

TOLREMO therapeutics Announces TT125-802 is the First CBP/p300 Bromodomain Inhibitor to Show Clinical Activity in Solid Tumors

- Deep and durable responses in drug-resistant KRAS-G12C- and EGFR-mutant NSCLC
- Best-in-class safety profile highlights potential for development in combination to treat patients with KRAS-G12C-mutant NSCLC, EGFR-mutant NSCLC and R/R multiple myeloma

Basel, June 04, 2025 – TOLREMO therapeutics AG (TOLREMO), a clinical stage biotechnology company pioneering non-oncogene addiction in cancer, today announced data from its ongoing Phase I study of TT125-802, a novel, orally administered bromodomain inhibitor of CBP/p300, for patients with advanced solid tumors who have relapsed or are refractory to standard-of-care therapies, including an abstract published at the 2025 American Society of Clinical Oncology (ASCO) annual meeting. Updated data from the study was discussed at TOLREMO's SAB meeting during ASCO and recorded in a virtual ASCO data update.

To date TT125-802 shows impressive anti-tumor activity in advanced solid tumors, including deep and durable responses in non-small cell lung cancer (NSCLC). In addition, TT125-802 demonstrates a best-in-class safety profile without thrombocytopenia, the primary toxicity associated with its class of inhibitors.

"Five out of seven NSCLC patients on this study experienced tumor shrinkage following progression on their prior therapy. The two patients with KRAS-G12C- or EGFR-mutant NSCLC – the two tumor types we had preclinically selected as target indications - each showed deep and durable responses to single agent TT125-802. There remains a large and urgent need for more effective and tolerable therapies, and TT125-802 has the potential to offer an improved therapeutic option through resistance-targeted combinations," said Florian Vogl, M.D., PhD, Chief Medical Officer at TOLREMO.

"EGFR- and KRAS-targeted therapies have historically been limited by intrinsic and acquired resistance. Inhibiting transcriptional mechanisms of resistance via CBP/p300 represents an exciting and much needed opportunity for more effective and tolerable therapies. TT125-802 has shown impressive activity in NSCLC even as a monotherapy, and I look forward to combining the drug with EGFR and KRAS-G12C inhibitors to provide better treatment options to my patients," added **Pasi Jänne**, **M.D.**, **PhD**, **Scientific Advisory Board member at TOLREMO** and Senior Vice President for Translational Medicine and the Director of the Belfer Center for Applied Cancer Science at the Dana-Farber Cancer Institute and a Professor of Medicine at Harvard Medical School.

"The best-in-class safety and activity in solid tumors is particularly notable because we didn't set out to develop a CBP/p300 inhibitor - initially, TT125-802 was developed phenotypically to inhibit non-oncogene addiction in cancer," said **Stefanie Flückiger-Mangual**, **PhD**, **CEO** and **co-founder of TOLREMO**. "By starting with the biology around transcriptional addiction, it led us to CBP/p300 as a novel target in this space and yielded best-in-class chemistry, enabling a wider therapeutic window and broadening the clinical potential of our asset both in hematological malignancies as well as in solid tumors."

Dr. Jänne and Dr. Omar Saveedra Santa Gadea, a medical oncologist at NEXT Oncology Hospital Quirónsalud in Barcelona, Spain also shared their perspective on the importance of these results, including specific review of clinical case studies as part of an ASCO video update at https://www.tolremo.com.

About TOLREMO



TOLREMO therapeutics is pioneering a comprehensive new approach to tackle non-oncogene addiction in cancer by blocking transcriptional escape pathways that operate parallel to the primary oncogene signaling axis. Leveraging our proprietary phenotypic screening platform, we have uncovered a novel role for CBP/p300 as an epigenetic master regulator of transcriptional resistance. Our clinical compound, TT125-802, is an orally available small molecule inhibitor of the bromodomain of CBP/p300 with a differentiated, best-in-class safety profile and activity as single agent in solid tumors. Targeting non-oncogene addiction represents a differentiated strategy to address a major challenge in cancer treatment, with significant therapeutic potential both as a monotherapy as well as in combination with targeted therapies in solid tumors and hematological malignancies.

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