Press release

For media and investors only

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GSK enters agreement to acquire CMG1A46 from Chimagen Biosciences to expand immunology pipeline

- CMG1A46 is an investigational T cell-engager with potential to deeply deplete uncontrolled B cells present in autoimmune diseases such as lupus
- Unmet need remains for patients with lupus, especially those who are refractory to current standards of care
- Agreement reinforces GSK's portfolio in the treatment of lupus and underlying drivers of autoimmune disease

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GSK plc (LSE/NYSE: GSK) and Chimagen Biosciences (Chimagen), a privately held biotechnology company, today announced an agreement for GSK to acquire CMG1A46, a clinical-stage dual CD19 and CD20-targeted T cell-engager (TCE), from Chimagen for $300 million upfront. GSK plans to develop and commercialise CMG1A46 with a focus on B cell-driven autoimmune diseases, such as systemic lupus erythematosus (SLE) and lupus nephritis (LN), with potential to expand into related autoimmune diseases.

For over a decade, GSK has been a pioneer in the treatment of lupus. This agreement underscores the importance of novel therapeutic approaches to address the heterogeneity of lupus manifestations and the continued burden, particularly in patients who suffer from severe disease and are refractory to current standard of care.[1]

Tony Wood, Chief Scientific Officer, GSK, said: "Through our work in systemic lupus erythematosus and lupus nephritis, we increasingly understand the underlying drivers of B cell-driven diseases. As a novel therapeutic option directed at deep B cell depletion, CMG1A46 offers exciting potential which we are pleased to take forward to address unmet need in lupus and related autoimmune conditions."

CD20 is an established target in the treatment of autoimmune diseases and there is growing clinical evidence that CD19 shows promise as a differentiated therapeutic approach given its presence on more B cell types.[2] In preclinical studies, CMG1A46, designed to target both CD19 and CD20, has shown rapid, deep B cell depletion both in the bloodstream and in tissues which could lead to more durable responses in patients.

Zhenhao Zhou, Chief Executive Officer, Chimagen, said: "We are excited by the potential of CMG1A46 to improve the lives of patients suffering from autoimmune conditions and grateful to have GSK accelerate that vision. This agreement provides further validation of our proprietary T cell-engager platform, and we are eager to continue our mission of developing novel multi-specific antibody therapeutics."

CMG1A46 is currently in phase I clinical trials in leukaemia and lymphoma in both the US and China. GSK aims to begin a phase I trial in lupus in 2025.

Terms of the agreement

Under the terms of this agreement, GSK will pay $300 million upfront to acquire full global rights to CMG1A46. In addition, Chimagen will be eligible to receive success-based development and commercial milestone payments for CMG1A46 totalling $550 million.

This agreement is subject to customary conditions, including applicable regulatory agency clearances under the Hart-Scott-Rodino Act in the US.

About CMG1A46

CMG1A46, a dual CD19 and CD20-targeted T cell-engager (TCE), is an IgG-like molecule with high affinity for CD19 and CD20 positive B cells and low affinity for CD3, which could lower toxicities typically associated with TCEs.

About Chimagen Biosciences
Chimagen Biosciences is a clinical-stage biotechnology company focused on the discovery and development of novel multi-specific T cell-engagers and NK cell-engagers generated by proprietary antibody engineering platforms. Its mission is to advance breakthrough therapies that improve the lives of patients with cancer and autoimmune diseases. For more information, please visit www.chimagen.com.

About GSK
GSK is a global biopharma company with a purpose to unite science, technology, and talent to get ahead of disease together. Find out more at gsk.com.

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References
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2 Schett, G et al. CD19 CAR T-Cell Therapy in Autoimmune Disease - A Case Series with Follow-up. N Engl J Med. 2024 Feb 22;390 (8):687-700.