MIRA™
Pharmaceuticals

NASDAQ: **MIRA**
February 2026

Management

Erez Aminov
Chief Executive Officer & Executive Chairman

► CEO & Chairman of MIRA Pharmaceuticals (Nasdaq: MIRA) and Telomir Pharmaceuticals (Nasdaq: TELO), advancing a unique pipeline designed with safety at its core to address major unmet needs in neuroscience, oncology, metabolism, and aging.

► 18+ years of leadership experience, raising capital, securing FDA IND clearance, initiating Phase 1 trials, and overseeing all aspects of company operations and decision-making.

► Known for a results-driven, mission-focused approach, building partnerships and delivering therapies that align with patient safety, unmet need, and long-term commercial success.

Itzchak Angel, PhD
Chief Scientific Advisor

► Over 40 years of experience in guiding medical, pharmaceutical, drug, and business development in both large and emerging companies.

► Expertise in small molecules, botanical drugs, