Press release
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Lundbeck to present new patient data on neuroendocrine and neuroimmunology programs at ENDO 2026

- Upcoming presentations at ENDO 2026 highlight Lundbeck's Focused Innovator strategy and continued expansion into rare neuroendocrine diseases with high unmet medical needs
- The scientific program showcases Lundbeck's investigational neurohormonal and neuroimmunological targeted therapeutic candidates, asedebart and Lu AG22515, respectively
- Preliminary Phase II data for asedebart, an investigational anti-adrenocorticotropic hormone (ACTH) monoclonal antibody in Cushing's disease (CD), demonstrate Lundbeck's biomarker-supported approach to evaluating novel therapeutic mechanisms in rare endocrine disorders
- Preclinical characterization of the CD40L blocker Lu AG22515 will be shared as well as data from a patient study in thyroid eye disease (TED,) reflecting exploratory studies of CD40L modulation in autoimmune disease biology, with potential relevance to neuroinflammation implicated in several neurological and endocrine diseases

Valby, Denmark, Wednesday 10 June 2026 – H. Lundbeck A/S (Lundbeck) today announced that new data will be presented at the 2026 Endocrine Society's Annual Meeting (ENDO), taking place June 13–16 in Chicago, Illinois, U.S. Lundbeck will present preliminary Phase II data for asedebart (Lu AG13909) in CD, reflecting Lundbeck's expansion into neuroendocrine diseases. In addition, preclinical and Phase Ib clinical exploratory findings on Lu AG22515 in patients with TED will be shared, providing insights into the broader therapeutic potential of CD40L pathway modulation across inflammatory and immunological disorders.

"Lundbeck's presence at ENDO 2026 reflects how we have expanded upon our neuroscience heritage in recent years. This involves pursuing biological drug targets within hormonal and immunological signaling pathways that offer the potential to deliver highly differentiated therapeutics for neurological and neuroendocrinology indications with high medical unmet need," said Johan Luthman, EVP and Head of Research & Development at Lundbeck. "Through this approach, we have made significant progress across our rare disease programs, enabling decisive, biomarker-supported patient studies that facilitate early development decisions, as exemplified by our ENDO 2026 scientific program."

Asedebart data provide insight into ACTH neutralization in Cushing's disease
Among the highlights are preliminary Phase II data for asedebart, an investigational anti-adrenocorticotropic hormone (ACTH) monoclonal antibody, being evaluated in adults with CD.

CD is a rare neuroendocrine disorder typically caused by an ACTH-secreting pituitary adenoma, leading to chronic excess cortisol production and substantial physical and neuropsychiatric burden.[1,2] While surgery is the standard first-line treatment, many patients experience

persistent or recurrent disease despite available pharmacologic options, and significant unmet need remains.

The Phase II data being presented include impact on urinary free cortisol (UFC) levels following individualized dose titration of asedebart, alongside safety and tolerability assessments, supporting further understanding of direct ACTH neutralization in CD.

The CD study builds on earlier Phase I findings in classic congenital adrenal hyperplasia (CAH) (ClinicalTrials.gov: NCT05669950), which showed pharmacodynamic effects on key adrenal steroid biomarkers. The CD data presented at ENDO add to Lundbeck's evaluation of targeting ACTH-driven endocrine conditions with links to brain function — using early clinical and pharmacodynamic evidence to assess therapeutic potential in areas of significant unmet need.

Asedebart has received Orphan Drug Designation (ODD) for CAH in the European Union and the United States as well as ODD in Japan for the treatment of patients with CD and CAH.

Lu AG22515 data provide insight into CD40L pathway biology
At ENDO, Lundbeck will also present preclinical characterization findings for the investigational CD40L blocker Lu AG22515. CD40L is a key immune signaling molecule heavily implicated in a wide range of immune disorders, neurology and potentially endocrine conditions.[3] The presentation will describe the inhibitory effect and PK/PD profile of Lu AG22515, including effects on membrane-bound and soluble CD40L, B-cell activation and differentiation, proinflammatory cytokine production and in vivo antibody responses.

Additionally, clinical findings will be presented from an exploratory Phase Ib open label study on AG22515 in TED patients. TED is an autoimmune disorder that can cause proptosis, diplopia, pain, disfigurement and, in severe cases, visual impairment or vision loss.[4] The presentation will cover three areas: pharmacodynamic assessments designed to evaluate CD40L pathway engagement, safety and tolerability, and preliminary clinical efficacy in TED, including effects on proptosis and other measures of disease activity. The data further enhance the understanding of CD40L pathway modulation in TED and other CD40L-mediated immune disorders.

