Rodman &Renshaw®

December 20, 2024
Healthcare: Biotechnology
Scilex Holding Company (SCLX)
COMPANY NOTE

SCLX: Introducing KDS2010, a Novel Oral Drug in Phase 2 Trials for Alzheimer's and Obesity

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Scilex Bio, a joint venture primarily owned by Scilex Holding Company (SCLX; Buy) in collaboration with IPMC Company, announced progress on KDS2010 (tisolagiline). This novel reversible and selective monoamine oxidase B (MAO-B) inhibitor, licensed globally through NeuroBiogen's exclusive rights, is being evaluated for its potential in treating neurodegenerative diseases such as Alzheimer's disease (AD). In addition to its MAO-B inhibitory activity, KDS2010 also modulates astrocytic GABA inhibition. We are maintaining our Buy rating and 12-month price target of $13/share.

GABA, the brain's primary inhibitory neurotransmitter, plays a crucial role in maintaining the balance between neuronal excitation and inhibition. Astrocytes, a type of glial cell, are integral to regulating GABA activity. In neurodegenerative diseases such as Alzheimer's, disruptions in GABAergic signaling and impaired astrocytic regulation of neurotransmitter systems contribute to cognitive decline and neuroinflammation. KDS2010 works by inhibiting astrocytic GABA signaling, which helps reduce neuroinflammation and restore the balance between excitatory and inhibitory signals in the brain, potentially improving cognitive function in AD.

Reactive astrocytes contribute to AD pathology by producing hydrogen peroxide (H_2O_2). KDS2010, a selective and reversible MAO-B inhibitor, blocks MAO-B-mediated aberrant GABA and H_2O_2 production in reactive astrocytes. This mechanism reduces neuronal inhibition, neuroinflammation, and neurodegeneration while promoting neuroregeneration.

In the dentate gyrus of Alzheimer's mouse models, astrocytic GABA released onto presynaptic GABA receptors lowers the spike probability of granule cells. By suppressing GABA production in reactive astrocytes, KDS2010 restores spike probability, synaptic plasticity, and cognitive function in mice. Postmortem analyses of Alzheimer's patients reveal significantly elevated astrocytic GABA and MAO-B levels, underscoring the therapeutic potential of targeting astrocytic GABA synthesis to address memory impairment.

While short-term treatment with irreversible MAO-B inhibitors has shown cognitive benefits in AD patients, long-term results have been disappointing due to compensatory upregulation of the GABA-synthesizing enzyme diamine oxidase (DAO). KDS2010 overcomes these limitations by selectively and reversibly inhibiting MAO-B. Prolonged KDS2010 treatment significantly reduces astrocytic GABA levels, alleviates astrogliosis, enhances synaptic transmission, and improves learning and memory in AD mouse models.

KDS2010 has demonstrated favorable safety, tolerability, and pharmacokinetics for once-daily dosing in Phase 1 clinical trials involving 88 healthy young and elderly adults across Korean and Western populations. A randomized, double-blind, placebo-controlled Phase 2 trial to assess the safety and efficacy of KDS2010 in Alzheimer's patients with mild cognitive impairment and mild dementia is currently enrolling 114 participants in South Korea, with a US cohort expected to begin in 2025. The clinical trial to evaluate the safety and efficacy of KDS2010 in 75 overweight or obese patients currently enrolling in South Korea with a cohort in US to be added in 2025.

Details of the preclinical and clinical data are discussed below.

Stock Data

Rating	Buy
Price Target	$13.00
Exchange	NASDAQ
Price	0.44
52-Week High	2.63
52-Week Low	0.44
Cash (M)	$7
Market Cap (M)	88
Shares Outstanding (M)	192
3 Month Avg Volume	1,740,984

Estimates

	2022A	2023A	2024E
			(Curr.)
Revenue (M) | $ | Year end: December			
Q1	-	10.6A	10.9A
Q2	-	12.6A	16.4A
Q3	-	10.1A	20.0E
Q4	-	13.5A	25.0E
FY	38.0A	46.7A	72.3E
EPS | $ | Year end: December			
Q1	-	(0.22)A	(0.24)A
Q2	-	(0.19)A	(0.31)A
Q3	-	(0.63)A	(0.07)E
Q4	-	(0.25)A	0.01E
FY	(0.17)A	(0.88)A	(0.59)E

One Year Performance Chart

MAO-B in Astrocytes and its Role in GABA Release as a Therapeutic Target for Alzheimer's disease (AD)

Amyloid-beta (Aβ) is a core pathological hallmark of AD, characterized by the abnormal accumulation of Aβ plaques in the brain. These plaques are formed when amyloid precursor protein (APP) is improperly cleaved, leading to the aggregation of insoluble Aβ peptides. The buildup of Aβ plaques triggers a cascade of events, including neuroinflammation, oxidative stress, and synaptic dysfunction, which contribute to neuronal loss and cognitive decline. Aβ deposition also promotes the activation of reactive astrocytes and microglia, further exacerbating brain damage and impairing learning and memory functions, which are central symptoms of AD.

Reactive astrocytes cluster around Aβ plaques in AD brains (Step a), which leads to an increase in putrescine concentration (Step b) in these reactive astrocytes (step c). MAO-B acts on putrescine to produce GABA (step d), a key inhibitory neurotransmitter. This GABA is subsequently released from reactive astrocytes through the Best1 channel (step e). The released GABA then binds to extrasynaptic GABA receptors (GABA-R) on postsynaptic neurons (step f), leading to inhibitory signaling (Figure 1).

This cascade of events disrupts normal synaptic activity and contributes to learning and memory impairments observed in AD. MAO-B plays a critical role of in GABA synthesis and release, suggesting that targeting MAO-B may provide a therapeutic strategy to reduce GABA release from reactive astrocytes and alleviate memory deficits in AD. By inhibiting MAO-B, it may be possible to restore excitatory-inhibitory balance in affected brain regions and mitigate the detrimental effects of GABA overproduction.

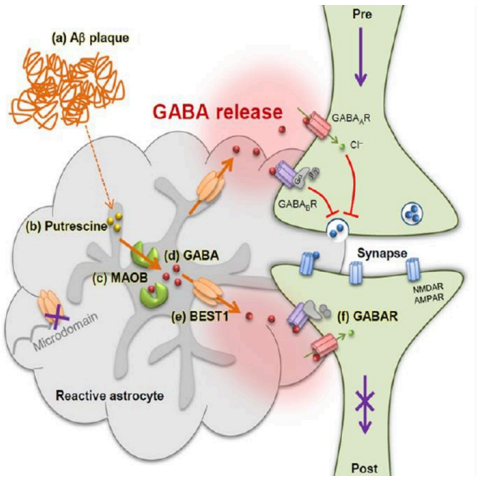
Limitations and Deficiencies of Existing Monoamine Oxidase-B (MAO-B) Inhibitors

Selegiline and rasagiline, two commonly used irreversible MAO-B inhibitors, have demonstrated short-term efficacy but failed to produce satisfactory long-term outcomes. Clinical studies report that although selegiline, as the most widely used MAO-B inhibitor, can improve cognitive deficits in AD patients, its long-term therapeutic effects have been disappointing. This failure to rescue cognitive function over extended treatment underscores a critical gap in the treatment of neurodegenerative diseases.

Specific unmet needs for newly developed MAO-B inhibitors are focusing on three key areas:

- *Selectivity versus MAO-A.* Existing inhibitors such as selegiline and rasagiline show limited selectivity for MAO-B over MAO-A, with selegiline having a selectivity ratio of ~150 and rasagiline ~50. This lack of specificity can contribute to off-target effects
- *Reversible inhibition.* Unlike irreversible inhibitors, reversible inhibitors offer potential advantages, including better control over inhibition and improved safety profiles.
- *Off-target selectivity in the CNS.* Existing reversible inhibitors, such as safinamide, have been shown to impact other neural pathways, leading to undesirable side effects such as inhibition of sodium channels, calcium channels, glutamate release, and dopamine reuptake. These off-target effects can negatively impact the nervous system and reduce therapeutic efficacy.

Figure 1: Mechanism of Astrocyte-GABA Interaction in AD



Source: NeuroBiogen Corporate Presentation, December 2024

KDS2010 as a Selective Reversible MAO-B Inhibitor

KDS2010, also known as tisolagiline, is a highly selective and reversible inhibitor of monoamine oxidase B (MAO-B) (Figure 2A). MAO-B activity is upregulated in Alzheimer's disease, contributing to increased ROS production and neuroinflammation. KDS2010's ability to modulate MAO-B activity and oxidative stress could offer neuroprotective benefits. By selectively inhibiting MAO-B, KDS2010 increases dopamine levels in the brain while reducing oxidative stress.

KDS2010's selectivity and reversibility have been demonstrated in previous studies.

Selectivity. KDS2010 exhibits an IC_{50} of 7.6nM for MAO-B and a 12,500-fold selectivity over MAO-A, indicating its strong preference for inhibiting MAO-B (Figure 2B & C).

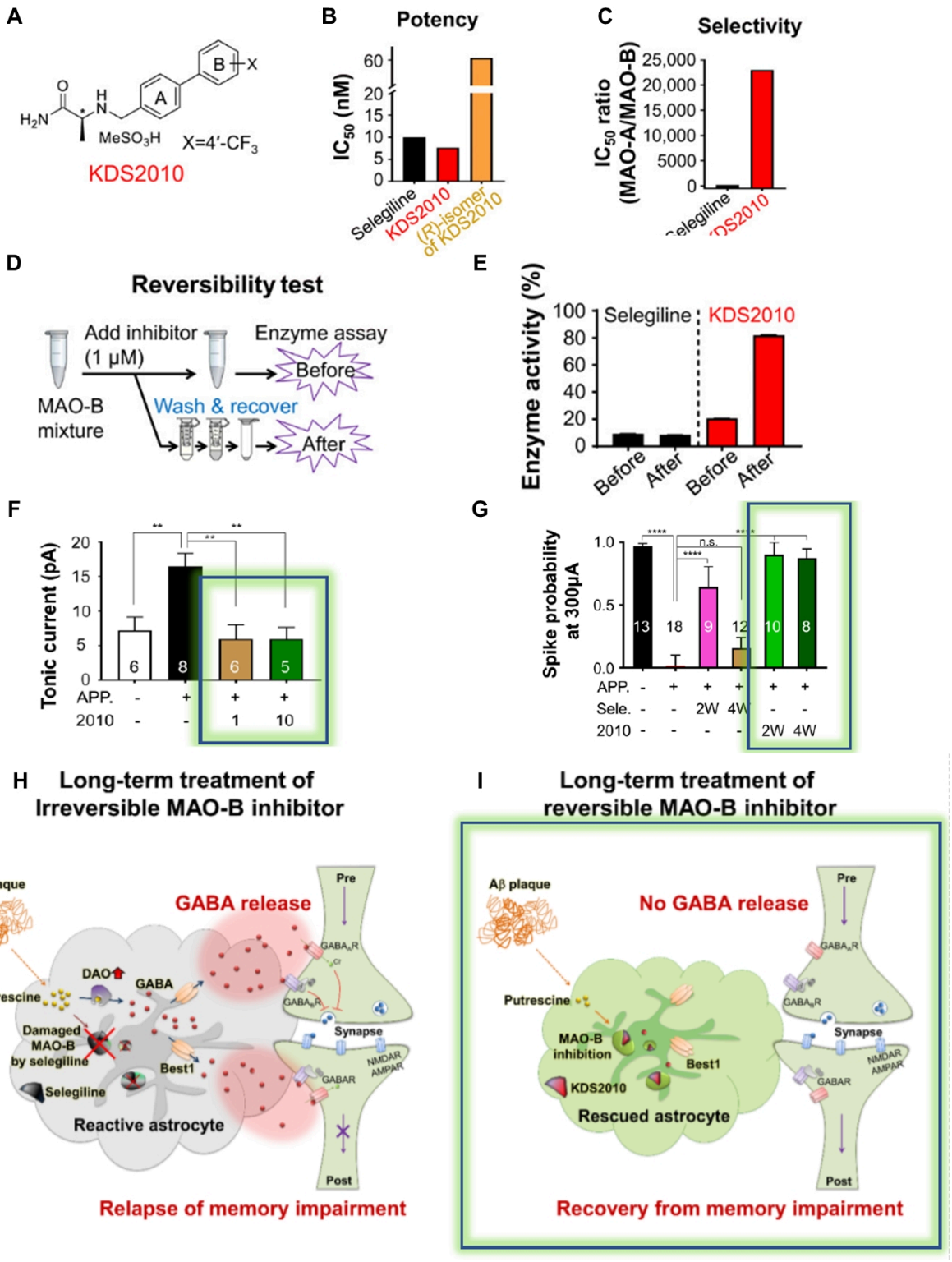
Reversibility. KDS2010's reversible inhibition of MAO-B has been confirmed through studies showing its ability to bind temporarily to the enzyme, allowing for better control over enzyme activity and minimizing long-term adaptation risks. KDS2010 demonstrated over 80% recovery of enzyme activity, whereas selegiline showed minimal recovery (Figure 2D & E).

