PHARVARIS

Deucrictibant: Beyond HAE-1/2

June 4, 2025

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This presentation includes data for an investigational product not yet approved by regulatory authorities. Certain information contained in this Presentation relates to or is based on studies, publications, surveys and other data obtained from third-party sources and the Company’s own internal estimates and research. While the Company believes these third-party sources to be reliable as of the date of this presentation, it has not independently verified, and makes no representation as to the adequacy, fairness, accuracy or completeness of, any information obtained from third-party sources. In addition, all of the market data included in this Presentation involves a number of assumptions and limitations, and there can be no guarantee as to the accuracy or reliability of such assumptions. Finally, while we believe our own internal research is reliable, such research has not been verified by any independent source.

Agenda

Danny M. Cohn, M.D., Ph.D., Department of Vascular Medicine, Amsterdam UMC (an accredited center of ACARE)

Berndt Modig, CEO Pharvaris

Anne Lesage, Ph.D., CEDO Pharvaris

Peng Lu, M.D., Ph.D., CMO Pharvaris

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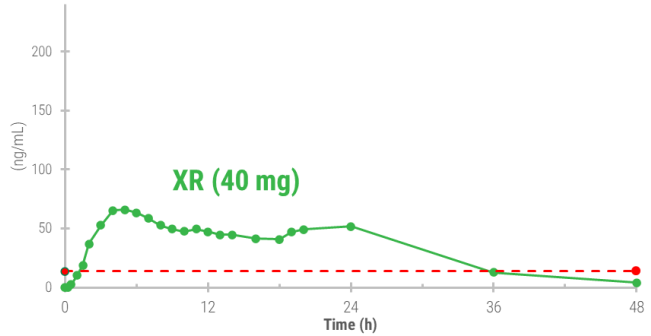
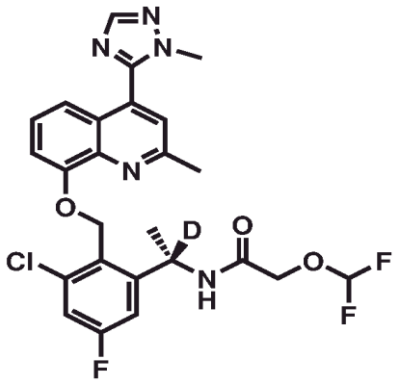
Deucrictibant has the potential to address unmet needs of people living with bradykinin-mediated angioedema

deucrictibant extended-release (XR) tablet

sustained absorption[1]



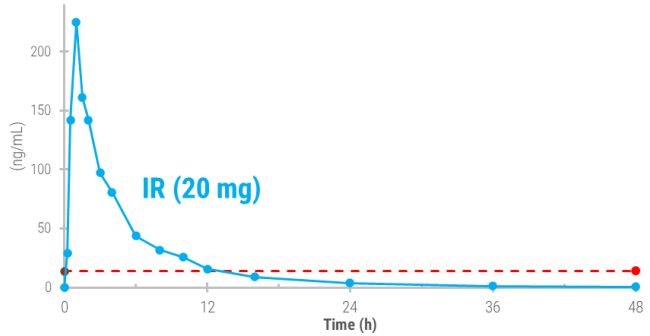
In studies, deucrictibant maintained sustained therapeutic exposure over 24 hours[2] from day one, allowing for once-daily oral prevention of AE-BK attacks[3]



deucrictibant

deucrictibant immediate-release (IR) capsule

rapid absorption[4]

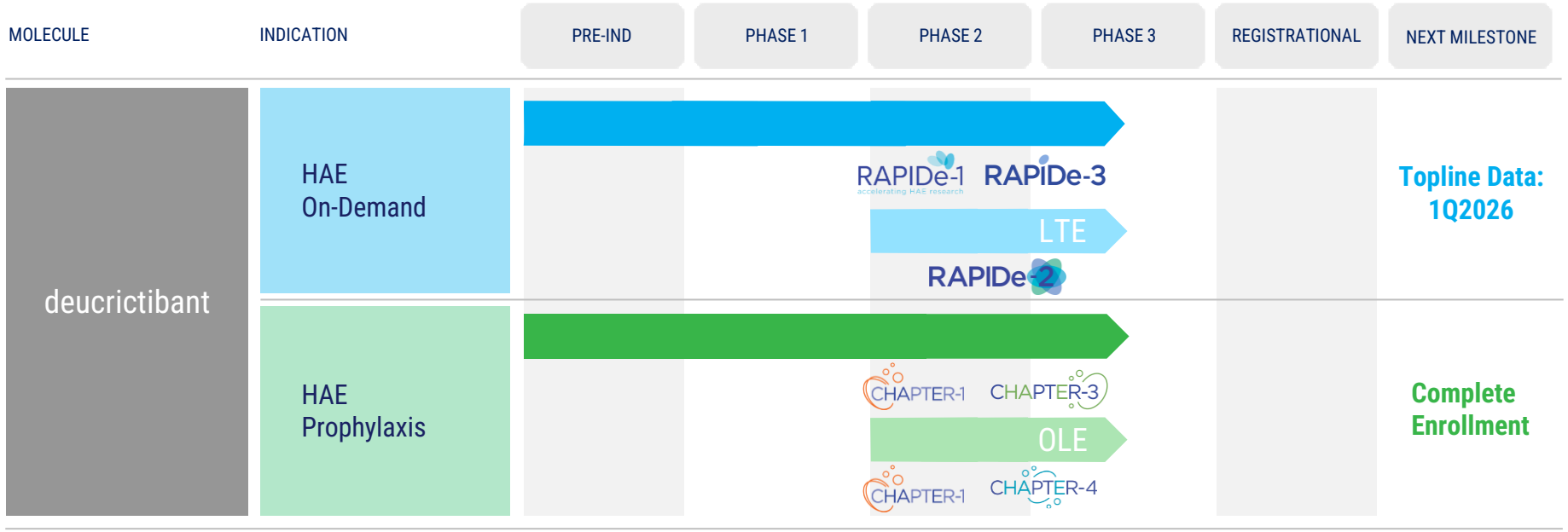


In studies, deucrictibant rapidly reached therapeutic exposure within 15-30 minutes[5], making it optimal for on-demand oral treatment of AE-BK attacks[6]

Two oral products with the same active ingredient for the prevention and treatment of bradykinin-mediated angioedema attacks

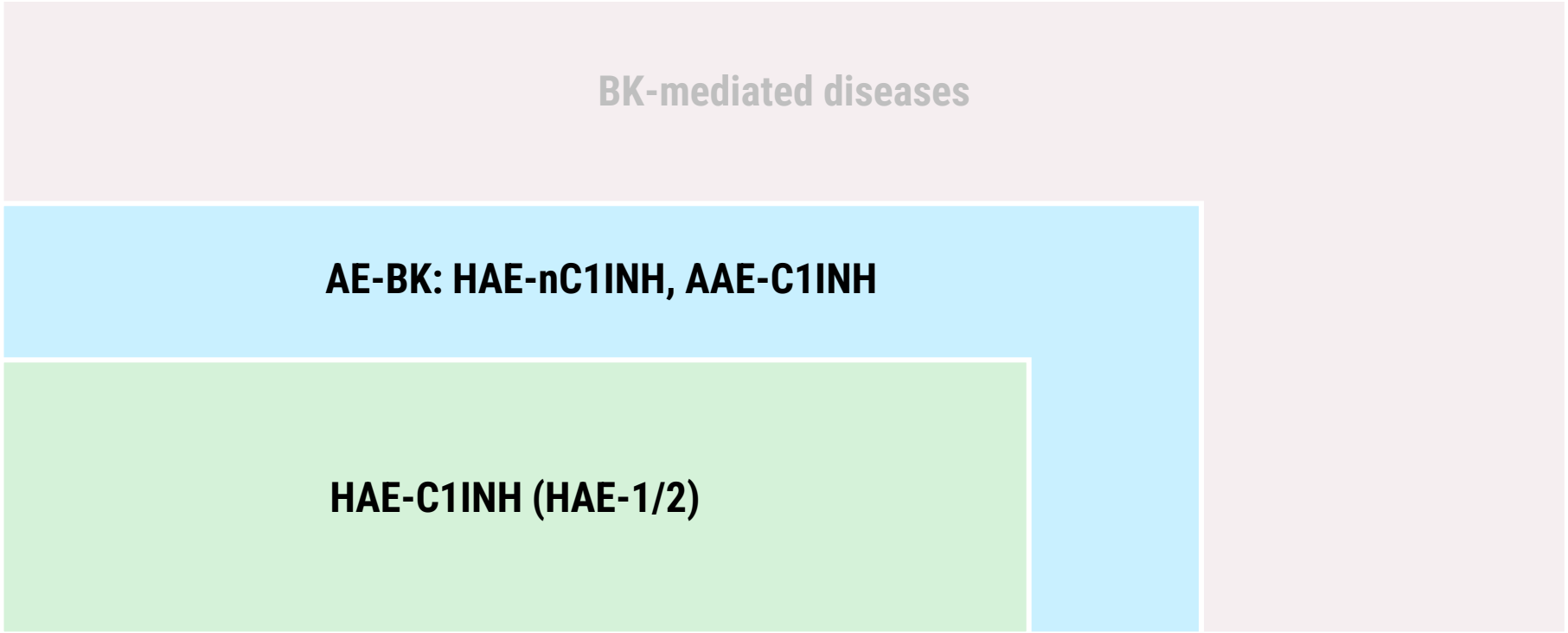
Notes: AE-BK: bradykinin-mediated angioedema. Source: [1]Zhang Z et al. C1-INH WS 2025. [2]Lesage A et al. IDDST 2024. [3]NCT06669754. [4]Crabbe R et al. AAAAI 2021. [5]Maurer M et al. AAAAI 2023. [6]NCT06343779.

Deucrictibant for the prevention and treatment of bradykinin-mediated angioedema



Notes: HAE: hereditary angioedema. LTE: long-term extension. OLE: open-label extension.
Source: RAPIDe-1 (NCT04618211). RAPIDe-2 (NCT05396105). RAPIDe-3 (NCT06343779). CHAPTER-1 (NCT05047185). CHAPTER-3 (NCT06669754). CHAPTER-4 (NCT06679881).

Pharvaris aspires to leverage its bradykinin B2 receptor expertise to develop therapies for AE-BK, beyond HAE-1/2



AAE-C1INH: acquired angioedema due to C1INH deficiency. AE-BK: bradykinin-mediated angioedema. C1INH: C1 inhibitor. HAE-1/2: hereditary angioedema type 1 and type 2. HAE-C1INH: HAE with C1INH deficiency. HAE-nC1INH: HAE with normal C1INH.

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Bradykinin-mediated angioedema defined in international guidelines (WAO/EAACI)

Bradykinin-mediated angioedema (AE-BK)				Unknown mediator
C1INH deficiency/defect		C1INH normal		Idiopathic AE
Inherited	Acquired	Inherited	Acquired	Acquired
HAE-C1INH HAE-1 HAE-2	**AAE-C1INH**	**HAE-nC1INH** HAE-FXII HAE-PLG HAE-KNG …* HAE-UNK	**AE-DI (drug induced)** AE-ACEI AE-tPA	**AE-UNK**

AAE-C1INH: acquired angioedema due to C1INH deficiency. AE: angioedema. AE-ACEI: angioedema due to angiotensin-converting enzyme inhibitors. AE-BK: bradykinin-mediated angioedema. AE-DI: drug-induced angioedema. AE-tPA: angioedema due to tissue-type plasminogen activator. AE-UNK: angioedema due to unknown cause. C1INH: C1 inhibitor. EAACI: European Academy of Allergy and Clinical Immunology. HAE-1: hereditary angioedema type 1. HAE-2: hereditary angioedema type 2. HAE-C1INH: HAE with C1INH deficiency. HAE-FXII: hereditary angioedema due to factor XII mutation. HAE-KNG: hereditary angioedema due to kininogen 1 mutation. HAE-nC1INH: HAE with normal C1INH. HAE-PLG: hereditary angioedema due to plasminogen mutation. HAE-UNK; hereditary angioedema due to unknown mutation. WAO: World Allergy Organization.

*Other types of HAE-nC1INH associated with known genetic mutations for which a role, including indirect or conditional, of bradykinin as mediator of angioedema symptoms is not excluded not listed.

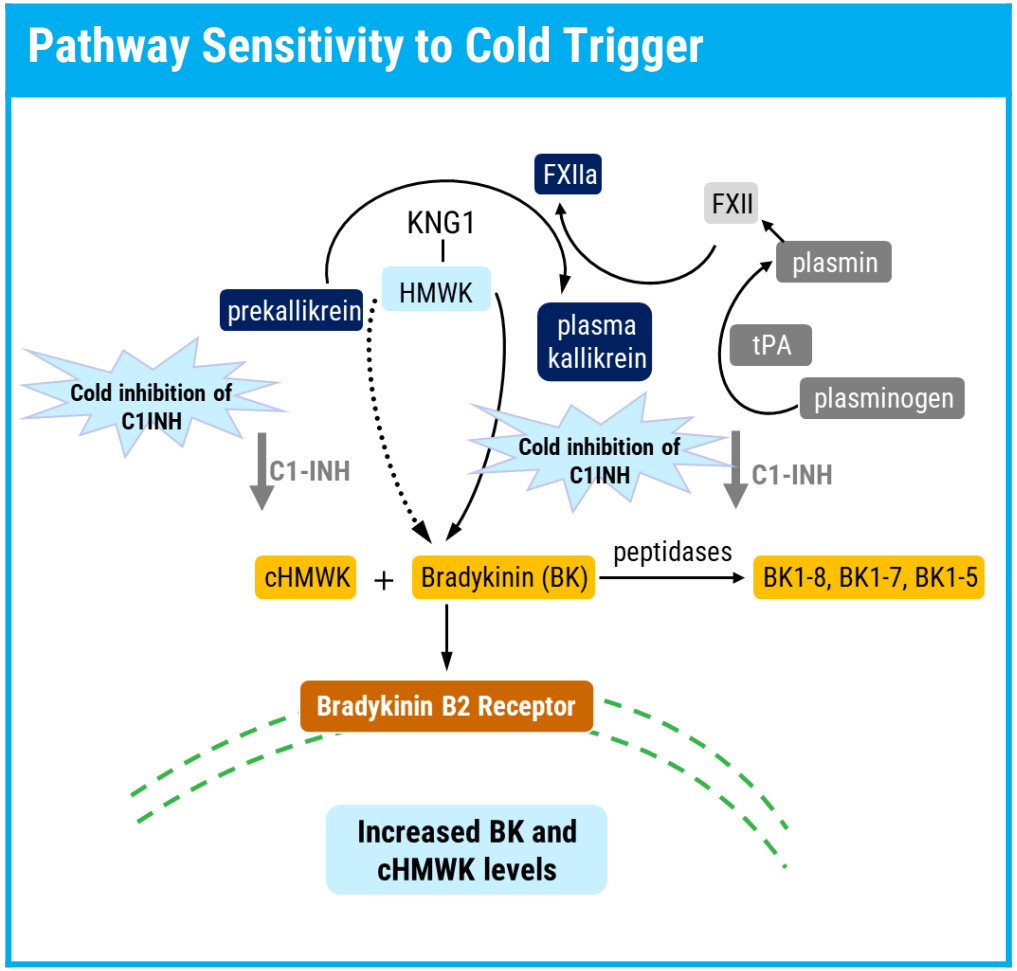
Adapted from: Mauer M et al. Allergy 2022;77:1961-1990; Reshef A et al. J Allergy Clin Immunol 2024;154:398-411.

