 2023 Dec 1;22(12):1160-206.; **6** Ablewhite J, et al. Clinical Rehabilitation. 2022 Oct;36(10):1400-10.; **7** Byun E, et al. Topics in stroke rehabilitation. 2019 Apr 3;26(3):187-94.

Bayer's Asundexian – A Groundbreaking Moment for Secondary Stroke Prevention

The Phase III OCEANIC-STROKE study signals a **breakthrough in secondary stroke prevention**, with **asundexian significantly reducing ischemic stroke**, without increasing ISTH major bleeding, compared to placebo with antiplatelet therapy. [1]

These groundbreaking results will enable us to rewrite the future for stroke survivors and their families.

Bayer. Bayer's Asundexian Met Primary Efficacy and Safety Endpoints in Landmark Phase III OCEANIC-STROKE Study in Secondary Stroke Prevention. Bayer Newsroom. Available at: https://www.bayer. com/en/us/news-stories/oceanic-stroke [Accessed November 2025]

Q&A

Please follow the instructions provided to ask your questions.