Preclinical Proof-of-Concept of KDS2010 in Treating AD

Tonic GABA inhibition and reduced spike probability in APP/PS1 mouse models are associated with Alzheimer's pathology. KDS2010 has demonstrated therapeutic potential as both short-term and long-term treatments of AD in preclinical models. KDS2010 significantly decreases tonic GABA current in APP/PS1 mouse models (Figure 2F). KDS2010 treatment in APP/PS1 mice also restores spike probability significantly, particularly after four weeks of treatment (Figure 2G).

While long-term selegiline treatment relies on diamine oxidase (DAO) as a compensatory GABA-synthesizing enzyme (Figure 2H), KDS2010 effectively prevented the aberrant tonic GABA current in APP/PS1 mice during prolonged treatment. Unlike selegiline, KDS2010 does not trigger compensatory mechanisms, significantly reducing elevated astrocytic GABA levels and astrogliosis (Figure 2I). Additionally, KDS2010 enhances synaptic transmission, suppresses abnormal GABA production and astrogliosis, restores spike probability, and improves learning and memory deficits in APP/PS1 mice.

Figure 2: Preclinical Proof-of-Concept of KDS2010 in Treating AD



Source: Park et al., 2019 & NeuroBiogen Corporate Presentation, December 2024

Preclinical Proof-of-Concept of KDS2010 in Treating Obesity

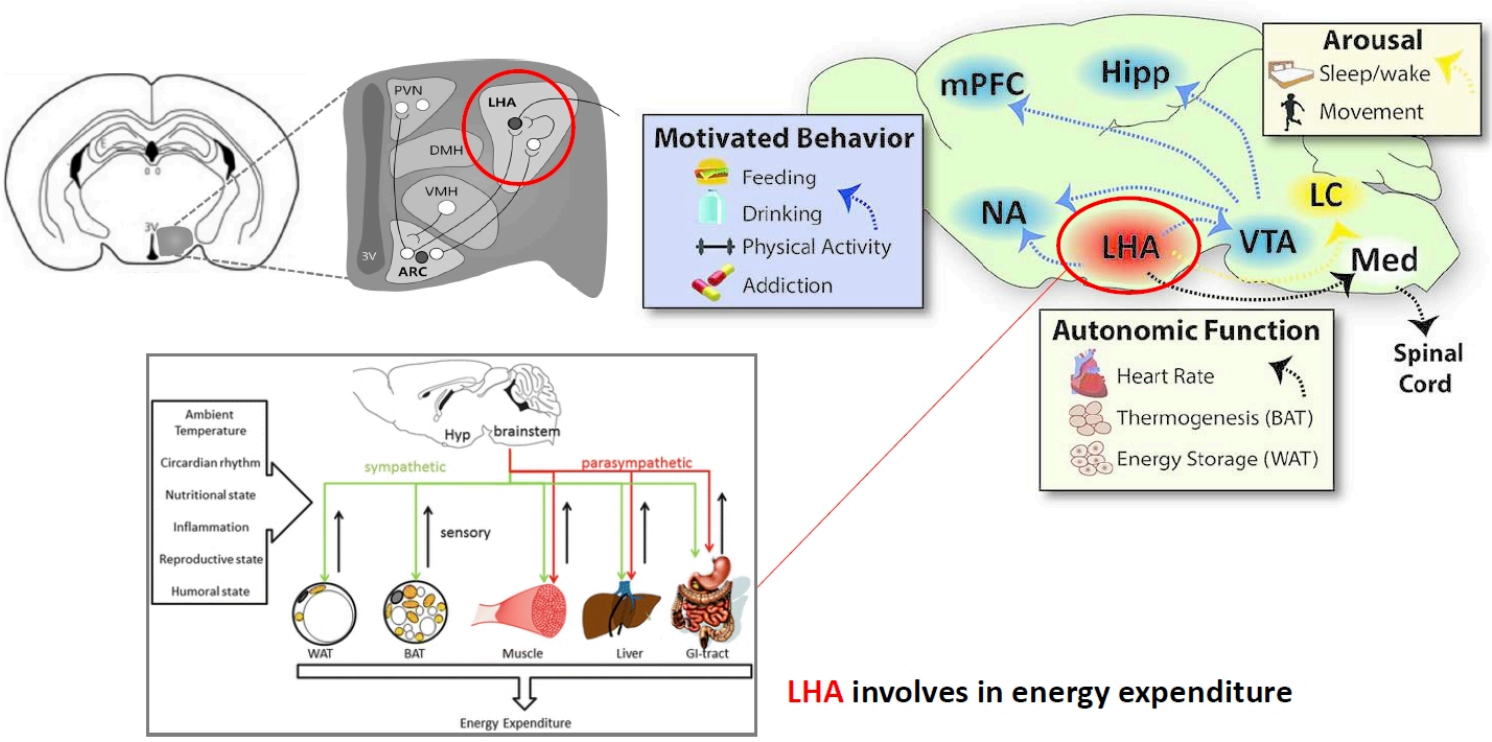
The lateral hypothalamic area (LHA) plays a critical role in regulating food intake and energy balance (Figure 3). GABRA5-positive neurons in the LHA is a distinct subpopulation of GABAergic neurons that polysynaptically project to both brown and white adipose tissues. In male diet-induced obesity (DIO) mouse models, inhibition of $GABRA5_{LHA}$ neurons decreases fat thermogenesis and promotes weight gain, whereas silencing the GABRA5 gene in the LHA mitigates weight gain, suggesting that $GABRA5_{LHA}$ neuron activity suppresses fat accumulation offers a potential therapeutic strategy for obesity.

KDS2010 is an effective intervention for reducing obesity and normalizing altered neural activity in the LHA. In the preclinical study, six-week-old C57BL/6J mice were divided into four groups: a chow diet with or without KDS2010 and a high-fat diet (HFD) with or without KDS2010 (Figure 4A). Treatment with KDS2010 began at 15 weeks, and analyses were conducted at weeks 22-23 (Figure 4B). Mice on the HFD developed obesity, as evidenced by increased body weight and fat mass, whereas those on the HFD supplemented with KDS2010 exhibited significantly reduced body weight and fat mass without a reduction in cumulative calorie intake (Figure 4C, D, E & F). This suggests that the anti-obesity effects of KDS2010 are not driven by decreased food consumption but rather by direct metabolic or neural effects. Importantly, lean mass was preserved in all groups, highlighting the specificity of KDS2010's actions in reducing fat mass (Figure 4G & H).

At the neurophysiological level, the study revealed that HFD-fed mice exhibited elevated tonic GABAergic inhibition in the LHA, a brain region critical for regulating energy homeostasis (Figure 5A). KDS2010 significantly attenuated this heightened tonic inhibition in both chow- and HFD-fed mice, effectively restoring neural activity to normal levels (Figure 5B & E). Electrophysiological recordings showed reduced GABAergic tonic currents and normalized neuronal firing rates in HFD mice treated with KDS2010 (Figure 5F & J). These effects were confirmed through additional experimental validations, including viral injections into the LHA (Figure 5S to U).

