

SCEMBLIX - Mechanism of action (MOA) - HCP

Prescribing information

Image



Image



SCEMBLIX® \blacktriangledown (asciminib) is indicated for the treatment of adult patients with Philadelphia chromosome-positive chronic myeloid leukaemia (Ph + CML) in chronic phase (CP), previously treated with two or more tyrosine kinase inhibitors, and without a known T315I mutation. 1

Mechanism of action (MOA)

SCEMBLIX®▼ (asciminib) is the first and only STAMP inhibitor²⁻⁴

Learn how the unique MOA of SCEMBLIX enhances its specificity in treating CML.²

SCEMBLIX targets a different site on BCR-ABL1 - the myristoyl pocket²⁻⁴



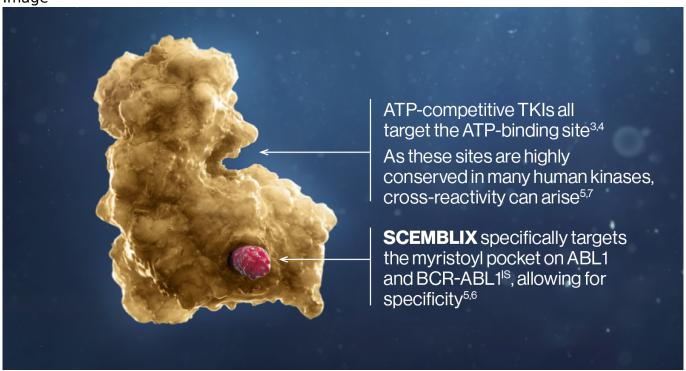


In people who do not have CML, the myristoyl pocket is occupied by the N-terminal portion

of ABL1, maintaining the protein in an inactive conformation. 5,6

In BCR-ABL1, the myristoyl pocket is vacant, activating the kinase. 5,6

Image



SCEMBLIX is a first-in-class STAMP inhibitor. Binding specifically to the myristoyl pocket, it potently inactivates BCR-ABL1 via allosteric inhibition.²

With its unique MOA, SCEMBLIX offers a different approach for treating CML⁵⁻⁷

SCEMBLIX demonstrated superior efficacy and a favourable safety profile vs bosutinib at Week 24²

Explore the data

ATP, adenosine triphosphate; BCR-ABL, breakpoint cluster region and Abelson murine leukaemia viral oncogene homologue; CI, confidence interval; CML, chronic myeloid leukaemia; CP, chronic phase; MMR, major molecular response; MOA, mechanism of action; Ph + CML, Philadelphia chromosome-positive chronic myeloid leukaemia; STAMP, specifically targeting the ABL1 myristoyl pocket; TKI, tyrosine kinase inhibitor.

For further information, please refer to the **Summary of Product Characteristics**.

References:

- 1. SCEMBLIX (asciminib) Summary of Product Characteristics.
- 2. Réa D, et al. *Blood* 2021;138(21):2031-2041.
- 3. Redaelli S, et al. J Clin Oncol 2009;27(3):469-471.
- 4. Schoepfer J, et al. J Med Chem 2018;61(18):8120-8135.
- 5. Hughes TP, et al. N Engl J Med 2019;381(24):2315-2326.
- 6. Manley PW, et al. Leuk Res 2020;98:106458.
- 7. lacob RE, et al. *PLoS One* 2011;6(1):e15929.

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