Diagnosis of HAE-nC1INH and (H)AE-UNK is facilitated by guidelines, but remains difficult

	HAE-C1INH	AAE-C1INH	HAE-nC1INH with known genetic mutations	(H)AE-UNK
Laboratory findings				
C1INH level	**Low** (HAE-1), normal (HAE-2)	**Low**	Normal	Normal
C1INH function	**Low**	**Low**	Normal	Normal
C4 level	**Low**	**Low**	Normal	Normal
C1q level	Normal	**Low**	Normal	Normal
Genetic mutation	**SERPING1**	Absent	**Various genes***	Unknown
Anti-C1INH antibodies	Absent	**Present**/Absent	Absent	Absent
Family history	**Present**	Absent	**Present**	**Present** (HAE-UNK) Absent (AE-UNK)

AAE-C1INH: acquired angioedema due to C1INH deficiency. AE-UNK: angioedema due to unknown cause. C1INH: C1 inhibitor. C1q: complement component C1q. C4: complement component C4. HAE-1: hereditary angioedema type 1. HAE-2: hereditary angioedema type 2 HAE-C1INH: HAE with C1INH deficiency. HAE-nC1INH: HAE with normal C1INH. HAE-UNK; hereditary angioedema due to unknown mutation.
*Genes with mutations identified in HAE-nC1INH: F12, PLG, KNG1, ANGPT1, HS3ST6, MYOF, CPN1, DAB2IP.
Derived from: Mauer M et al. Allergy 2022;77:1961-199; Magerl M et al. J Allergy Clin Immunol Glob 2025;4:100446; Zuraw BL et al. Clin Rev Allergy Immunol 2025;68:24.

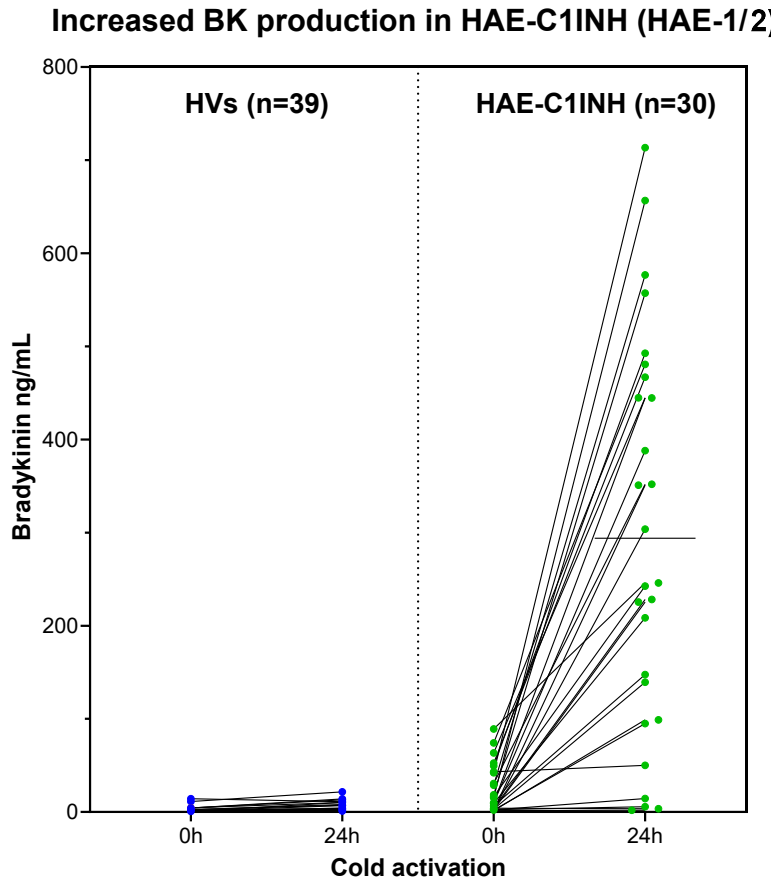
Novel approach to assess bradykinin formation may identify patients with bradykinin-mediated angioedema



BK: bradykinin. C1INH: C1 inhibitor. cHMWK: cleaved high molecular kininogen. FXII: Factor 12. HMWK: high molecular weight kininogen. i/cHMWK: intact/cleaved high molecular kininogen. tPA: tissue plasminogen activator.

Our novel biomarker assay reveals sensitized BK formation in patients with HAE-C1INH (HAE-1/2)

Bradykinin-induced angioedema (AE-BK)				Unknown mediator
C1INH deficiency/defect		C1INH normal		Idiopathic AE
Inherited	Acquired	Inherited	Acquired	Acquired
HAE-C1INH HAE-1 HAE-2	AAE-C1INH	HAE-nC1INH HAE-FXII HAE-PLG HAE-KNG1 … HAE-UNK	AE-DI (drug induced) AE-ACEi AE-tPA	AE-UNK

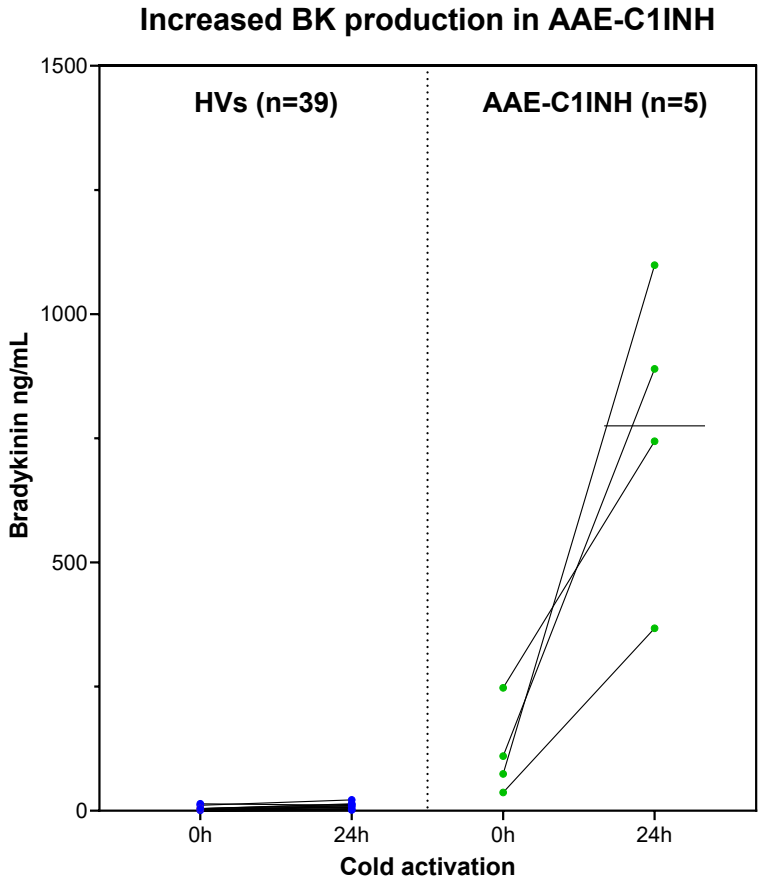


- BK production is dramatically increased by cold in plasma from HAE-C1INH patients
- Clear differentiation between healthy volunteers and HAE patients

BK: bradykinin. C1INH: C1 inhibitor. HAE: hereditary angioedema. HAE-1/2: hereditary angioedema type 1 and type 2. HAE-C1INH: HAE with C1INH deficiency. HVs: healthy volunteers.
Pardali E et al. Presented at the 14th C1 Inhibitor Deficiency and Angioedema Workshop; May 29–June 1, 2025; Budapest, Hungary.