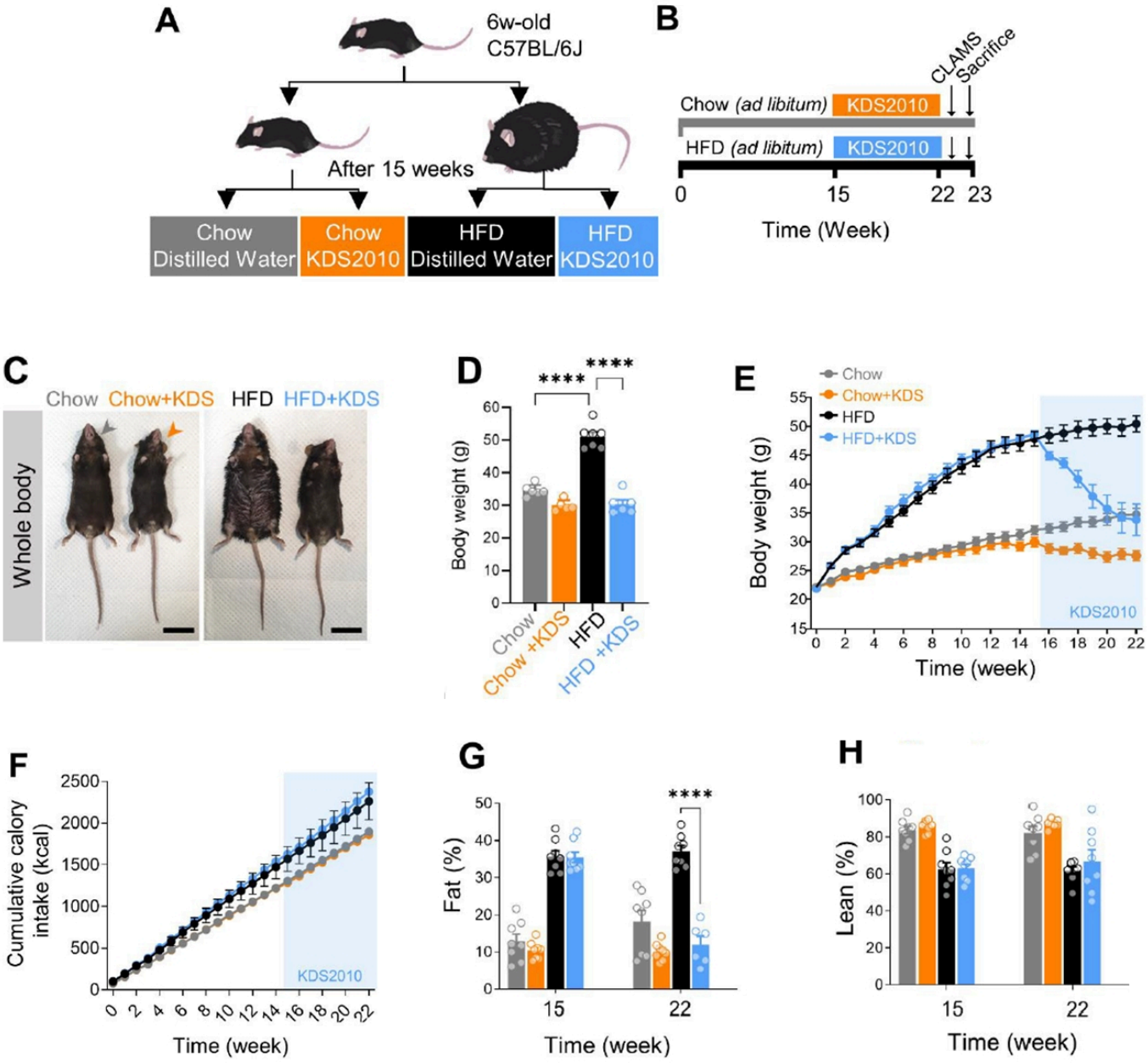
Together, the findings indicate that KDS2010 exerts dual effects: reducing obesity by targeting fat mass and restoring neuronal function by alleviating excessive tonic inhibition in the LHA. This combination of systemic and neural impacts underscores the potential of KDS2010 as a promising therapeutic for obesity and its associated neurophysiological dysfunctions.

Figure 3: Lateral Hypothalamic Area (LHA) Involves in Energy Expenditure



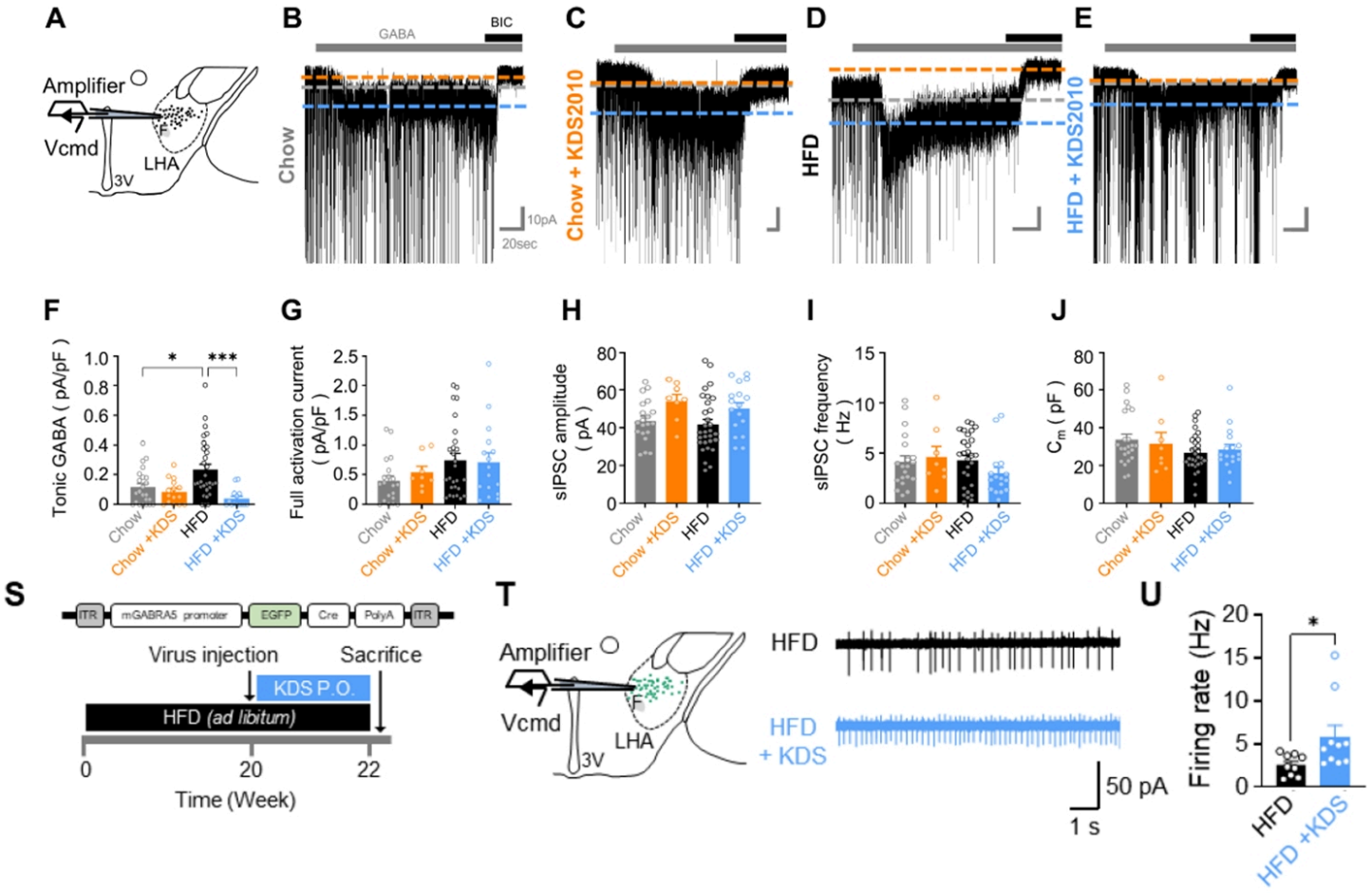
Source: Park et al., 2019 & NeuroBiogen Corporate Presentation, December 2024

