# Highlight Recap | Elpiscience Showcases Three Innovative Research Studies at SITC 2025, Demonstrating Original Innovation on the Global Academic Stage

From November 5 to 9, the 40th Annual Meeting of the Society for Immunotherapy of Cancer (SITC) was grandly held at National Harbor, Maryland, USA. As one of the world's largest and most influential academic congresses in cancer immunotherapy, Elpiscience was selected to present for the fifth consecutive year, showcasing three of its latest research achievements through poster presentations. Key highlights included:

- Bispecific Myeloid Engager (BiME) antibodies that harness the DC–CD8⁺ T cell axis to generate durable antitumor immunity;
- ES009, a differentiated and potentially best-in-class LILRB2 inhibitor, demonstrating favorable safety, tolerability, PK/PD, and preliminary antitumor activity in a first-in-human Phase 1 study;
- ES038, a novel NK cell engager (NKCE) targeting LILRB4 via dual engagement of NKG2A and NKG2C, showing strong therapeutic potential for AML.

The three scientific advances from Elpiscience drew significant attention at the conference, demonstrating strong recognition from the international scientific community and further highlighting the company's innovative strengths and leadership in immuno-oncology. Moving forward, Elpiscience will continue to focus on first-in-class innovation in tumor immunology, accelerate clinical translation, and bring innovative therapies from China to patients worldwide earlier.

Key Research Highlights Presented at SITC 2025:

- **Bispecific Myeloid Engager (BiME) Antibodies Harness the DC–CD8⁺ T Cell Axis to Drive Durable Antitumor Immunity**

Abstract No.: 1168

**Research Overview:**

ES004-B5 is a pan-allelic anti-human SIRP α antibody developed by Elpiscience, which potently blocks the SIRP α -CD47 interaction and enhances macrophage phagocytosis when combined with anti-TAA(tumor-associated antigens) IgG1 therapies. Structural analysis indicates that ES004-B5 can potentially block both trans (CD47-SIRP α ) and cis (SIRP α -CD18) inhibitory pathways in macrophages. To further unlock its therapeutic value, the research team generated a series of bispecific myeloid engagers (BiMEs) targeting both SIRPα and TAAs. These molecules significantly enhanced macrophage and dendritic-cell phagocytosis, promoted CD8⁺ T cell activation, and demonstrated potent antitumor efficacy in multiple in vivo models. These BiMEs

markedly enhanced macrophage/DC phagocytic activity and promoted $CD8^+$ T cell activation, leading to robust anti-tumor effects in multiple murine tumor models. Further research demonstrates that BiMEs primarily act through the dendritic cell (DC)-$CD8^+$ T cell axis, resulting in potent and durable anti-tumor immune memory. This study further uncovers the antitumor potential of myeloid cells, providing a new strategy for myeloid-targeted therapy.

**Key Findings:**

- Elpiscience's anti-SIRP α and BiME molecules recognize a unique epitope overlapping both CD47- and CD18- binding sites on SIRP α
- BiME activates macrophages and DCs both *in vitro* and *in vivo*
- BiME promotes CD8+ T enrichment in TME *in vivo*
- BiME drives tumor inhibition primarily via the DC-$CD8^+$ T axis
- BiME induces robust and durable antitumor immune memory

- **A first-in-human study evaluating the safety, tolerability, pharmacokinetics, pharmacodynamics, and preliminary antitumor activity of the LILRB2 inhibitor ES009 in patients with advanced solid tumors**

Abstract No.: 596

**Research Overview:**

ES009 is a differentiated mAb targeting LILRB2 with best-in-class potential. By targeting and binding to LILRB2, ES009 reprograms immunosuppressive M2 macrophages into pro-inflammatory M1 macrophages, alleviates M2-mediated T cell suppression, and reshapes the immunosuppressive tumor microenvironment into one more conducive to anti-tumor immunity.

This report presents the results of a first-in-human Phase I clinical study on ES009 (Clinical Trial Number: NCT06007482). The trial is an open-label, multicenter, phase 1 study conducted in Australia to evaluate the safety, tolerability, PK, PD, and preliminary antitumor activity of ES009 in patients with advanced solid tumors who had received and progressed on or were intolerant to standard therapies. Twelve patients—including colorectal cancer, cholangiocarcinoma, NSCLC, ovarian cancer, and submandibular gland cancer — were enrolled. ES009 was administered intravenously every three weeks at doses ranging from 30 to 1600 mg. The DLT assessment period was 21 days.

**Key Findings:**

- ES009 demonstrated favorable safety and tolerability in patients with advanced solid tumors, with no DLTs observed and the maximum tolerated dose (MTD) not reached.
-At dose levels ≥ 300 mg, ES009 exhibited desirable pharmacokinetic and pharmacodynamic properties.
- Among 11 evaluable patients, 8 patients achieved stable disease as their best overall response, resulting in a disease control rate (DCR) of 72.7%. The median progression-free survival was 2.8 months
- As a monotherapy, ES009 showed preliminary antitumor activity with good safety and tolerability, supporting further clinical development

- ES038, a novel NKCE Molecule Targeting LILRB4 in AML via Dual Engagement of NKG2A and NKG2C

Abstract No.: 435

**Research Overview:**

Natural killer cell engagers (NKCEs) offer a versatile platform to enhance NK cell-mediated r cytotoxicity. However, conventional NKCEs often fall short in fully activating NK cell due to inhibitory signals such as NKG2A. In addition, AML, particularly in relapsed or refractory cases, remains a significant clinical challenge. LILRB4 has emerged as one of the highly promising new therapeutic targets.

ES038, a novel NKCE that targets LILRB4 on AML cells while simultaneously blocking inhibitory (NKG2A) and stimulating activating (NKG2C) receptors on NK cells, this dual-targeting strategy enhances NK cell activation and improves their therapeutic potential.

**Key Findings:**

- ES038 is a NK cell engager targeting LILRB4 via dual engagement of NKG2A and NKG2C
- Dual engagement of NKG2A and 2C exhibit superior activity than NKG2A alone
- Dual engagement of NKG2A and 2C could modulate NKG2A+NKG2C+ cell population with stronger IFN $\gamma$ % and CD107a% expression
- NK cells from AML patients treated with ES038 can kill LILRB4-expressing cells
- By coordinately modulating NKG2A and NKG2C pathways, ES038 represents a novel dual-targeting immunotherapy to break through the therapeutic bottleneck of AML

**About Elpiscience**

Elpiscience is a clinical-stage biopharmaceutical company dedicated to the development of innovative immunotherapies for oncology and autoimmune diseases. By advancing breakthrough biologics and leveraging global strategic partnerships, Elpiscience has built a differentiated pipeline to deliver transformative treatment solutions for patients worldwide.

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