Increased pathway sensitivity to cold and BK generation in patients with AAE-C1INH

Bradykinin-induced angioedema (AE-BK)				Unknown mediator
C1INH deficiency/defect		C1INH normal		Idiopathic AE
Inherited	Acquired	Inherited	Acquired	Acquired
HAE-C1INH HAE-1 HAE-2	AAE-C1INH	HAE-nC1INH HAE-FXII HAE-PLG HAE-KNG1 ...* HAE-UNK	AE-DI (drug induced) AE-ACEi AE-tPA	AE-UNK

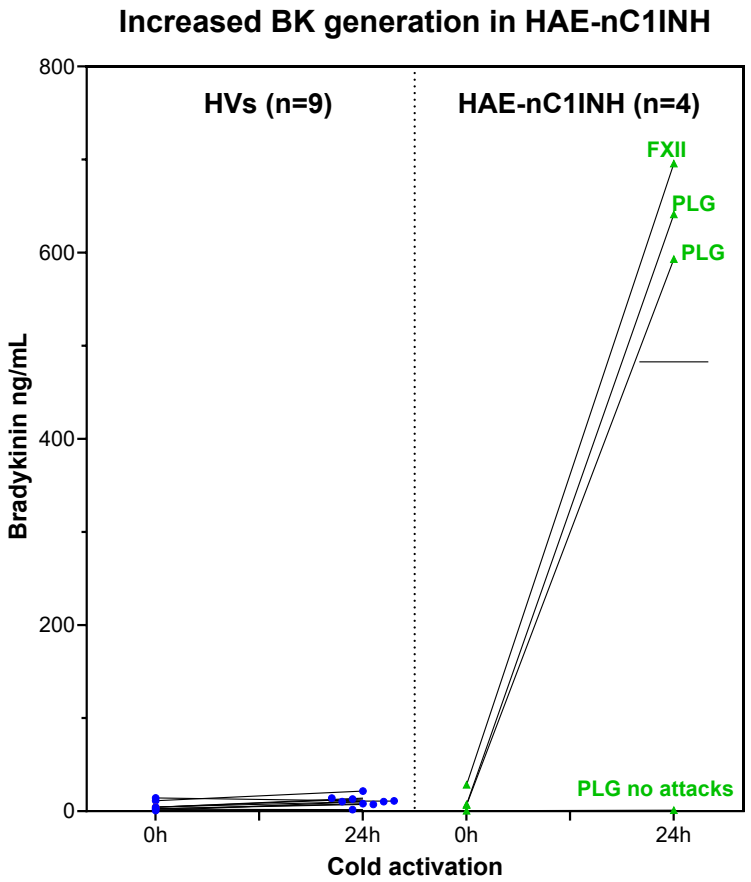


- BK production is dramatically increased by cold in plasma from AAE-C1INH patients
- Clear differentiation between healthy volunteers and AAE-C1INH patients

AAE-C1INH: acquired angioedema due to C1INH deficiency. BK: bradykinin. C1INH: C1 inhibitor. HVs: healthy volunteers.
Pharvaris, data on file.

Increased BK-formation to cold may support the identification of patients with HAE-nC1INH with genetic mutation

Bradykinin-induced angioedema (AE-BK)				Unknown mediator
C1INH deficiency/defect		C1INH normal		Idiopathic AE
Inherited	Acquired	Inherited	Acquired	Acquired
HAE-C1INH HAE-1 HAE-2	AAE-C1INH	HAE-nC1INH HAE-FXII HAE-PLG HAE-KNG1 ...* HAE-UNK	AE-DI (drug induced) AE-ACEi AE-tPA	AE-UNK



- Symptomatic HAE-FXII and HAE-PLG patients show a remarkably increase in BK production following cold activation
- In clinical practice, this assay may support the identification of people with nC1INH (FXII and PLG) in the absence of genetic testing
- Other mutations under investigation

BK: bradykinin. FXII: factor XII. HAE-FXII: HAE-FXII: hereditary angioedema due to factor XII mutation. HAE-KNG: hereditary angioedema due to kininogen 1 mutation. HAE-nC1INH: HAE with normal C1INH. HAE-PLG: hereditary angioedema due to plasminogen mutation. HMWK: high molecular weight kininogen. LMWK: low molecular weight kininogen. PLG; plasminogen. HVs: healthy volunteers.

Pardali E et al. Presented at the 14th C1 Inhibitor Deficiency and Angioedema Workshop; May 29–June 1, 2025; Budapest, Hungary.

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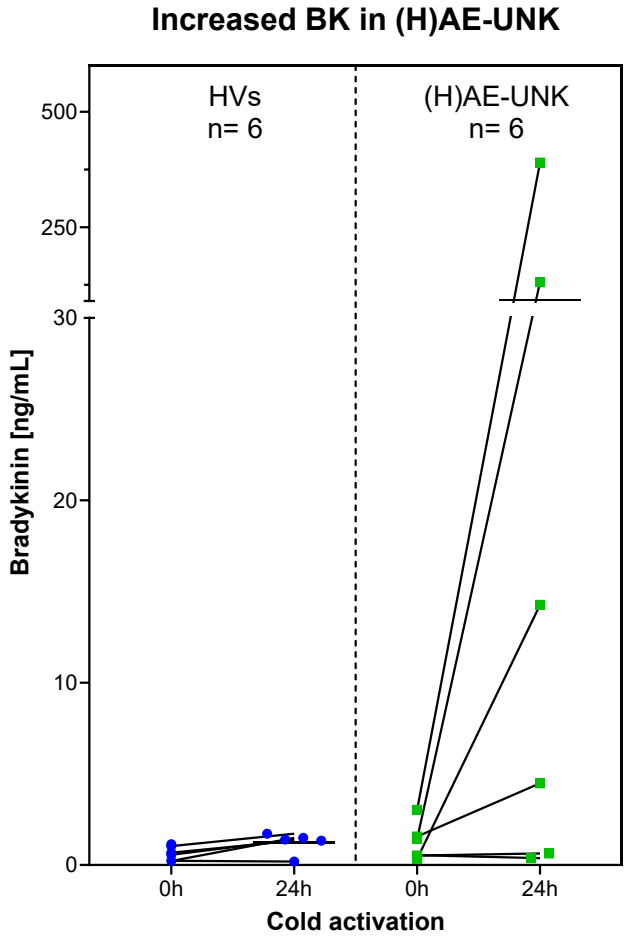
Cold activation enables identification of a BK signature in (H)AE-UNK patients

Bradykinin-induced angioedema (AE-BK)				Unknown mediator
C1INH deficiency/defect		C1INH normal		Idiopathic AE
Inherited	Acquired	Inherited	Acquired	Acquired
HAE-C1INH HAE-1 HAE-2	AAE-C1INH	HAE-nC1INH HAE-FXII HAE-PLG HAE-KNG1 - HAE-UNK	AE-DI (drug induced) AE-ACEi AE-tPA	AE-UNK



- Our biomarker assay can identify patients within the (H)AE-UNK population for whom BK production is sensitive to triggers
- Angioedema attacks in these patients are expected to be mediated by bradykinin and to respond to bradykinin B2 receptor antagonism

AE-UNK: angioedema due to unknown cause. BK: bradykinin. HVs: healthy volunteers, HAE-UNK; hereditary angioedema due to unknown mutation.
Pharvaris, data on file.

The clinically validated biomarker assay can be used to identify individual people with BK-mediated angioedema

Sensitivity to cold is a surrogate marker for involvement of BK-formation in (H)AE

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Evaluation of efficacy and safety of deucrictibant in AE-BK

Type of AE-BK	Deucrictibant for ODT	Deucrictibant for LTP
HAE-C1INH (HAE-1, HAE-2)	• RAPIDe Phase 2 results (RCT, LTE) • Eligible in ongoing RAPIDe Phase 3	• CHAPTER Phase 2 results (RCT, LTE) • Eligible in ongoing CHAPTER Phase 3
HAE-nC1INH **with genetic mutations** **without genetic mutations (UNK)**		
AAE-C1INH		

AAE-C1INH: acquired angioedema due to C1INH deficiency. AE-BK: bradykinin-mediated angioedema. HAE-1: hereditary angioedema type 1. HAE-2: hereditary angioedema type 2. HAE-C1INH: hereditary angioedema with C1INH deficiency. HAE-nC1INH, hereditary angioedema with normal C1INH. LTE: long-term extension. LTP: long-term prophylaxis. ODT: on-demand treatment. RCT: randomized controlled trial. UNK: unknown.

Diagnosis of HAE-nC1INH per current expert consensus

HAE-nC1INH presents a diagnostic challenge, given the current lack of validated biochemical tests to confirm diagnosis. As such, diagnosis must be made on clinical criteria.

History of recurrent angioedema without hives

Absence of exposure to medications known to cause angioedema

Family history

Normal C1INH level and function, C4 level

Known genetic mutation (some types)

- Response to a bradykinin B2 receptor antagonist
- Lack of response to mast-cell targeting therapies (high-dose antihistamines, omalizumab, montelukast)

C1INH: C1 inhibitor. C4: complement component C4HAE-nC1INH, hereditary angioedema with normal C1INH.

Derived from: Magerl M et al. J Allergy Clin Immunol Glob 2025;4:100446; Zuraw BL et al. Clin Rev Allergy Immunol 2025;68:24; O'Connor ME et al. Presented at the AAAAI/WAO Joint Congress; February 28–March 3, 2025; San Diego, CA, USA.

Treatment of AE-BK could benefit from controlled trial data for understudied AE types and additional treatment options

Despite the variety of treatment options, there remains key unmet needs in the treatment of AE-BK, including:

HAE-nC1INH Trial Data

There is a **lack of controlled trial data** for prophylactic and on-demand treatments for HAE-nC1INH.[1-4]

- Includes subtypes without identified genetic mutations.

AAE-C1INH Trial Data

There is also a need for both **controlled trial data** and **approved** prophylaxis and on-demand treatments for AAE-C1INH.[5]

Treatment Options Addressing Patient Needs

There is unmet medical need for prophylactic and on-demand treatments for HAE-C1INH that will be able to address the **individual patient needs** such as:[6-8]

- **Efficacy**
- **Safety/tolerability**
- **Administration regimen** and related experiences (e.g., portability, handling)

AAE-C1INH: acquired angioedema due to C1INH deficiency. AE-BK: bradykinin-mediated angioedema. AE: angioedema. HAE-C1INH: hereditary angioedema with C1INH deficiency. HAE-nC1INH, hereditary angioedema with normal C1INH.

[1]Fijen LM et al. N Engl J Med 2022;386:1026-1033. [2]Zuraw BL et al. Clin Rev Allergy Immunol 2025;68:24. [3]Riedl MA et al. Front Immunol 2025;16:1502325. [4]Andembry® Summary of product characteristics, Mar 2025, https://www.ema.europa.eu/en/documents/product-information/andembry-epar-product-information_en.pdf (accessed 3 June 2025). [5]Petersen RS et al. J Allergy Clin Immunol 2024;154:179-183. [6]Covella B et al. Future Pharmacol 2024;4:41-53. [7]Betschel SD et al. J Allergy Clin Immunol Pract 2023;11:2315-2325. [8]Betschel SD et al. Allergy Asthma Clin Immunol 2024;20:43.

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Evaluation of efficacy and safety of deucrictibant in AE-BK

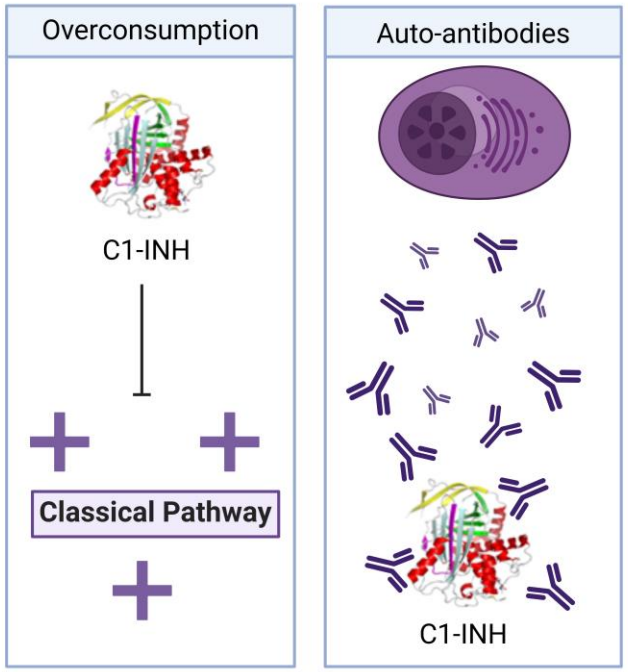
Type of AE-BK	Deucrictibant for ODT	Deucrictibant for LTP
HAE-C1INH (HAE-1, HAE-2)	• RAPIDe Phase 2 results (RCT, LTE) • Eligible in ongoing RAPIDe Phase 3	• CHAPTER Phase 2 results (RCT, LTE) • Eligible in ongoing CHAPTER Phase 3
HAE-nC1INH **with genetic mutations** **without genetic mutations (UNK)**	• Eligible in ongoing RAPIDe Phase 3	• Eligible in ongoing CHAPTER Phase 3
AAE-C1INH		

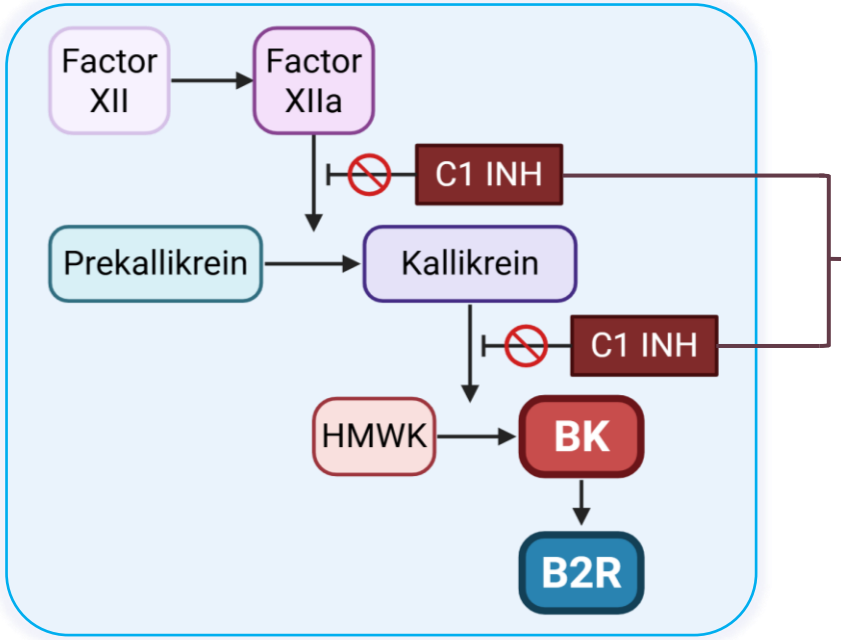
AAE-C1INH: acquired angioedema due to C1INH deficiency. AE-BK: bradykinin-mediated angioedema. HAE-1: hereditary angioedema type 1. HAE-2: hereditary angioedema type 2. HAE-C1INH: hereditary angioedema with C1INH deficiency. HAE-nC1INH, hereditary angioedema with normal C1INH. LTE: long-term extension. LTP: long-term prophylaxis. ODT: on-demand treatment. RCT: randomized controlled trial. UNK: unknown.

Acquired angioedema with C1 inhibitor deficiency (AAE-C1INH)

- Estimated prevalence approx. 1:500,000
- Due to overconsumption or neutralization of C1INH
- Secondary (e.g., lymphoproliferative/autoimmune conditions, MGUS)
- No ODT/LTP approved therapies for angioedema manifestations

Bradykinin-induced angioedema (AE-BK)				Unknown mediator
C1INH deficiency/defect		C1INH normal		Idiopathic AE
Inherited	Acquired	Inherited	Acquired	Acquired
HAE-C1INH HAE-1 HAE-2	AAE-C1INH	HAE-nC1INH HAE-FXII HAE-PLG HAE-KNG1 …* HAE-UNK	AE-DI (drug induced) AE-ACEi AE-tPA	AE-UNK