Figure 4: KDS2010 Effectively and Rapidly Reduces Obesity



Source: NeuroBiogen Corporate Presentation, December 2024

Figure 5: KDS2010 Attenuates the Elevated Tonic Inhibition in LHA

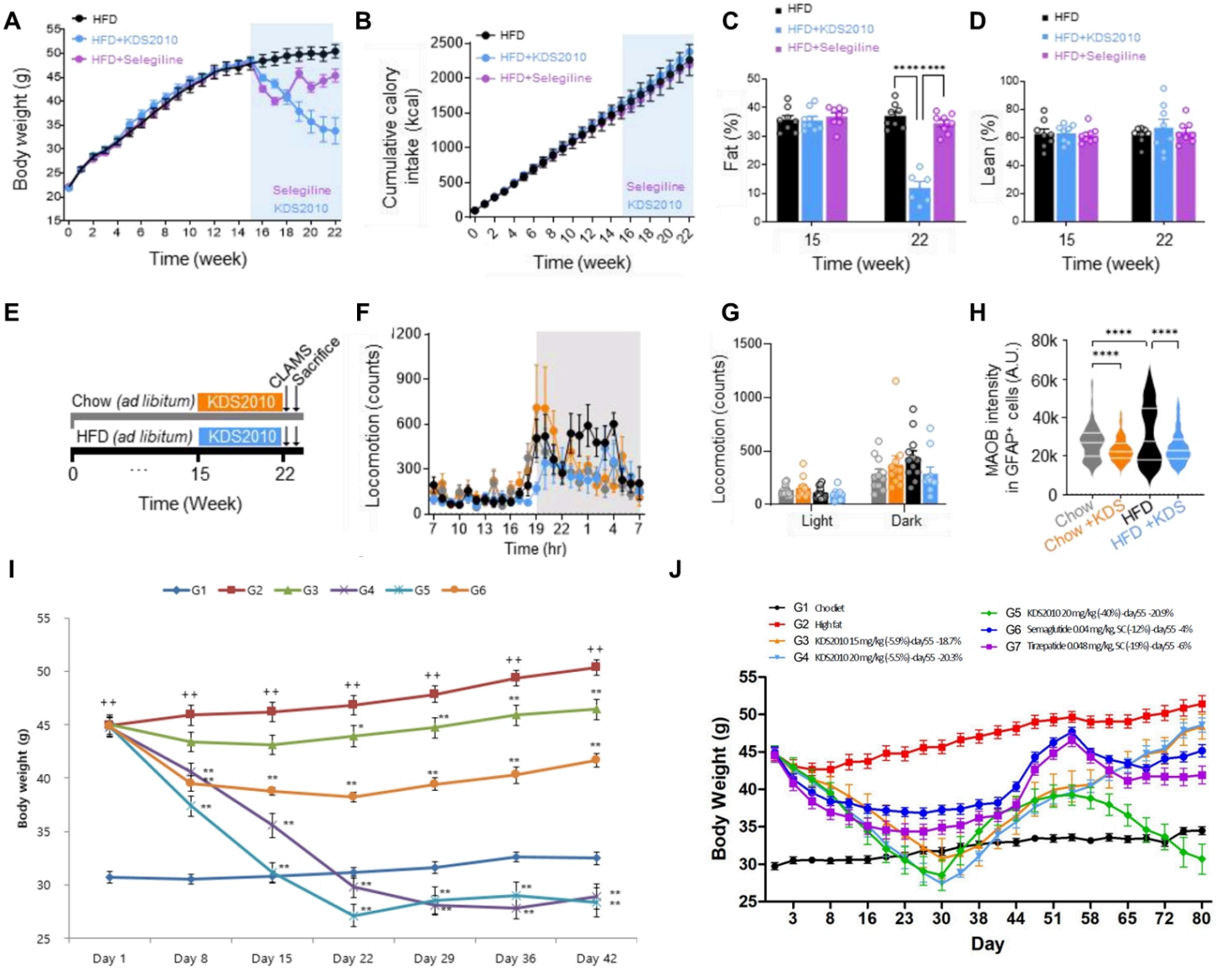


Source: NeuroBiogen Corporate Presentation, December 2024

Unlike Selegiline, another selective MAO-B inhibitor that only temporarily reduces fat, KDS2010 consistently reduced fat in high-fat diet-fed C57BL/6J mice after 15 days of treatment (Figure 6A-D). Importantly, KDS2010 treatment does not impair locomotion or physical activity levels, as demonstrated in open-field tests (Figure 6E-H). Additionally, KDS2010 showed significant obesity improvement following six weeks of repeated oral administration (Figure 6I). Notably, KDS2010 achieves substantial weight reduction comparable to that of a GLP-1 agonist (Figure 6J).

This slide emphasizes KDS2010's efficacy in reducing fat mass, improving obesity outcomes, and preserving normal locomotor function, with comparative effectiveness against existing treatments.