Asedebart and Lu AG22515 are investigational drugs not approved for marketing by any regulatory authority worldwide, and the efficacy and safety of both molecules have not been established.

Details of Lundbeck presentations at ENDO 2026

Therapeutic Area	Presentation content	Presentation Type	Reference
Cushing's disease Asedebart Lu AG13909	A Phase II, Open-label, Dose-titration Trial to Investigate the Safety, Tolerability, Pharmacokinetics, and Efficacy of the Novel Anti-ACTH Antibody Asedebart in Adults with Cushing's Disease	Oral presentation	Sun 14 June 14:45-15:00 CT Room W183BC
Thyroid eye disease (TED) Lu AG22515	Results From a Phase 1b Trial Evaluating CD40-Ligand Blocker Lu AG22515 in Patients with Moderate-to-Severe Thyroid Eye Disease	Oral presentation	Mon 15 June 14:15-14:30 CT W184ABC
Thyroid eye disease (TED) Lu AG22515	Preclinical Pharmacokinetic and Pharmacodynamic Profile of Lu AG22515, a CD40-Ligand Blocker in Development for Thyroid Eye Disease	Rapid-fire presentation and poster	Sun 14 June 09:00- 16:00 CT ENDOExpo; Poster floor

About Cushing's disease

Cushing's disease is a rare neuro-endocrine disorder caused by a pituitary adenoma that secretes excess ACTH, leading to chronic overproduction of cortisol.[1] The condition is associated with significant morbidity and increased mortality, and patients may experience a wide range of physical and neuropsychiatric symptoms.[2] First-line treatment is surgical removal of the tumor; however, not all patients are eligible, achieve sustained remission, or benefit fully from current available treatment options, highlighting an ongoing unmet need for effective and well-tolerated therapies.

About asedebart (Lu AG13909)

Asedebart is a humanized anti-ACTH monoclonal antibody designed specifically to recognize ACTH with high affinity. It blocks the binding of ACTH to the melanocortin 2 receptor in the adrenal glands and thereby inhibits the neurohormonal signalling of ACTH. This inhibition causes a decreased secretion of glucocorticoids, mineralocorticoids and androgens from the adrenal glands. [5,6] ACTH plays a key role in the biosynthesis of adrenal steroids[7] and is therefore considered a promising therapeutic target in conditions characterized by elevated ACTH levels.[6]

About thyroid eye disease (TED)

Thyroid eye disease (TED) is a rare autoimmune condition associated with thyroid dysfunction, characterized by inflammation and expansion of retro-orbital tissues. Clinical manifestations include proptosis, double-vision, pain, swelling, and disfigurement, and in severe cases may

lead to visual impairment or loss of vision. TED can have a substantial impact on daily functioning, quality of life and psychological wellbeing.[4]

Current treatments are not optimal for all patients and may be associated with significant adverse effects, highlighting the need for additional therapeutic approaches that target underlying autoimmune disease biology.[4]

About Lu AG22515

Lu AG22515 is an investigational CD40L blocker being evaluated in CD40L mediated autoimmune diseases. Lu AG22515 is a recombinant fusion protein that binds CD40 ligand (CD40L) and human serum albumin (HSA) to extend half-life. By blocking the interaction between CD40L and the CD40 receptor, Lu AG22515 is designed to modulate a key immune co-stimulatory pathway involved in B-cell activation, antibody responses and inflammatory signaling.

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About H. Lundbeck A/S

Lundbeck is a biopharmaceutical company focusing exclusively on brain health. With more than 70 years of experience in neuroscience, we are committed to improving the lives of people with neurological and psychiatric diseases.

Brain disorders affect a large part of the world's population, and the effects are felt throughout society. With the rapidly improving understanding of the biology of the brain, we hold ourselves accountable for advancing brain health by curiously exploring new opportunities for treatments.

As a focused innovator, we strive for our research and development programs to tackle some of the most complex neurological challenges. We develop transformative medicines targeting people for whom there are few or no treatments available, expanding into neuro-specialty and neuro-rare from our strong legacy within psychiatry and neurology.

We are committed to fighting stigma and we act to improve health equity. We strive to create long term value for our shareholders by making a positive contribution to patients, their families, and society as a whole.

Lundbeck has more than 5,000 employees in more than 20 countries and our products are available in more than 80 countries. For additional information, we encourage you to visit our corporate site www.lundbeck.com and connect with us via LinkedIn.

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