Press release
Valby, 2 December, 2025

Lundbeck to present comprehensive new bexicaserin dataset in patients with rare childhood-onset epilepsies, at American Epilepsy Society (AES) Annual Meeting

- New data to be presented at the 2025 AES Annual Meeting indicate sustained reductions in seizure frequency as early as two weeks after treatment initiation with bexicaserin[1]
- Bexicaserin is an investigational compound in development for the treatment of seizures associated with a broad range of Developmental and Epileptic Encephalopathies (DEEs)
- DEEs are a group of severe childhood-onset epilepsies with the majority of DEEs resistant to conventional anti-seizure medications[2]
- Eight presentations at AES highlight Lundbeck's dedication to improving outcomes for people with rare epilepsies

Valby, Denmark, 2 December 2025 – H. Lundbeck A/S (Lundbeck) today announced that new pipeline data regarding bexicaserin (LP352), a novel investigational drug for the treatment of seizures associated with Developmental and Epileptic Encephalopathies (DEEs), will be presented at the 2025 American Epilepsy Society Congress in Atlanta, USA (Dec 5 – 9). The comprehensive dataset includes a recent post-hoc analysis from Phase 1b/2a PACIFIC trial and Open-Label Extension (OLE) indicating patients with DEEs experienced an early response to bexicaserin which was sustained over the long-term.[1]

DEEs are a group of childhood onset epilepsies characterized by drug-resistant seizures and developmental and intellectual disabilities.[2] In DEEs, excessive electrical activity in the brain results in seizures and cognitive impairments, leaving patients heavily dependent on caregivers.[2,3] Currently, only a few types of DEEs have authorized antiseizure medications (ASMs), leaving most DEE patients vulnerable and without an approved treatment.[3]

"Most DEEs are resistant to conventional anti-seizure medicines, leaving patients with very few options," said Johan Luthman, EVP and Head of Research & Development at Lundbeck. "The data for bexicaserin are compelling—indicating early seizure reductions that are sustained across diverse DEE syndromes. These results underscore its potential as a first-in-class treatment across this devastating range of epilepsies."

The new analyses indicate that there were fewer countable motor seizures as early as two weeks following bexicaserin treatment initiation and that these reductions were maintained over a full year of open label observation. This observed response was consistent across DEE subtype.[1]

During the PACIFIC trial, bexicaserin was generally well tolerated with no significant safety concerns. Three participants in the bexicaserin group reported a serious adverse event (ankle fracture x 2, constipation, and increased seizures). Throughout the titration period, 16.3% of bexicaserin-treated patients discontinued due to an adverse event, while across the

maintenance period, 4.7% of bexicaserin-treated participants discontinued due to an adverse event. No new safety concerns were observed during the open label extension period.

During the AES congress, Lundbeck will also showcase two-year follow-up results from the PACIFIC trial, further reinforcing bexicaserin's durable and consistent effect across a broad range of DEEs, along with the drug's mode of action, pharmacokinetics, and negligible drug-drug interaction.[4-10]

The eight presentations at AES 2025 demonstrate the breadth of Lundbeck's R&D program and ongoing dedication to reducing the burden of these devastating rare childhood onset epilepsies.

Bexicaserin is an investigational compound that is not approved for marketing by any regulatory authority worldwide. The efficacy and safety of bexicaserin have not been established.

Lundbeck's scientific presentations at AES:

Bexicaserin for the Treatment of Seizures in Developmental and Epileptic Encephalopathies: Interim Analysis of an Expanded Access Program for Participants on Treatment for up to 2 Years[4]	Poster presentation: Caitlin Sylvia	Saturday, Dec 6
Analyses of Bexicaserin for the Treatment of Seizures in Developmental and Epileptic Encephalopathies: Response Over Time and Responder Rates in the Phase 1b/2a PACIFIC Trial Open-Label Extension[1]	Poster presentation: David G. Vossler, MD	Saturday, Dec 6
Patterns of Antiseizure Medication Utilization in Participants with Developmental and Epileptic Encephalopathies and Responses to Bexicaserin Treatment in the Phase 1b/2a PACIFIC Trial[5]	Poster presentation: Pavel Klein, MD	Saturday, Dec 6
Bexicaserin Has Negligible Drug–Drug Interaction Potential When Co-Administered With Anti-Seizure Medications That Are Inhibitors or Inducers of CYP/UGT Enzymes[6]	Poster presentation: Nuggehally R. Srinivas	Sunday, Dec 7
Bexicaserin Selectively Activates 5-HT_{2C} Receptors to Modulate Activity in the Thalamocortical Circuit Relevant for Absence Seizures[7]	Poster presentation: Jesper Frank Bastlund	Sunday, Dec 7
An Ethnobridging Study Evaluating the Safety, Tolerability, and Pharmacokinetics of Bexicaserin[8]	Poster presentation: Rosa Chan	Monday, Dec 8
Lack of Potential for Bexicaserin to Prolong QT Interval at Supratherapeutic Doses[9]	Poster presentation: Jonathan Williams	Monday, Dec 8