Figure 6: KDS2010 Reduced Fat without Affecting Locomotion



Source: NeuroBiogen Corporate Presentation, December 2024

KDS2010 Clinical Trial Phase 1

Study Design. A dose-escalation Phase 1 clinical trial was conducted to evaluate the safety, tolerability, pharmacokinetics (PK), pharmacodynamics (PD), and food effect on the bioavailability of KDS2010 following oral administration in healthy young and elderly male participants. The study was randomized, double-blind, placebo-controlled, and included both single and multiple dosing. The trial started on September 13, 2022, and finished on July 14, 2023.

The primary objectives were to assess the safety, tolerability, and PK of single and multiple oral doses of KDS2010 in healthy young and elderly participants and to evaluate the impact of food on the safety, tolerability, and PK of KDS2010. The secondary objectives were to investigate the PD effects of single and multiple oral doses of KDS2010 in the same population and to examine ethnic differences in safety, tolerability, and PK of KDS2010 in healthy young and elderly participants.

The Single Ascending Dose (SAD) study involved 48 healthy adult participants in a randomized, double-blind, placebo-controlled design (Figure 7A). The trial featured single-dose escalation, with a fixed-sequence, two-period crossover study conducted in Cohort 3 to evaluate the effect of food on the bioavailability of KDS2010 (Figure 7B). Adjustments to the dose escalation plan could lead to changes in the dose used for food effect evaluation.

The Multiple Ascending Dose (MAD) study involved 40 healthy adults, including 32 younger adults and 8 elderly participants, in a randomized, double-blind, placebo-controlled design. The trial featured multiple dose escalation with once-daily (q.d.) dosing over a 7-day repeat period (Figure 7C).

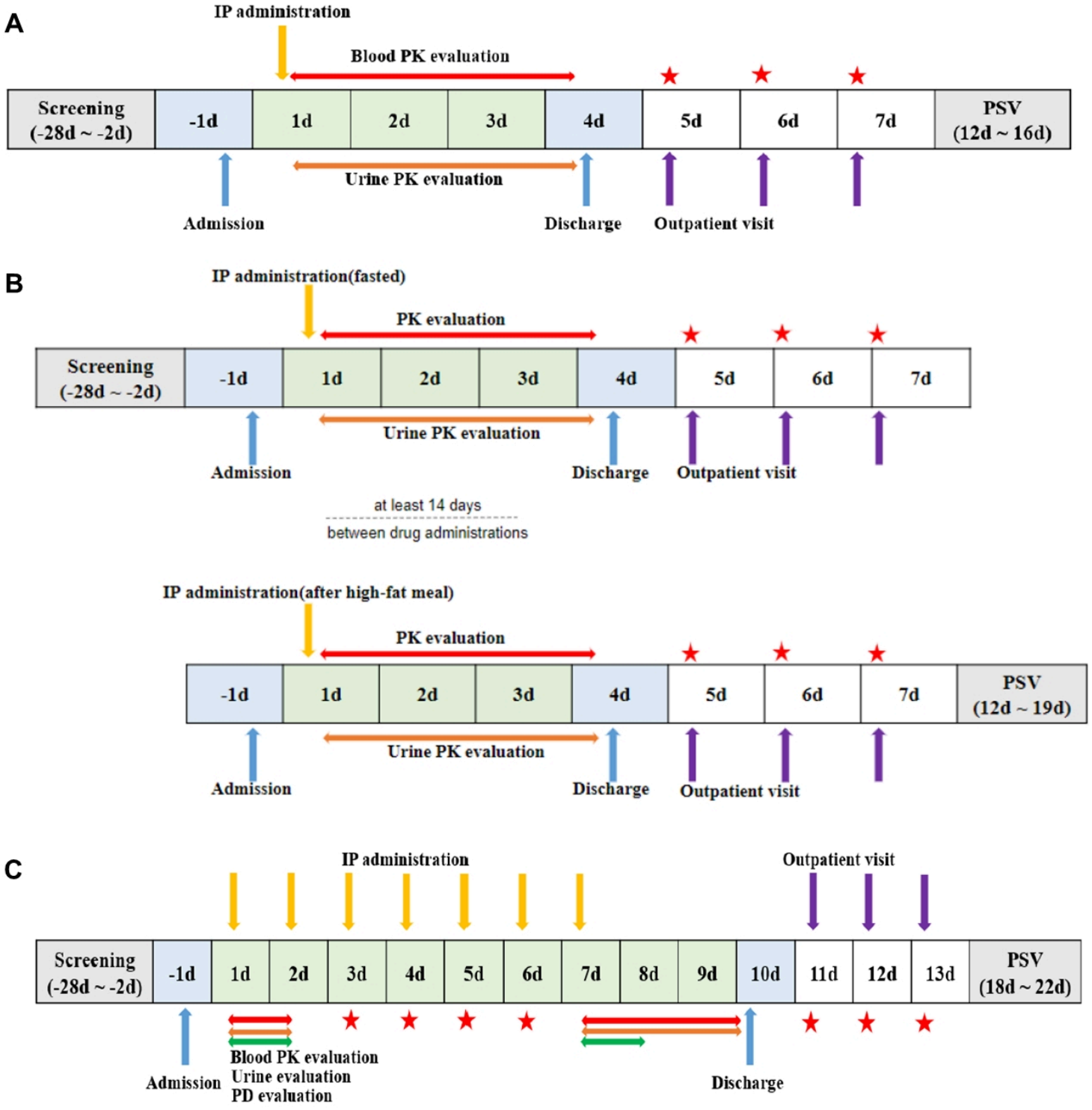
Safety. In the SAD study, no serious adverse events (SAEs) or suspected unexpected serious adverse reactions (SUSARs) occurred. There were 32 treatment-emergent adverse events (TEAEs) reported in 15 of 36 (41.7%) participants who received the study drug, with a relatively high number of TEAEs observed at the highest dose (960 mg). Among these, 19 adverse drug reactions (ADRs) were noted in 8 participants (22.2%), predominantly neuropsychiatric side effects such as drowsiness and dry eyes, all of which resolved completely.

In the MAD study, no SAEs or SUSARs occurred either. A total of 24 TEAEs were reported in ten of 30 (33.3%) participants receiving the study drug, with 11 TEAEs in younger adults (20.8%) and 13 in elderly participants (83.3%). ADRs were observed in 12 cases from seven participants (23.3%) receiving the study drug, including digestive symptoms like dyspepsia, with higher rates in the elderly group (66.7%). For the placebo group, 11 TEAEs occurred in five of ten (50.5%) participants, with five ADRs from three participants (30.0%). All ADRs were mild and resolved completely in all subjects.

