Compass Therapeutics Presents Novel Mechanism of Action Data for CTX-10726 (PD-1 x VEGF-A Bispecific Antibody) at the SITC 2026 Annual Meeting

November 6, 2026

- *CTX-10726, a PD-1 x VEGF-A bispecific antibody discovered and engineered at Compass, exhibits a unique mechanism of action that has not been previously reported in this class of drugs, characterized by VEGF-A-dependent cleavage of PD-1 receptors from T cells resulting in conversion of PD-1 positive T cells into PD-1 negative T cells.*
- *PD-1 receptor cleavage by CTX-10726 was only observed in the presence of VEGF-A, suggesting targeted activity in the tumor microenvironment.*
- *A Phase 1 dose-escalation study of CTX-10726 in patients in the post-checkpoint setting with advanced solid tumors, including renal cell, gastroesophageal, endometrial, and hepatocellular cancers, is ongoing.*
- *In checkpoint inhibitor resistant preclinical mouse models, the combination of CTX-10726 and Compass's CD137 agonist, CTX-471, inhibited tumor growth more than either agent alone.*

BOSTON, Nov. 06, 2026 (GLOBE NEWSWIRE) -- Compass Therapeutics, Inc. (Nasdaq: CMPX), a clinical-stage, oncology-focused biopharmaceutical company developing proprietary antibody-based therapeutics to treat multiple human diseases, will present data on the novel mechanism of action for CTX-10726 at the 41st Society for Immunotherapy of Cancer (SITC) Annual Meeting. CTX-10726 is currently in a Phase 1 dose-escalation study in patients in the post-checkpoint setting with multiple types of advanced solid tumors.

Data highlights from the presentation include:

- **Novel PD-1 Cleavage Mechanism.** In addition to the expected dual blockade of VEGF-A and PD-1, preclinical studies also revealed a novel mechanism of action for CTX-10726. Specifically, the multimerization of CTX-10726 facilitates the cleavage and removal of PD-1 from the surface of T cells, converting PD-1 positive T cells into PD-1 negative T cells and extending the mechanism beyond simply checkpoint blockade and angiogenesis inhibition.

- **VEGF-A Enhanced Potency.** Adding VEGF-A increased the potency of CTX-10726 approximately ten-fold in a human immune-cell assay measuring interferon-gamma production, reducing the EC_{50} from 2.0 to 0.2 nM. In vitro studies showed PD-1 receptor cleavage by CTX-10726 in the presence of VEGF-A compared to PD-1 receptor internalization in the absence of VEGF-A. Neither pembrolizumab nor bevacizumab alone demonstrate comparable PD-1 cleavage. Two in vivo models further demonstrated the VEGF-A-dependent nature of PD-1 receptor cleavage by CTX-10726.

- **Combined Activity with CTX-471**. A mouse surrogate of CTX-10726 combined with CTX-471 (CD137 agonist antibody) further enhanced anti-tumor activity in mouse models, accompanied by coordinated upregulation of chemokines, interferon-stimulated genes, and inflammatory cytokines. These changes were associated with increased immune cell recruitment and establishment of a more permissive tumor microenvironment.

"These data reveal a new and differentiated mechanism of action for CTX-10726, setting it apart in this exciting new class of therapeutics," said Thomas Schuetz, MD, PhD, Chief Executive Officer. "We believe PD-1 receptor cleavage results in more potent and potentially more durable checkpoint inhibition, which supports and explains what we saw in our earlier studies. The dependence of this mechanism on VEGF links a traditionally anti-angiogenic agent directly to checkpoint control and may enhance the activity of CTX-10726 within VEGF-A-rich tumor microenvironments. We are also encouraged by the combination results with CTX-471 in checkpoint-resistant models, supporting further investigation of these complementary approaches."

The Phase 1 multiple ascending dose-escalation study of CTX-10726 includes four dosing cohorts (0.3, 1.0, 3.0, and 10.0 mg/kg) in a 3+3 format. The multi-center study is enrolling post-checkpoint patients with a prioritized set of locally advanced, unresectable or metastatic malignancies, including renal cell carcinoma, gastroesophageal cancer, hepatocellular carcinoma, and endometrial cancer, in whom standard of care therapies have failed.

Details of the presentation are as follows:

Title: VEGF-dependent cleavage of cell surface PD-1 mediated by CTX-10726, a novel PD-1×VEGF-A bispecific antibody
Date & Time: Saturday, November 7, 2026 from 10:00 AM–2:00 PM MT and 6:00–8:10 PM MT
Poster number: 678
Location: Phoenix Convention Center – North Building – Lower Level – Exhibition Halls 4-5

A copy of the presentation materials can be accessed on the Compass website at https://www.compasstherapeutics.com/pipeline/ once the presentation has concluded.

About CTX-10726
CTX-10726 is a tetravalent PD-1 x VEGF-A bispecific antibody discovered and engineered at Compass. CTX-10726 is designed to synergistically deliver VEGF-A blockade and checkpoint inhibition, potentially applicable to multiple solid tumor indications. The bispecific antibody demonstrates a highly stable structure with high affinity target binding. CTX-10726 exhibits more potent PD-1 suppression and a novel mechanism of action compared with data reported for other drugs in the class.

About Compass Therapeutics
Compass is a clinical-stage, oncology-focused biopharmaceutical company discovering and developing proprietary antibody-based therapeutics to treat multiple diseases. The company's scientific focus is on the relationship between angiogenesis, the immune system and tumor growth. Compass has a robust pipeline of novel product candidates designed to target multiple key biological pathways to drive an effective anti-tumor response, including angiogenesis modulation, immune activation within the tumor microenvironment, and reduction of tumor-driven immunosuppression. Compass is advancing their discovery candidates through clinical development to commercial-stage assets. Compass Therapeutics, Inc. was founded in 2014 and is headquartered in Boston, MA. For more information, visit www.compasstherapeutics.com.

Forward-Looking Statements
This press release contains forward-looking statements. Statements in this press release that are not purely historical are forward-looking statements. Such forward-looking statements include, among other things, references to Compass's financial position to continue advancing its product candidates, expectations about cash runway, business and development plans, and statements regarding Compass's product candidates, including their development and clinical trial milestones such as the expected trial design, timing of enrollment, patient dosing and data readouts, regulatory plans with respect to Compass's product candidates and the therapeutic potential thereof. Actual results could differ from those projected in any forward-looking statements due to numerous factors. Such factors include, among others, Compass's ability to raise the additional funding it will need to continue to pursue its business and product development plans, the inherent uncertainties associated with developing product candidates and operating as a development stage company, Compass's ability to identify additional product candidates for development, Compass's ability to develop, complete clinical trials for, obtain approvals for and commercialize any of its product candidates, competition in the industry in which Compass operates and market conditions. These forward-looking statements are made as of the date of this press release, and Compass assumes no obligation to update the forward-looking statements, or to update the reasons why actual results could differ from those projected in the forward-looking statements, except as required by law. Investors should consult all of the information set forth herein and should also refer to the risk factor disclosure set forth in the reports and other documents Compass files with the U.S. Securities and Exchange Commission (SEC) available at www.sec.gov, including without limitation Compass's latest Annual Report on Form 10-K, Quarterly Report on Form 10-Q and subsequent filings with the SEC.

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