BioInvent Publication in JECCR: Tailored FcγR Blockade with BI-1206 and BI-1607 Enhances Cancer Antibody Therapies and Overcomes Resistance in Preclinical Models

- Preclinical proof-of-concept data support two distinct FcγR-blockade strategies to enhance cancer antibody therapies and overcome resistance
- BI-1206 boosts αPD-1 antibody activity by protecting effector CD8+ T cells from macrophage-mediated depletion, a mechanism identified as limiting αPD-1 efficacy
- Findings reinforce the mechanistic rationale for BI-1206's ongoing Phase 2a evaluation with pembrolizumab in first-line NSCLC and uveal melanoma; initial data expected second half of 2026
- Complementary Fc-engineered variant BI-1607 enhances αCTLA-4 antibody activity, including ipilimumab, by deepening intratumoral Treg depletion

Lund, Sweden – July 28, 2026 – BioInvent International AB ("BioInvent") (Nasdaq Stockholm: BINV), a leader in the discovery of novel immune#modulatory antibodies, today announce that preclinical proof#of#concept data supporting the mechanistic rationale for BI-1206 and BI-1607 in combination with immune checkpoint blockade have been published in the peer-reviewed Journal of Experimental & Clinical Cancer Research (JECCR).

The publication, titled "Tailored FcγR Blockade Enhances Immune Checkpoint Therapy and Overcomes Resistance," identifies tailored FcγR blockade as a strategy to overcome resistance to PD-1-directed checkpoint therapy. BI-1206, an anti-FcγRIIB antibody with a wild-type Fc domain, was shown to enhance the activity of the approved αPD-1 antibodies nivolumab and pembrolizumab by protecting effector CD8+ T cells from macrophage-mediated depletion. These findings provide the mechanistic foundation for BioInvent's ongoing Phase 2a evaluation of BI-1206 in combination with pembrolizumab in first-line NSCLC and uveal melanoma, with initial data expected in the second half of 2026. In the same study, structurally distinct anti-FcγRIIB antibody variants, BI-1607 and its murine surrogate, engineered with an Fc-silenced Fc domain, were shown to enhance αCTLA-4 antibodies, including ipilimumab, through selective FcγRIIB blockade. The BI-1607 program is currently paused as BioInvent focuses resources on advancing its lead programs BI-1206 and BI-1808.

"This work resolves a longstanding question in the field: whether and how FcγRs affect immune checkpoint blockade efficacy," said Björn Frendéus, Chief Scientific Officer of BioInvent and corresponding author of the study. "The answer depends on which immune cells express the checkpoint target. When a checkpoint target sits on suppressive regulatory T cells, as with CTLA-4, FcγR engagement helps antibodies deplete them, an effect BioInvent's Fc-silenced BI-1607 was designed to amplify. When the target sits on the effector CD8+ T cells that deliver

anti-tumor activity, as with PD-1, FcγR engagement causes those cells to be cleared instead. Blocking all FcγRs with an Fc-competent antibody like BI-1206 protects those CD8+ T cells and preserves αPD-1 efficacy. These findings provide the mechanistic foundation for BI-1206, our lead FcγRIIB-blocking antibody, and directly support the ongoing Phase 2a evaluation of BI-1206 in combination with pembrolizumab in first-line NSCLC and uveal melanoma."

"Despite the transformative impact of checkpoint inhibitors, most patients still fail to benefit long-term from these therapies," said Mark S. Cragg, Professor of Experimental Cancer Biology, Antibody and Vaccine Group, School of Cancer Sciences, University of Southampton and co-senior author of the study. "The mechanistic insights described in this paper give us a clear path to designing next-generation antibody combinations that address the underlying biology limiting current immunotherapy responses. This has been another highly productive collaboration between the Antibody and Vaccine Group at Southampton and BioInvent, and we look forward to seeing these principles translate into meaningful clinical benefit."

Publication overview: Mechanistic basis for enhancing PD-1 and CTLA-4 checkpoint therapy
Immune checkpoint inhibitors have transformed cancer treatment, yet most patients either do not respond or develop resistance during therapy. Understanding and overcoming that resistance is one of the field's most pressing challenges. Further limiting patient benefit, CTLA-4 checkpoint therapy is often dose-limited by toxicity.