PK / PD. In the SAD study, time-concentration profiles demonstrated that KDS2010 was absorbed without lag time following a single dose and exhibited distribution and clearance consistent with a one-compartment model (Figure 8A). A comparison of PK between Caucasian and Korean participants showed similar major PK parameters (Figure 8C). Regarding food effect, systemic exposure to KDS2010 was not influenced by food intake. The median T_{max} was three hours in the fasting state (range: three to eight hours) and six hours following a high-fat meal (range: two to eight hours) (Figure 8E).

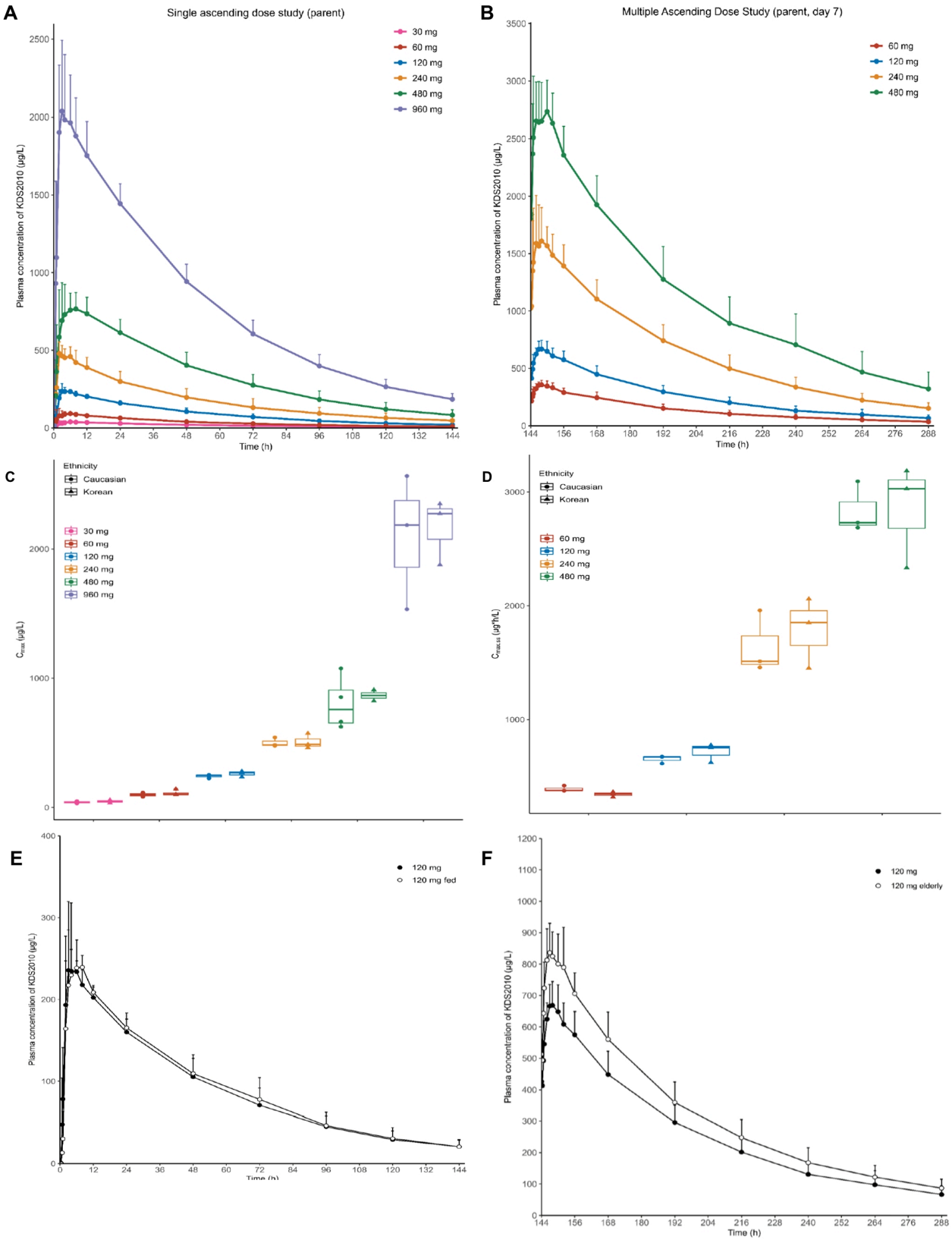
In the MAD study, time-concentration profiles were similar to those observed with single doses (Figure 8B). In elderly subjects, there was an approximate 1.25-fold increase in systemic exposure, compared to younger adults (Figure 8F). PK comparisons between Caucasian and Korean participants revealed similar major PK parameters (Figure 8D).

Figure 7: KDS2010 Phase 1 Study Design



Source: NeuroBiogen Corporate Presentation, December 2024

Figure 8: PK / PD Results of KDS2010 in Phase 1 Study



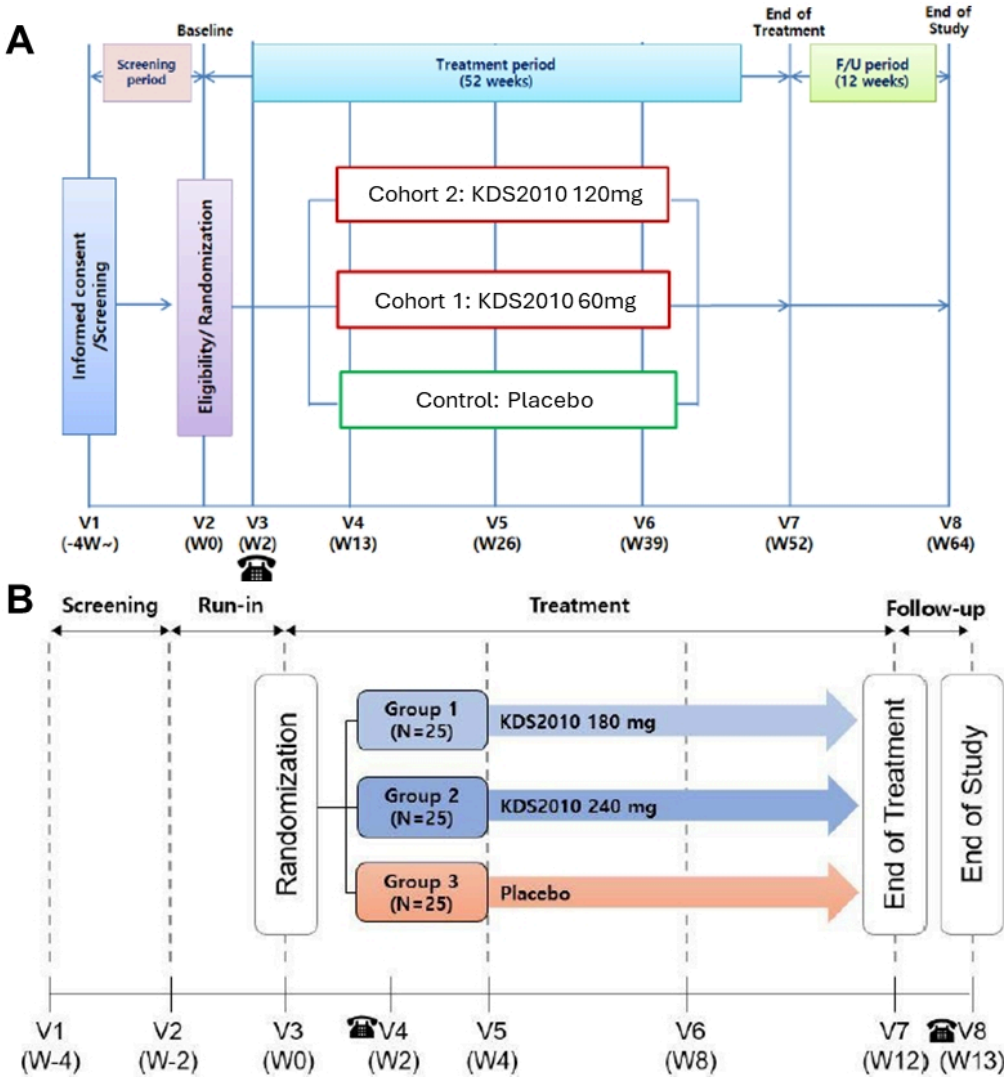
Source: NeuroBiogen Corporate Presentation, December 2024

