



Immuneering Presents Genetic Data at AACR Annual Meeting Demonstrating Mechanism to Improve Durability and Survival, Supporting Use of Atebimetinib in First-Line Pancreatic Cancer and Beyond

April 20, 2026

Analysis of circulating tumor DNA from atebimetinib-treated patients shows acquired MAPK pathway alterations are rare, supporting observed durable first-line activity

Atebimetinib-treated tumors rarely acquire the genetic alterations most commonly associated with resistance to RAS inhibitors, providing molecular rationale to treat with atebimetinib early

NEW YORK, April 20, 2026 (GLOBE NEWSWIRE) -- Immuneering Corporation (Nasdaq: IMRX), a late-stage clinical oncology company focused on keeping cancer patients alive and helping them thrive, today announced the presentation of new genetic data at the 2026 American Association for Cancer Research (AACR) Annual Meeting taking place April 17-22, 2026 in San Diego, CA.

“Atebimetinib is designed to promote survival by three mechanisms: shrinking tumors durably, preserving body mass by counteracting muscle wasting, and maintaining performance status by maximizing tolerability,” said Ben Zeskind, Ph.D., Chief Executive Officer of Immuneering. “We believe these characteristics have the potential to both yield the best survival in the first-line, and to give patients the best chance of reaching and benefitting from second-line treatment. Today’s data at AACR add genetic rationale to support atebimetinib’s observed durable tumor shrinkage and its optimal use in the first-line setting by showing that the most common RAS-inhibitor resistance mechanisms are rarely seen in atebimetinib-treated patients.”

Inhibitors of RAS, RAF, or MEK often provide only temporary benefit due to pervasive resistance, as tumors acquire new mutations or mechanisms of escape within the MAPK pathway. Atebimetinib, a novel Deep Cyclic Inhibitor of MEK, is engineered to mitigate the selective pressure that typically drives these resistance mechanisms, with the goal of more durable anti-tumor activity. Immuneering presented circulating tumor DNA (ctDNA) data from 123 patients treated with atebimetinib, showing that acquired MAPK pathway alterations are rarely seen. These findings suggest that Deep Cyclic Inhibitors have the potential to overcome the limitations of conventional MAPK inhibition and provide a more sustained clinical benefit for patients, while potentially preserving sensitivity to subsequent treatments.

Key Findings from the AACR Presentation:

- **Rare MAPK pathway reactivation:** Across 86 patients treated with atebimetinib monotherapy and 37 patients treated in combination with chemotherapy, emergent and acquired mutations rarely converged on the RAS/MAPK pathway, in contrast to what is commonly observed with chronic RAS-targeted therapies.
- **Diffuse, non-convergent resistance patterns:** Emergent resistance following atebimetinib treatment utilized a variety of non-MAPK pathways, rather than converging on a single escape mechanism.
- **Limited early adaptive resistance:** ctDNA analysis showed minimal early molecular evolution during treatment, indicating that atebimetinib is not driving adaptive resistance and may impose less selective pressure than continuous pathway inhibition.
- Taken together, the data position atebimetinib as a differentiated MEK inhibitor with potential to drive deep and durable antitumor activity.

“Our AACR data further validate the scientific foundation of our platform, demonstrating that Deep Cyclic Inhibition can fundamentally alter how tumors evolve under therapy, unlocking opportunities to improve treatment durability,” said Brett Hall, Ph.D., Chief Scientific Officer of Immuneering. “Deep Cyclic Inhibition of MEK avoids the continuous selective pressure that typically drives tumors to become resistant to treatment via reactivation of the MAPK pathway. This, combined with atebimetinib’s tolerability profile, has the potential to improve depth and durability of response in a broad range of cancers, starting with first-line pancreatic cancer.”

Poster Presentation Details:

Title: Atebimetinib’s Deep Cyclic Inhibition of MEK Constrains MAPK-Axis Adaptive and Acquired Alterations in Patients with RAS-Mutant Tumors

Session Category: Experimental and Molecular Therapeutics

Session Title: Targeting Drug Resistance 2: RAS Signaling

Poster Number: 1873

Poster Board Number: 6

Session Date: April 20, 2026

Session Time: 9:00 AM – 12:00 PM ET

Location: Poster Section 19

The poster is available on the publications section of Immuneering's website at <https://immuneering.com/publications>.

