Dual Clinical Efficacy of NLX-112 in Parkinson's Disease Presented at Scientific Meetings

Ground-breaking Non-Dopaminergic Drug Combining Both Anti-Dyskinetic and Anti-Parkinsonian Properties

NJ, USA, August 22, 2023 /EINPresswire.com/ -- Neurolixis, Inc., a private, clinical-stage company developing innovative drug therapies for the treatment of neurologic disorders with high unmet medical needs, is disclosing the positive results of its clinical Phase 2A trial with NLX-112 (befiradol) in Parkinson's disease at international scientific meetings. The positive findings were presented in July at the World Parkinson's Congress in Barcelona, Spain as a 'Hot Topics' oral presentation (viewable here) and will also be presented at the forthcoming Movement Disorders Society meeting in Copenhagen, Denmark, on □□□□□□□□□□, □□□□□□ □□. □□□□□□ #□□□□.

> The positive effects on both LID and parkinsonism could make NLX-112 a highly innovative, dual efficacy therapy, thanks to its novel mechanism, distinct to that of current medications for Parkinson's."
>
> *Adrian Newman-Tancredi, PhD, DSc (CEO, Neurolixis)*

NLX-112 is a first-in-class, non-dopaminergic drug candidate which was investigated in a randomized, double-blind, placebo-controlled Phase 2A trial in patients presenting troubling levodopa-induced dyskinesia (LID). The study was jointly supported by The Michael J Fox Foundation and Parkinson's UK. NLX-112 met the primary endpoint of safety and tolerability, and also met its secondary endpoint of significantly reducing LID. Notably, NLX-112 also elicited robust anti-parkinsonian efficacy, significantly reducing motor impairment in study participants. Taken together, the data point to a groundbreaking dual-acting activity of NLX-112, combining both anti-LID and anti-parkinsonian properties.

□□. □□□□□□ □□□□□□-□□□□□□□□, CEO of Neurolixis, commented: "We're thrilled about the results from this trial. The positive effects seen on both dyskinesia and parkinsonism could make NLX-112 a highly innovative, non-dopaminergic dual efficacy therapy, thanks to its first-in-class neurochemical properties, distinct from those of current medications for Parkinson's."

The dual efficacy profile of NLX-112 has not been observed for other anti-parkinsonian drugs and may arise from NLX-112's original mechanism of action. Indeed, whereas levodopa and most other existing antiparkinsonian drugs target the dopamine neurotransmitter system in the brain, NLX-112 targets the serotonin (5-hydroxytryptamine, 5-HT) system and specifically 5-HT1A receptors. NLX-112 is exceptionally selective for 5-HT1A receptors and fully activates them, a profile which differentiates it from previous serotonergic drugs and likely underlie the dual-acting clinical benefits seen in the present study.

□□□□□ □□□□□□□□□'□ □□□□□□□ □□□ □□□□□□□□-□□□□□□□ □□□□□□□□□□

Parkinson's disease is the second most prevailing neurodegenerative disease after Alzheimer disease, and causes movement impairment, including shaking, stiffness, and difficulty with balance and coordination. About 1% of people over the age of 60 are affected and about 10 million people globally, and the incidence of Parkinson's is rising with the aging of the population. The mainstay drug for treatment of Parkinson's is levodopa, which limits motor dysfunction in the early stages of the disease. However, after prolonged treatment it can cause involuntary movements called dyskinesia. Up to 80 percent of people with Parkinson's will experience LID after ten years of taking levodopa. The main medication available to manage dyskinesia is amantadine, which can have challenging side-effects and does not work for everyone.

□□□□□ □□□ □□□-□□□ □□□□ □□□□□

The study (ClinicalTrials.gov ID: NCT05148884) was conducted at 5 centers in Sweden and enrolled 27 subjects with troubling LID. Subjects were maintained on a stable dose of levodopa and other anti-parkinsonian medications. Study drug dosing was up-titrated over 4 weeks to a maximum of 2 mg/day (1 mg b.i.d.); dosing was kept stable for 2 weeks (until day 42) and then down-titrated over 2 weeks. Subjects received a levodopa challenge (150 % of regular dose) at baseline and at the end of the up-titration and steady-state periods. Dyskinesia and parkinsonism were assessed using the Unified Dyskinesia rating Scale (UDysRS) and the Unified Parkinson's Disease Rating Scale (UPDRS), respectively.

22 patients (15 on NLX-112, 7 on PBO) completed the 8-week treatment according to the protocol. Safety and tolerability were similar between the NLX-112 and placebo groups. In motor assessment measures at day 42, NLX-112 significantly reduced UDysRS total score (p=0.0016) and parts 3+4 objective evaluation scores (p=0.0005), whereas placebo group changes were not significant. Moreover, NLX-112 also significantly reduced UPDRS total score (p=0.0021) and part 3 motor impairment score (p=0.0072), whereas placebo group changes were not significant.

□□□□□ □□□□□□□□□□

Neurolixis is a biopharmaceutical company focused on the discovery and early development of novel drugs for the treatment of central nervous system disorders with unmet medical needs and sizeable market opportunity, including movement disorders, autism, pain and depression. The Neurolixis drug candidates are serotonin 5 HT1A receptor 'biased agonists' which enables them to better target specific brain regions controlling CNS disorders. NLX-112 has previously shown favorable safety and tolerance in over 600 subjects during Ph1 and Ph2 trials for other

indications. NLX-112 is also under study as a potential treatment for other motor disorders such as spinocerebellar ataxia and essential tremor. Other drug candidates in the Neurolixis pipeline are NLX-101, which is in clinical Phase 1 for treatment of rare autism spectrum disorders (Rett syndrome and Fragile X syndrome), and NLX-204, a preclinical candidate for treatment of pain and mood deficits.

For more information, contact Pim Cerutti, Liberi Group Life Science Business Development & Strategy; pimcerutti@LiberiGroup.com

PIM CERUTTI
LIBERI GROUP
email us here
Visit us on social media:
LinkedIn
