Monte Rosa Therapeutics Presents Preclinical Data at American Association for Cancer Research (AACR) Annual Meeting 2026 on the Potential of its Cyclin E1 (CCNE1)-directed Molecular Glue Degrader to Treat CCNE1-amplified Solid Tumors

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CCNE1-directed molecular glue degrader (MGD) induced deep tumor regressions in CCNE1-amplified in vivo models of ovarian, breast, and gastric cancers

CCNE1-directed MGD demonstrated superior selectivity and reduced off-target activity compared to CDK2 inhibitors

Oral presentation on April 21, 2026, at 2:30 p.m. PT

BOSTON, April 20, 2026 (GLOBE NEWSWIRE) -- Monte Rosa Therapeutics, Inc. (Nasdaq: GLUE), a clinical-stage biotechnology company developing novel molecular glue degrader (MGD)-based medicines, today announced the company will present preclinical data highlighting the potential of its highly selective, first-in-class cyclin E1 (CCNE1)-directed MGD, MRT-55811, to treat CCNE1-amplified solid tumors at the American Association for Cancer Research (AACR) Annual Meeting 2026, being held April 17-22 in San Diego, CA.

"CCNE1 MGDs represent a first-in-class opportunity to directly target a frequently amplified driver oncogene in several solid tumor cancer populations with high unmet medical need. In CCNE1-amplified *in vivo* models of ovarian, gastric, and breast cancer, MRT-55811 demonstrated compelling monotherapy anti-tumor activity," said Sharon Townson, Ph.D., Chief Scientific Officer of Monte Rosa Therapeutics. "MRT-55811 also exhibited superior selectivity when compared to clinical-stage CDK2 inhibitors, suggesting that our CCNE1-directed MGDs could avoid the dose-limiting toxicities reported for these less selective agents. We believe that our oral CCNE1 degrader has the potential to provide clinical benefit across multiple cancer types where CCNE1 is amplified. These data also reinforce the power of our QuEEN™ discovery engine, as cyclin E1 represents yet another previously undruggable target we've successfully targeted. We anticipate submitting an IND for this program later this year."

The presentation, "Selective targeting of CCNE1 using molecular glue degraders for the treatment of CCNE1 amplified cancers" (Abstract Presentation Number 6778), will be presented by Ralph Tiedt, Ph.D., Vice President, Biology, Monte Rosa Therapeutics, at the Minisymposium, "Targeted Protein Degradation and Non-canonical Oncogenic Signaling," on April 21, 2026, from 2:30 p.m. to 4:30 p.m. PT.

Summary of results:

- MRT-55811 exhibited potent degradation and high selectivity for CCNE1, with no detectable degradation of closely related cyclins or cyclin-dependent kinases (CDKs), and favorable drug-like properties.
- MRT-55811 induced deep cyclin E1 degradation and downstream pathway suppression, as well as co-degradation of CDK2 within the cyclin E1/CDK2 holoenzyme complex in CCNE1-amplified cell lines.
- MRT-55811 demonstrated superior selectivity compared with clinical-stage CDK2 inhibitors, which exhibited significant off-target activity, as evidenced by kinome profiling and genetic modeling.
- In CCNE1-amplified cancer cell lines, MRT-55811 selectively inhibited cellular proliferation, while sparing cell lines without amplification.
- *In vivo*, MRT-55811 monotherapy resulted in tumor regression and pathway suppression in multiple CCNE1-amplified models.
- MRT-55811 downmodulated retinoblastoma (RB) protein phosphorylation and E2F-driven gene expression, demonstrating on-target effects in tumors grown *in vivo*.