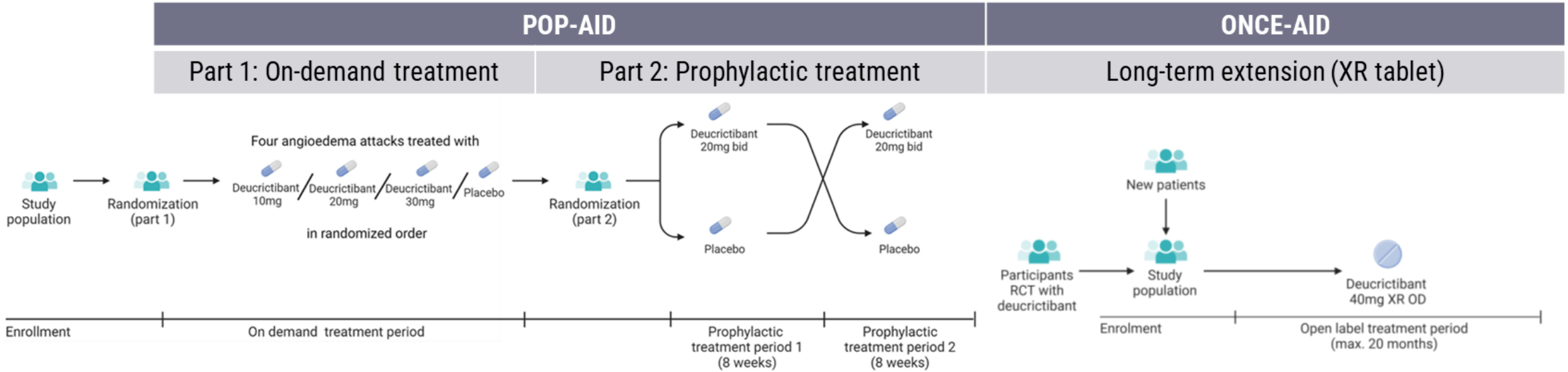
AAE-C1INH

- C1INH consumption
- C1INH neutralization

AAE-C1INH: acquired angioedema due to C1INH deficiency. B2R: bradykinin B2 receptor. BK: bradykinin. C1INH: C1 inhibitor. HMWK: high molecular weight kininogen. LTP: long-term prophylaxis. MGUS: Monoclonal gammopathy of undetermined significance. ODT: on-demand treatment.

Cicardi M et al. Allergy Asthma Clin Immunol 2010;6:14. de Lange M et al. Presented at the 14th C1 Inhibitor Deficiency and Angioedema Workshop; May 29–June 1, 2025; Budapest, Hungary

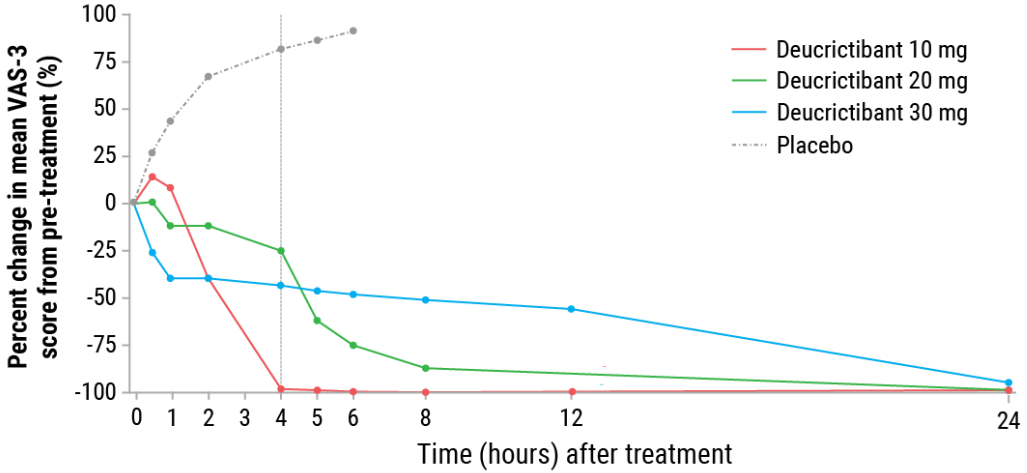
POP-AID and ONCE-AID study design



Bid: twice daily. OD: once daily. RCT: randomized controlled trial. XR: extended-release.

Petersen RS et al. J Allergy Clin Immunol 2024;154:179-183. de Lange M et al. Presented at the 14th C1 Inhibitor Deficiency and Angioedema Workshop; May 29–June 1, 2025; Budapest, Hungary.

POP-AID study: deucrictibant for treatment (Part 1) and prevention (Part 2) of AAE-C1INH attacks



Attacks per month	Patient 1	Patient 2	Patient 3
Baseline	1.2	1.2	0.9
Placebo	2.0	0.6	1.0
Deucrictibant	0	0	0

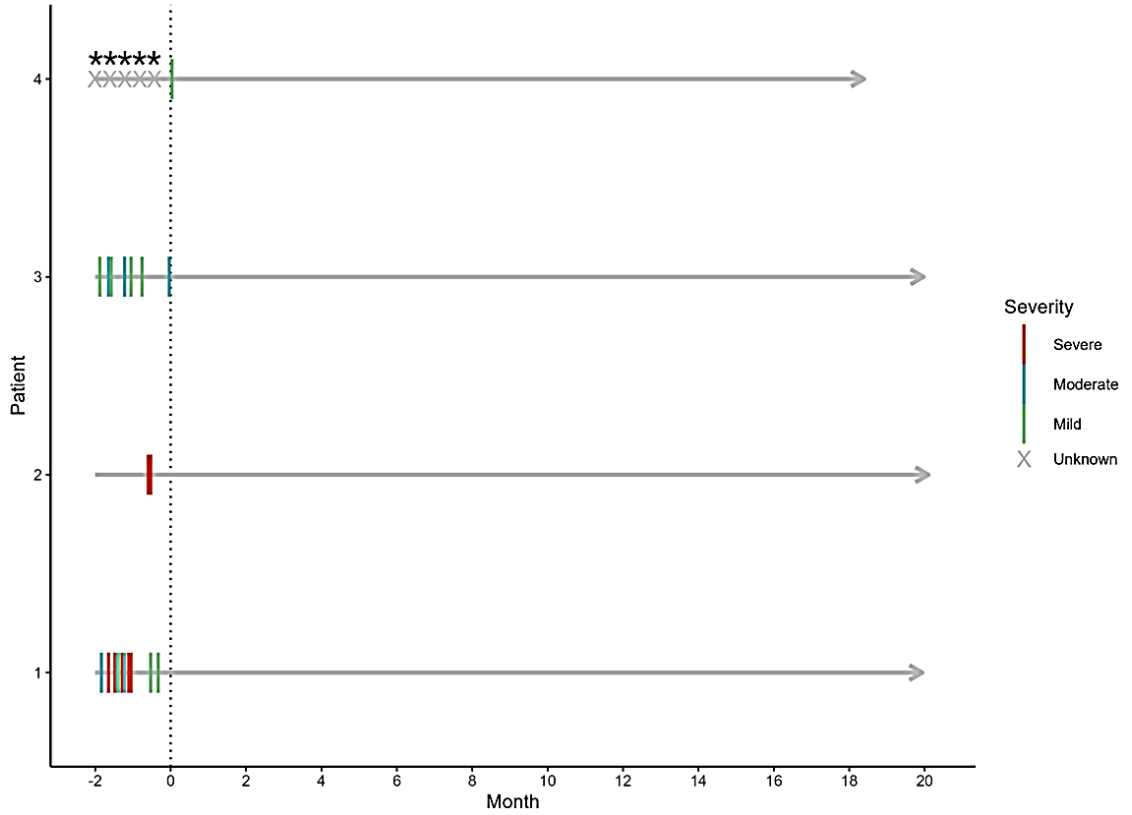
	Incidence in 4-wk period, no. (%)	
	Deucrictibant	Placebo
Any adverse event	5 (0.8)	4 (0.7)
Any drug-related adverse event	1 (0.2)*	0
Any serious adverse event	0	0
Treatment discontinued owing to an adverse event	0	0
Headache	1 (0.2)	0 (0)
Viral infection	0 (0)	2 (0.3)
Upper abdominal pain	1 (0.2)	0
Rash erythematous (sunburn-like)	1 (0.2)	0
Fatigue (heavy feeling throughout the whole body)	1 (0.2)	1 (0.2)
Dysgeusia (bitter taste)	1 (0.2)	1 (0.2)

Antagonism of the bradykinin B2 receptor with deucrictibant has the potential to effectively and safely treat and prevent angioedema due to acquired C1-inhibitor deficiency

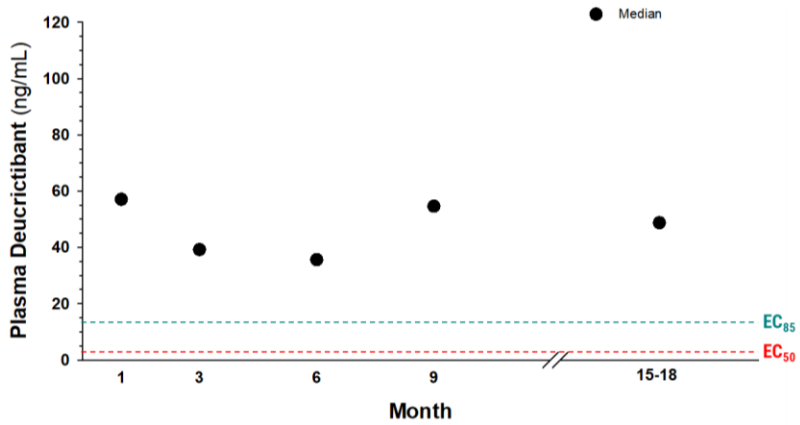
AAE-C1INH: acquired angioedema due to C1INH deficiency. VAS-3: 3-item visual analogue score.
Petersen RS et al. J Allergy Clin Immunol 2024;154:179-183.