Fc gamma receptors (FcγRs) regulate therapeutic antibody activity by engaging immune effector cells. Prior work established that different checkpoint antibodies have opposing FcγR requirements: αPD-1 antibodies can be undermined by FcγR-dependent depletion of the very effector CD8+ T cells they are designed to reactivate, whereas αCTLA-4 antibodies rely on FcγR engagement to deplete tumor-infiltrating regulatory T cells. The JECCR study reconciles these opposing requirements and identifies tailored FcγR blockade with BI-1206 as a strategy to overcome αPD-1 resistance and with BI-1607 to enhance αCTLA-4 efficacy in a tumor-selective manner, potentially improving tolerability.

About the Study

The study was conducted by researchers at BioInvent International and the University of Southampton's Antibody and Vaccine Group, School of Cancer Sciences. Stephen A. Beers and Mark S. Cragg (University of Southampton) and Ingrid Teige and Björn Frendéus (BioInvent) share senior authorship; Frendéus is corresponding author. The processed and raw single-cell RNA sequencing data have been deposited in the NCBI Gene Expression Omnibus under accession number GSE335831.

About BI-1206

FcγRIIB is an inhibitory Fc gamma receptor often upregulated in the tumor microenvironment. Alongside activating Fc gamma receptors, FcγRIIB expressed on immune effector cells can limit the efficacy of PD-1-directed checkpoint therapy. BI-1206 is a human anti-FcγRIIB monoclonal antibody with a wild#type Fc domain, designed to block FcγRs on tumor-associated macrophages, thereby preventing macrophage-mediated depletion of PD-1-coated CD8+ T cells and preserving αPD-1 antitumor activity. A Phase 2a study of BI-1206 in combination with MSD's

(Merck & Co., Inc., Rahway, NJ, USA) anti-PD-1 therapy KEYTRUDA® (pembrolizumab) is ongoing in treatment-naïve patients with advanced or metastatic non-small cell lung cancer (NSCLC) and uveal melanoma, with initial data expected in the second half of 2026 (NCT04219254). BI-1206 is also being evaluated in a separate Phase 2a triple combination with rituximab and Calquence® (acalabrutinib) in relapsed/refractory non-Hodgkin's lymphoma, where the antibody addresses a distinct resistance mechanism relevant to CD20-directed therapy (NCT03571568).

About BioInvent
BioInvent International AB (Nasdaq Stockholm: BINV) is a clinical-stage biotech company that discovers and develops novel and first-in-class immuno-modulatory antibodies for cancer therapy, with drug candidates in ongoing clinical programs in Phase 1/2 trials for the treatment of hematological cancer and solid tumors. The Company's validated, proprietary F.I.R.S.T™ technology platform identifies both targets and the antibodies that bind to them, generating many promising new immune-modulatory candidates to fuel the Company's own clinical development pipeline and providing licensing and partnering opportunities.

The Company generates revenues from research collaborations and license agreements with multiple top-tier pharmaceutical companies, as well as from producing antibodies for third parties in the Company's fully integrated manufacturing unit. More information is available on the Company's website.

For further information, please contact:
Cecilia Hofvander, VP Investor Relations
Phone: +46 (0)46 286 85 50
Email: cecilia.hofvander@bioinvent.com

BioInvent International AB (publ)
Co. Reg. No.: 556537-7263
Visiting address: Ideongatan 1
Mailing address: 223 70 LUND
Phone: +46 (0)46 286 85 50
www.bioinvent.com

The press release contains statements about the future, consisting of subjective assumptions and forecasts for future scenarios. Predictions for the future only apply as the date they are made and are, by their very nature, in the same way as research and development work in the biotech segment, associated with risk and uncertainty. With this in mind, the actual outcome may deviate significantly from the scenarios described in this press release.

Attachments

BioInvent Publication in JECCR: Tailored FcγR Blockade with BI-1206 and BI-1607 Enhances Cancer Antibody Therapies and Overcomes Resistance in Preclinical Models