Lead Discovery: Dual-Mechanism Protein Therapy for Inflammatory Control and 'Cold-to-Hot' Tumor Conversion

Korea Research Institute of Bioscience and Biotechnology(KRIBB)

ONCOLOGY **Lead**

Product Type	rProtein
Indication	CRC(colorectal Cancer)/ Solid tumor(including CAC)
Target	****
MoA(Mechanism of Action)	Reprogramming pro-tumor M2 macrophages into anti-tumor M1 macrophages and promoting IL-12 expression to convert the tumor microenvironment (TME) from 'cold' to 'hot', thereby maximizing the anti-cancer activity of T cells ($CD8^+$) and NK cells.
Competitiveness	• Mechanism: Triggers the anti-tumor immune system by inducing the activation of anti-tumor M1 macrophages. • Efficacy: Converts "cold tumors" into "hot tumors" through a multimodal mechanism that simultaneously stimulates both innate and adaptive immunity, delivering a differentiated, powerful anti-tumor effect. • Safety: Highly biocompatible, microbiota-derived proteint with low systemic toxicity, ideal for long-term and combination therapies.
Development Stage	Lead
Route of Administration	• **Current (*In Vivo*)**: Intraperitoneal (IP) injection • **Target (Clinical)**: Subcutaneous (SC) injection