ONCE-AID study: deucrictibant extended-release (XR) tablet for prevention of AAE-C1INH attacks

Efficacy (AAE-C1INH attacks)



Pharmacokinetics



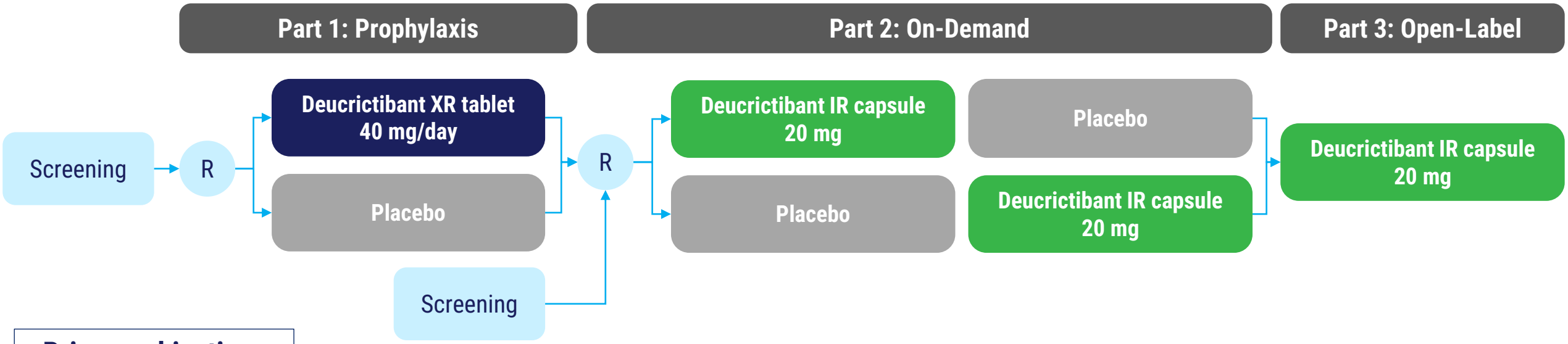
Safety (adverse events)

Incidence, n (grade)	
Any adverse event (AE)	29
Severity of AEs	
Mild	20
Moderate	6
Severe	3
Any drug-related AE	0
Any serious AE	1
Treatment discontinued due to AE	0

AAE-C1INH: acquired angioedema due to C1INH deficiency.
Adapted from: de Lange M et al. Presented at the 14th C1 Inhibitor Deficiency and Angioedema Workshop; May 29–June 1, 2025; Budapest, Hungary. Pharvaris, data on file.

CREAATE: deucrictibant for treatment of AAE-C1INH

Pivotal, global Phase 3 for both prophylaxis and on-demand treatment



Primary objectives

Part 1
Efficacy of deucrictibant XR tablet vs. placebo for prophylaxis against AAE-C1INH attacks

Part 2
Efficacy of deucrictibant IR capsule vs. placebo as on-demand treatment on time to symptom relief during AAE-C1INH attacks

Part 3
Safety and tolerability of deucrictibant IR capsule for on-demand treatment of AAE-C1INH attacks

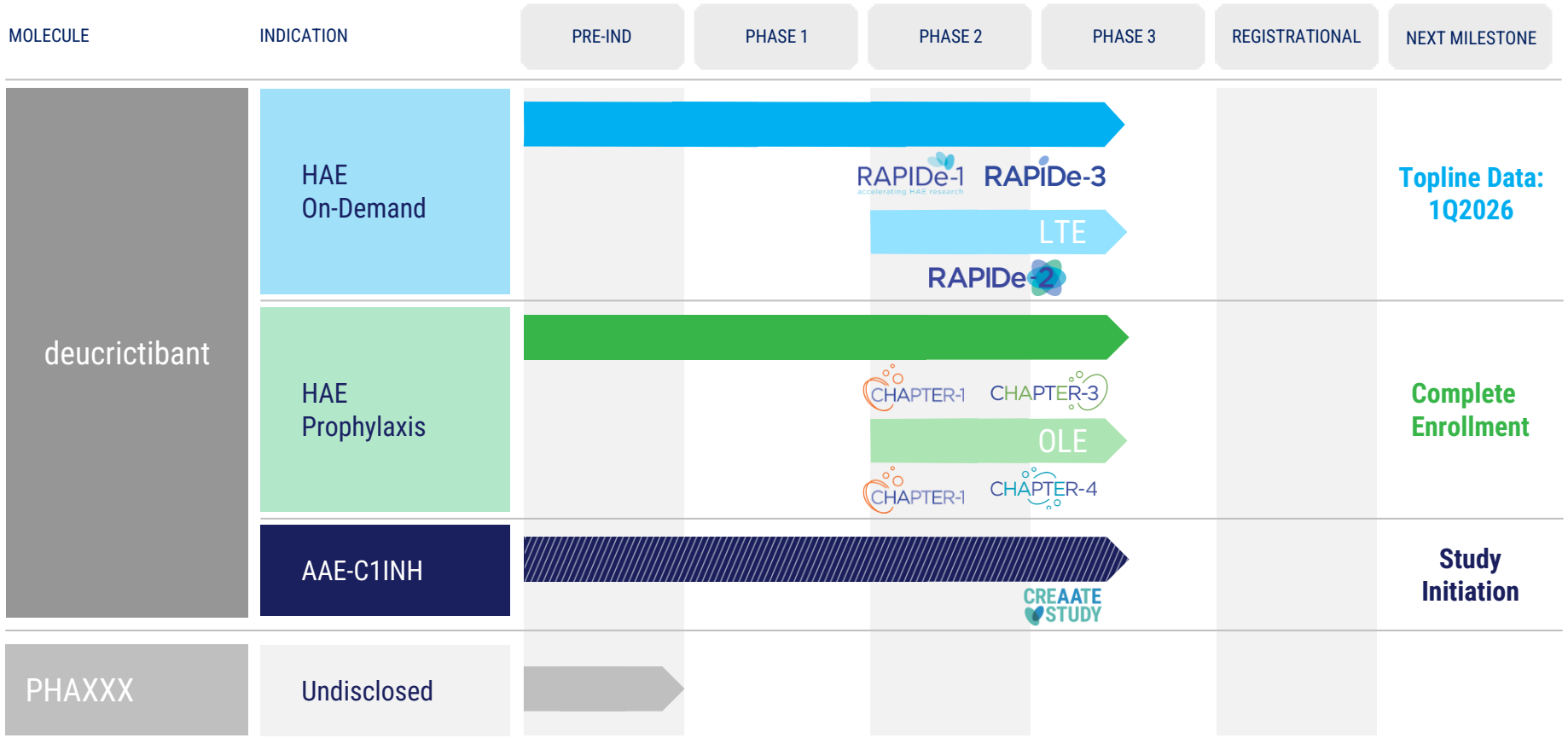
AAE-C1INH: acquired angioedema due to C1INH deficiency. IR: immediate-release. R: randomization. XR: extended-release.

