# **U**NOVARTIS

# SCEMBLIX - The unmet need in CML - HCP

### Scemblix® (asciminib) prescribing information

# Glivec® (imatinib) prescribing information

### Image



Image



SCEMBLIX®  $\mathbf{\nabla}$  (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 T3151 mutation.<sup>1</sup>

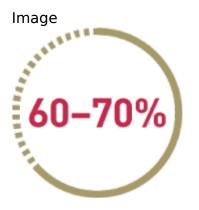
# The unmet need in chronic myeloid leukaemia

Tyrosine kinase inhibitors (TKIs) have improved CML prognosis, yet survival remains poor in patients on  $\geq$ 3rd line therapy.<sup>2,3</sup>

Many patients on TKIs face treatment failure or discontinuation due to intolerance, with up

to half of patients discontinuing 1st line imatinib within 5 years.<sup>3</sup>

During 2nd-line treatment:<sup>3</sup>



# of patients fail to achieve major molecular response (MMR) $(n=1279)^{*^3}$

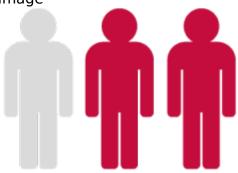


# of patients fail to achieve complete cytogenetic response (CCyR) within 2 years $(n=958)^{*^3}$

\*Results for MMR and CCyR rates are taken from multiple studies including different 2nd line medications. This is a meta-analysis.<sup>3</sup>

In CML patients, a low 8-year overall survival was associated with being on  $\geq$ 3rd line treatment.<sup>†2</sup>

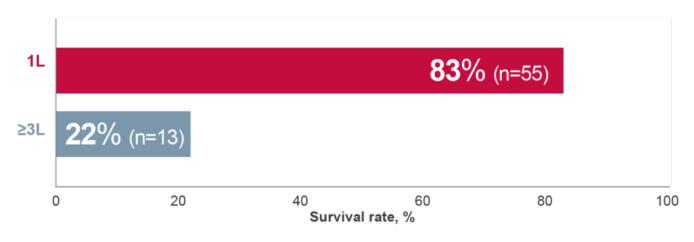
Image



In approximately 2/3 of those who had at least 1 TKI switch it was due to resistance<sup>†4</sup> (n=73/113)

#### 8-year overall survival, N=902

#### Image



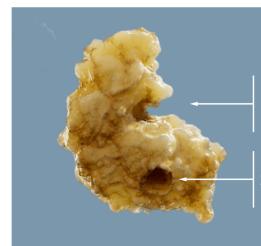
Adapted from Bosi GR, et al. 2019.<sup>2</sup>



Optimal monitoring is important to assess treatment benefits and inform the decision to switch.<sup>5,6</sup> When a TKI fails, timely switching can limit the progression of disease<sup>6</sup>

A 2nd generation TKI may have limited benefit in a  $\geq$ 3L setting, after the failure of another 2nd generation TKI and 1st generation TKI prior.<sup>3</sup> Patients in  $\geq$ 3L treatment may need a different mechanism of action to optimise outcomes.<sup>2,7-9</sup>

Image



First- and second-generation TKIs all target the ATP-binding site of BCR-ABL1<sup>10</sup>

**Myristoyl Pocket SCEMBLIX (asciminib)** binds to the myristoyl pocket, inhibiting kinase activity<sup>1</sup>

"You can end up running out of options for patients who experience side effects. Then you need to consider transplant after 4th line or chemotherapy options"

# Adapted from haematologist quote<sup>11</sup>

"In a year I had gone from taking one drug and living a normal life to having zero options"

# Adapted from 5L CML patient quote<sup>12</sup>

# SCEMBLIX is the first and only STAMP inhibitor, specifically targeting the ABL1 myristoyl pocket<sup>10,13,14</sup>

### Discover the MOA

The ASCEMBL trial did not restrict to Ph+ patients with CP-CML. SCEMBLIX is indicated in adults with Ph+ CP-CML previously treated with two or more tyrosine kinase inhibitors and without a known T315I mutation.<sup>1,13</sup>

<sup>†</sup>Data from a retrospective, non-interventional study conducted at 21 UK NHS secondary and tertiary care centres on 257 patients with CML.<sup>4</sup>

1L, first line; 3L, third line; ATP, adenosine triphosphate; BCR-ABL, breakpoint cluster region and Abelson murine leukaemia viral oncogene homologue; CCyR, complete cytogenetic response; CI, confidence interval; CML, chronic myeloid leukaemia; CP, chronic phase; MCyR, major cytogenetic response; MMR, major molecular response; MOA, mechanism of action; NHS, National Health Service; Ph+, Philadelphia chromosome positive; STAMP, specifically targeting the ABL1 myristoyl pocket; TKI, tyrosine kinase inhibitor.

For further information, please refer to the <u>Summary of Product Characteristics</u>.

### References

- 1. SCEMBLIX (asciminib) Summary of Product Characteristics.
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- 3. Cortes JE and Lang F. J Hematol Oncol 2021;14(1):44.
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- 5. Smith G, et al. Br J Haematol 2020;191:171-193.
- 6. Hochhaus A, et al. Leukaemia 2020;34:1495-1502.
- 7. Soverini S, et al. *Blood* 2009;114(10):2168-2171.
- 8. Garg RJ, et al. *Blood* 2009;114(20):4361-4368.
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- 10. Schoepfer J, et al. J Med Chem 2018;61(18):8120-8135.
- 11. Novartis data on file; Asc001.
- 12. Novartis data on file; Hae003.
- 13. Réa D, et al. *Blood* 2021;138(21):2031-2041.
- 14. Redaelli S, et al. J Clin Oncol 2009;27(3):469-471.
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