About CCNE1 MGDs

Cyclin E1 (CCNE1) is a well-recognized human oncogene and critical driver of cell cycle progression and cell proliferation and was historically considered an undruggable target. It acts as the regulatory subunit of the CCNE1-CDK2 holoenzyme, which coordinates G1-S cell cycle progression and drives cell proliferation through RB phosphorylation and repression. CCNE1 is frequently amplified or overexpressed across multiple cancer types, including ovarian, endometrial, gastric, breast, and others. Leveraging a cryptic pocket, Monte Rosa's CCNE1-directed MGDs selectively degrade the cyclin E1/CDK2 holoenzyme complex, while sparing other proteins such as other closely related cyclins or CDKs. As a result of this exquisite selectivity, CCNE1-directed MGDs represent an opportunity to directly and selectively target a frequently amplified driver oncogene across multiple cancers.

About Monte Rosa
Monte Rosa Therapeutics is a clinical-stage biotechnology company developing highly selective molecular glue degrader (MGD) medicines for patients living with serious diseases. MGDs are small molecule protein degraders that have the potential to treat many diseases that other modalities, including other degraders, cannot. Monte Rosa's QuEEN™ (Quantitative and Engineered Elimination of Neosubstrates) discovery engine combines AI-guided chemistry, diverse chemical libraries, structural biology, and proteomics to rationally design MGDs with unprecedented selectivity. Monte Rosa has developed the industry's leading pipeline of first-in-class and only-in-class MGDs, spanning autoimmune and inflammatory diseases, oncology, and beyond, with three programs in the clinic. Monte Rosa has ongoing collaborations with leading pharmaceutical companies in the areas of immunology, oncology, and neurology. For more information, visit **www.monterosatx.com**.

Forward-Looking Statements
This communication includes express and implied "forward-looking statements," including forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995. Forward-looking statements include all statements that are not historical facts and in some cases, can be identified by terms such as "may," "might," "will," "could," "would," "should," "expect," "intend," "plan," "objective," "anticipate," "believe," "estimate," "predict," "potential," "continue," "ongoing," or the negative of these terms, or other comparable terminology intended to identify statements about the future. Forward-looking statements contained herein include, but are not limited to, statements about the therapeutic potential of Cyclin E1 (CCNE1)-directed Molecular Glue Degraders to Treat CCNE1-amplified Solid Tumors, including using the companies CCNE1-MGD known as MRT-55811, that CCNE1 MGDs, including MRT-55811, represent a first-in-class opportunity to directly target a frequently amplified driver oncogene in several solid tumor cancer populations with high unmet medical need, that CCNE1-directed MGDs, including MRT-55811, could avoid the dose-limiting toxicities reported for clinical-stage CDK2 inhibitors, that oral CCNE1 degraders, including MRT-55811, have the potential to provide clinical benefit across multiple cancer types where CCNE1 is amplified, and regarding the timing of filing any IND with FDA later this year, among others. By their nature, these statements are subject to numerous risks and uncertainties, including those risks and uncertainties set forth in our most recent Annual Report on Form 10-K for the year ended December 31, 2025, filed with the U.S. Securities and Exchange Commission on March 20, 2026, and any subsequent filings, that could cause actual results, performance or achievement to differ materially and adversely from those anticipated or implied in the statements. You should not rely upon forward-looking statements as predictions of future events. Although our management believes that the expectations reflected in our statements are reasonable, we cannot guarantee that the future results, performance, or events and circumstances described in the forward-looking statements will be achieved or occur. Recipients are cautioned not to place undue reliance on these forward-looking statements, which speak only as of the date such statements are made and should not be construed as statements of fact. We undertake no obligation to publicly update any forward-looking statements, whether as a result of new information, any future presentations, or otherwise, except as required by applicable law. Certain information contained in these materials and any statements made orally during any presentation of these materials that relate to the materials or are based on studies, publications, surveys and other data obtained from third-party sources and our own internal estimates and research. While we believe these third-party studies, publications, surveys and other data to be reliable as of the date of these materials, we have not independently verified, and make no representations as to the adequacy, fairness, accuracy or completeness of, any information obtained from third-party sources. In addition, no independent source has evaluated the reasonableness or accuracy of our internal estimates or research and no reliance should be made on any information or statements made in these materials relating to or based on such internal estimates and research.

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