Evaluation of efficacy and safety of deucrictibant in AE-BK

Type of AE-BK	Deucrictibant for ODT	Deucrictibant for LTP
HAE-C1INH (HAE-1, HAE-2)	• RAPIDe Phase 2 results (RCT, LTE) • Eligible in ongoing RAPIDe Phase 3	• CHAPTER Phase 2 results (RCT, LTE) • Eligible in ongoing CHAPTER Phase 3
HAE-nC1INH **with genetic mutations** **without genetic mutations (UNK)**	• Eligible in ongoing RAPIDe Phase 3	• Eligible in ongoing CHAPTER Phase 3
AAE-C1INH	• Independent POP-AID study • Planned Phase 3 trial	• Independent POP-/ONCE-AID studies • Planned Phase 3 trial

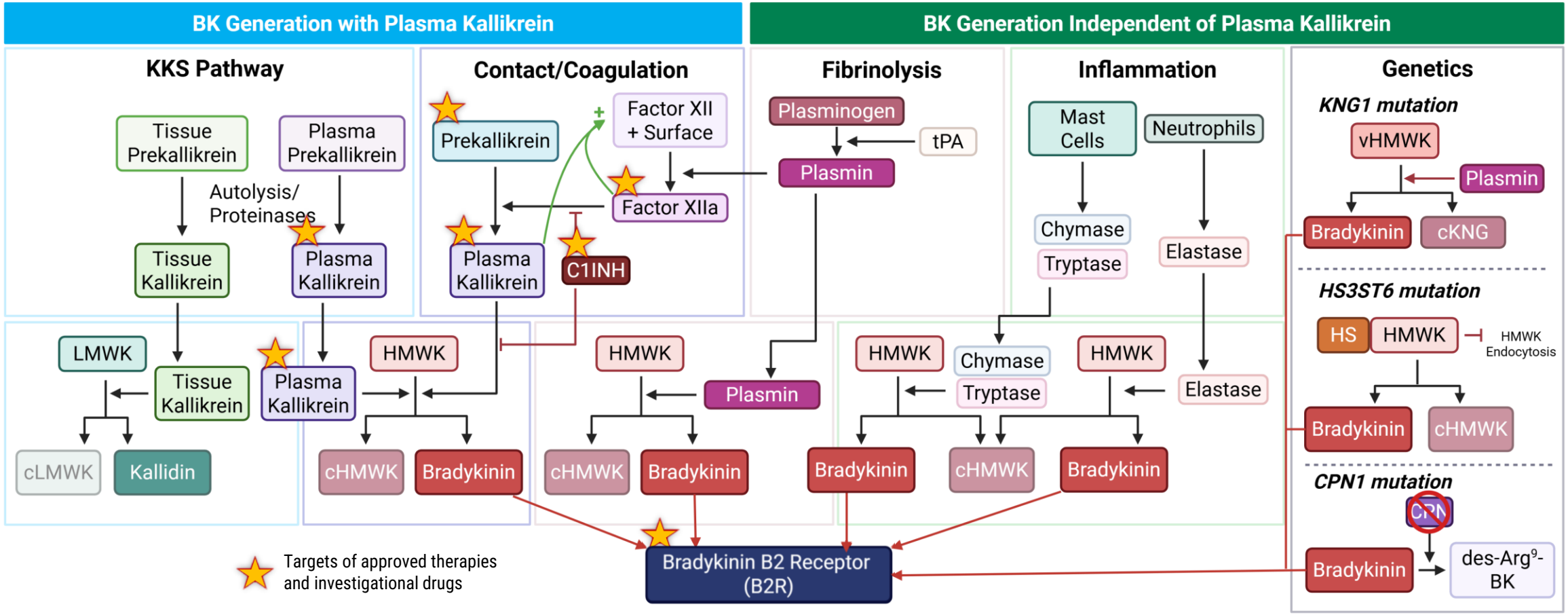
AAE-C1INH: acquired angioedema due to C1INH deficiency. AE-BK: bradykinin-mediated angioedema. HAE-1: hereditary angioedema type 1. HAE-2: hereditary angioedema type 2. HAE-C1INH: hereditary angioedema with C1INH deficiency. HAE-nC1INH, hereditary angioedema with normal C1INH. LTE: long-term extension. LTP: long-term prophylaxis. ODT: on-demand treatment. RCT: randomized controlled trial. UNK: unknown.

Deucrictibant for the prevention and treatment of bradykinin-mediated angioedema



Notes: AAE-C1INH: acquired angioedema due to C1 inhibitor deficiency. HAE: hereditary angioedema. LTE: long-term extension. OLE: open-label extension.
Source: RAPIDe-1 (NCT04618211). RAPIDe-2 (NCT05396105). RAPIDe-3 (NCT06343779). CHAPTER-1 (NCT05047185). CHAPTER-3 (NCT06669754). CHAPTER-4 (NCT06679881).

Current therapeutic approaches for AE-BK target different areas of the BK-forming pathways

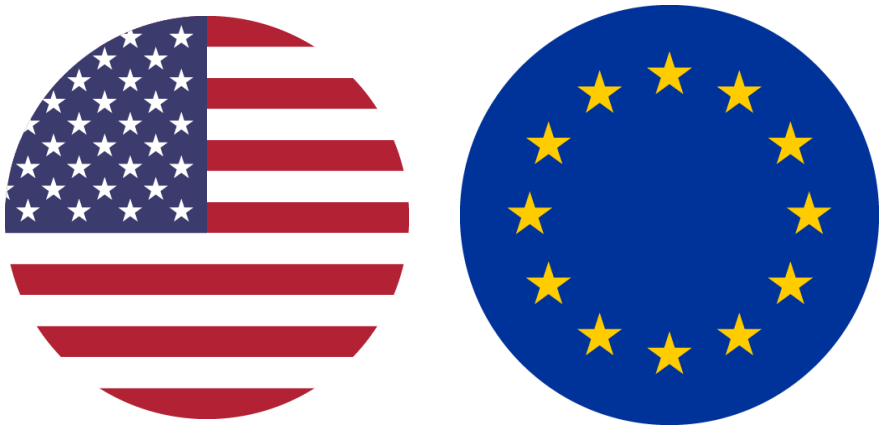


AE-BK, bradykinin-mediated angioedema; Arg, arginine; B2R, bradykinin B2 receptor; BK, bradykinin; CPN, carboxypeptidase N; C1INH, C1 esterase inhibitor; c, cleaved; factor XIIa, activated Factor XII; HMWK, high molecular weight kininogen; HS3ST6, heparan sulfate 3-*O*-sulfotransferase 6 gene; HS, heparan sulfate; KKS, kallikrein-kinin system; KNG, kininogen, LMWK, low molecular weight kininogen; tPA, tissue plasminogen activator; v, variant.

1. Maurer M, et al. *Allergy*. 2022;77(7):1961-1990. **2.** Ritchie BC. *Transfus Apher Sci*. 2003;29(3):259-267. **3.** Guo Y, et al. *Orphanet J Rare Dis*. 2022;17(1):399. **4.** Gakuba C, Gaberel T. *Primer on Cerebrovascular Diseases*. 2nd ed. 2017:82-86. **5.** Schmaier AH. *J Thromb Haemost*. 2016;14(1):28-39. **6.** Bryant JW, Shariat-Madar Z. *Cardiovasc Hematol Agents Med Chem*. 2009;7(3). **7.** Ramachandran R, et al. *Pharmacol Rev*. 2016;68(4). **8.** Kaplan AP. *Blood*. 2022;138(18):2732-2733. **9.** Cap AP. *Blood*. 2016;116(5):863-870. **10.** Kozik A, Moore RB, Potempa J, et al. *J Biol Chem*. 1998;273(50):33224-33229. **11.** Craig TJ, et al. *Lancet*. 2023;401(10382):1079-1090. **12.** Fijen LM, et al. *N Engl J Med*. 2022;386(11):1026-1033. **13.** Nunes LR, et al. *Arch Clin Cases*. 2023;10(3):138-141. **14.** Kanani A, et al. *Allergy Asthma Clin Immunol*. 2018;14(Suppl 2):59.

U.S. and EU regulatory authorities have granted Orphan Drug Designation for deucrictibant as a

> *"treatment of bradykinin-mediated angioedema"*



This presentation includes data for an investigational product not yet approved by regulatory authorities.

Q&A

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