Effects of Bexicaserin on Neural Circuits in the DBA/1 Mouse Model of Sudden Unexpected Death in Epilepsy (SUDEP)[10]	Poster presentation: Sheryl Vermudez	Monday, Dec 8

About DEEs

Developmental and Epileptic Encephalopathies (DEEs) are a group of rare neurodevelopmental disorders that typically manifest in early childhood.[2] These heterogeneous and severe epilepsy syndromes are characterized by refractory seizures, frequent epileptiform activity on electroencephalogram (EEG), and developmental stagnation or regression.[2] According to the International League Against Epilepsy (ILAE), DEEs currently encompass more than 10 syndromes, including Early Infantile DEE (EIDEE), Infantile Epileptic Spams Syndrome (IESS), Dravet Syndrome, and Lennox-Gastaut Syndrome with various etiologies among those mainly genetic (e.g., CDKL5, STXBP1, KCNT1, SCN2A).

About bexicaserin

Bexicaserin (LP352) is an oral, centrally acting 5-hydroxytryptamine 2C (5-HT2C) receptor superagonist with no engagement of the 5-HT2B and 5-HT2A receptor subtypes, potentially minimizing the risk of cardiovascular toxicity.[11] The most common TEAEs associated with bexicaserin the PACIFIC trial were somnolence, decreased appetite, constipation, diarrhea, lethargy, tremor, urinary tract infection, fatigue, pyrexia, agitation, and hypertension.

Bexicaserin is being evaluated in a global Phase 3 clinical program (the DEEp Program). The FDA has granted Breakthrough Therapy designation for bexicaserin for the treatment of seizures associated with DEEs for patients two years of age and older. Bexicaserin has also recently been granted Breakthrough Therapy Designation in China for the treatment of seizures associated with DEEs.

About the PACIFIC trial

The PACIFIC trial was a Phase 1b/2a randomized, double-blind, placebo-controlled clinical trial to assess the safety, tolerability, efficacy, and pharmacokinetics of bexicaserin in 52 participants between the ages of 12 and 65 years old with any type of DEE (Dravet syndrome, Lennox-Gastaut syndrome and DEE other) at 34 sites across the United States and Australia. Participants who had ≥4 countable motor seizures during the 28-day baseline period, while on a stable regimen of 1 to 4 concomitant antiseizure medications were included.[12] Three participants in the bexicaserin group reported a serious adverse event (ankle fracture x 2, constipation, and increased seizures). During the titration period, 16.3% of bexicaserin-treated patients discontinued due to an adverse event, while during the maintenance period 4.7% of bexicaserin-treated participants discontinued due to an adverse event.

Following a 28-d baseline period, study participants initiated a dose titration over a 15-day period and subsequently continued on the highest tolerated dose throughout the maintenance period of 60 days. Eligible participants were given the opportunity to enroll in a 52-week open-label extension (OLE) if they completed the 75-d RCT treatment period (titration and

maintenance). The OLE included patients with Dravet syndrome (n=3), Lennox-Gastaut syndrome (n=20) and DEE Other (n=18), who completed the PACIFIC trial (n=41).

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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 approximately 5,700 employees in more than 50 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.

References:

1. Vossler DG, et al. Poster Presentation: American Epilepsy Congress 2025
2. Scheffer IE, et al. Epilepsia. 2025;00:1-10
3. Samanta D, et al. *Epilepsy Behav*. 2025;170:110472
4. Sylvia C, et al. Poster Presentation: American Epilepsy Congress 2025
5. Klein P, et al. Poster Presentation: American Epilepsy Congress 2025
6. Srinivas NR, et al. Poster Presentation: American Epilepsy Congress 2025
7. Vermudez S, et al. Poster Presentation: American Epilepsy Congress 2025
8. Chan R, et al. Poster Presentation: American Epilepsy Congress 2025
9. Williams J, et al. Poster Presentation: American Epilepsy Congress 2025
10. Vermudez S, Tolpiko T, et al. Poster Presentation: American Epilepsy Congress 2025
11. Ren A, et al. J Medicinal Chem. 2025;68(11):10599-10618
12. Palmer EE, et al. Neurotherapeutics. 2021;18(3):1432–1444