Plan of Clinical Development for KDS2010

Alzheimer's Disease. The Phase 2a clinical trial for KDS2010-AD, with IND submission planned, is a 64-week randomized, double-blind, placebo-controlled, dose-finding study designed to assess the efficacy and safety of KDS2010 in 114 patients with AD with mild cognitive impairment (MCI) or mild dementia due to AD (Figure 9A). Participants, aged 50 to 85, will undergo 52 weeks of treatment followed by a 12-week follow-up period. The trial includes patients with MCI (Stage Two or Three per NIA-AA 2018, CDR-SB 0.5–2.0) and mild AD (Stage Four, CDR-SB 2.5–4.0), with MMSE scores ranging from 18 to 30 and amyloid PET confirmation. Primary endpoints include changes in CDR-SB (26 and 64 weeks), MMSE (26, 52, and 64 weeks), and ADAS-Cog13 (26, 52, and 64 weeks), alongside biomarker analyses at 26, 52, and 64 weeks, measuring MAO-B-specific activity, GFAP, P-tau181, P-tau217, Aβ-40, Aβ-42, NfL, BDNF, IL-1β, and TNF-α.

Obesity. The Phase 2a clinical trial for KDS2010-Obesity, with IND submission planned, is a 15-week randomized, double-blind, placebo-controlled, dose-finding study designed to evaluate the efficacy and safety of KDS2010 in 75 overweight or obese patients (Figure 9B). The study includes a two-week run-in period with therapeutic lifestyle changes, followed by 12 weeks of treatment and a one-week follow-up. Participants, aged 19 and older, will include males and females with a BMI of ≥30 kg/m² or ≥27 kg/m² with at least one weight-related comorbidity (treated or untreated), such as hypertension, dyslipidemia, or cardiovascular disease. The primary endpoints are the percentage of participants achieving ≥5% body weight reduction and the percentage change in body weight from baseline.

Figure 9: Clinical Trials for KDS2010



Source: NeuroBiogen Corporate Presentation, December 2024

Valuation and Risks

We arrive at our twelve-month price target of $13/share by assessing the after-tax, risk adjusted NPV of potential future cash flows from the company's ZTlido, ELYXYB and GLOPERBA programs, in addition to the estimated value of pipeline assets. For commercial-stage assets, the probability-adjusted, fully taxed (21%) NPV (15% discount rate) of potential cash flows through 2036 is ~$1.2B or $10/share. We estimate that the value of pipeline assets to be $300M, or $3/share. The combined total NPV of all the assets is ~$1.5B or $13/share, corresponding to our 12-month price target. Significant factors that could impede shares from reaching our price target include the failure of ELYXYB's label expansion into acute pain and lower-than-estimated sales. In addition, the company may not be able to raise additional funds to repay debt and to complete development of drug candidates.

Company description

Scilex Holding Company focuses on acquiring, developing, and commercializing non-opioid pain management products for the treatment of acute and chronic pain. Its commercial products include ZTlido (lidocaine topical system) 1.8%, a prescription lidocaine topical product for the relief of neuropathic pain associated with postherpetic neuralgia (PHN), which is a form of post-shingles nerve pain; ELYXYB, a ready-to-use oral solution for the acute treatment of migraine with or without aura in adults; and GLOPERBA, a liquid oral version of the anti-gout medicine colchicine indicated for the prophylaxis of painful gout flares in adults. The company is also developing three product candidates, including SP-102 (10 mg dexamethasone sodium phosphate viscous gel) (SEMDEXA), a novel viscous gel formulation of a corticosteroid used for epidural injections, which has completed a Phase 3 study to treat lumbosacral radicular pain or sciatica; SP-103 (lidocaine topical system) 5.4% (SP-103), a formulation of ZTlido for the treatment of chronic neck pain and low back pain (LBP) that has completed a Phase 2 trial; and SP-104 (4.5 mg low-dose naltrexone hydrochloride delayed-release capsules) (SP-104), a novel low-dose delayed-release naltrexone hydrochloride, which has completed Phase 1 trials for the treatment of fibromyalgia. The company is headquartered in Palo Alto, California.

Important Disclosures

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RETURN ASSESSMENT

Market Outperform (Buy): The common stock of the company is expected to outperform a passive index comprised of all the common stock of companies within the same sector.

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Market Underperform (Sell): The common stock of the company is expected to underperform a passive index comprised of all the common stock of companies within the same sector.

Related Companies Mentioned in this Report as of December 20, 2024

Company	Ticker	Rodman & Renshaw Rating	12 Month Price Target	Price
Scilex Holding Company	SCLX	Buy	13.00	0.44

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Scilex Holding Company Rating History as of 12/18/2024

Distribution of Ratings Table as of December 20, 2024				
			IB Service/Past 12 Months	
Ratings	Count	Percent	Count	Percent
BUY	61	91.04%	4	6.56%
HOLD	5	7.46%	0	0.00%
SELL	1	1.49%	0	0.00%
NOT RATED	0	0.00%	0	0.00%

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