Immuneering has guided to dosing the first patient in its pivotal Phase 3 MAPKeeper 301 trial of atebimetinib plus modified gemcitabine/nab-paclitaxel (mGnP) in patients with first-line metastatic pancreatic cancer in mid-2026. In the second half of the year, the company expects to dose the first patient in a Phase 2 trial of atebimetinib plus Libtayo® in patients with first-line RAS-mutant non-small cell lung cancer.

About Immuneering

Immuneering is a late-stage clinical oncology company focused on keeping cancer patients alive and helping them thrive. The Company is developing an entirely new category of cancer medicines, Deep Cyclic Inhibitors, designed to improve overall survival by three mechanisms: shrinking tumors durably with less resistance, preserving body mass by countering cachexia, and minimizing side effects to maximize performance status and combinability. Immuneering's lead product candidate, atebimetinib, is an oral, once-daily Deep Cyclic Inhibitor of MEK, designed to improve survival across many cancer indications, including MAPK pathway-driven tumors such as pancreatic cancer. The company expects to dose the first patient in mid-2026 in MAPKeeper 301, a globally randomized pivotal Phase 3 trial evaluating atebimetinib in combination with chemotherapy in first-line pancreatic cancer patients. The Company's development pipeline also includes additional combination opportunities and early-stage programs. For more information, please visit www.immuneering.com.

Forward-Looking Statements

This press release contains forward-looking statements, including within the meaning of the Private Securities Litigation Reform Act of 1995. All statements contained in this press release that do not relate to matters of historical fact should be considered forward-looking statements, including, without limitation, statements regarding: the treatment potential of atebimetinib, alone or in combination with other agents to treat cancer, including modified Gemcitabine/nab-paclitaxel (mGnP) in first-line pancreatic cancer and its potential to deliver overall survival with both durability and tolerability; the timing of dosing of additional studies, the ability of the three design mechanisms of atebimetinib to shrink tumors durably, improve overall survival and overcome the limitations of conventional MAPK inhibition, including to impose less selective pressure, and provide a more sustained clinical benefit for patients.

These forward-looking statements are based on management's current expectations. These statements are neither promises nor guarantees, but involve known and unknown risks, uncertainties and other important factors that may cause our actual results, performance or achievements to be materially different from any future results, performance or achievements expressed or implied by the forward-looking statements, including, but not limited to, the following: the risks inherent in oncology drug research and development, including target discovery, target validation, lead compound identification, and lead compound optimization; we have incurred significant losses, are not currently profitable and may never become profitable; our projected cash runway; our need for additional funding; our unproven approach to therapeutic intervention; our ability to address regulatory questions and the uncertainties relating to regulatory filings, reviews and approvals; the lengthy, expensive, and uncertain process of clinical drug development, including potential delays in or failure to obtain regulatory approvals; our reliance on third parties and collaborators to conduct our clinical trials, manufacture our product candidates, and develop and commercialize our product candidates, if approved; failure to compete successfully against other drug companies; protection of our proprietary technology and the confidentiality of our trade secrets; potential lawsuits for, or claims of, infringement of third-party intellectual property or challenges to the ownership of our intellectual property; our patents being found invalid or unenforceable; costs and resources of operating as a public company; and unfavorable or no analyst research or reports.

These and other important factors discussed under the caption "Risk Factors" in our Annual Report on Form 10-K for the period ended December 31, 2025, and our other reports filed with the U.S. Securities and Exchange Commission, could cause actual results to differ materially from those indicated by the forward-looking statements made in this press release. Any such forward-looking statements represent management's estimates as of the date of this press release. While we may elect to update such forward-looking statements at some point in the future, except as required by law, we disclaim any obligation to do so, even if subsequent events cause our views to change. These forward-looking statements should not be relied upon as representing our views as of any date subsequent to the date of this press release.

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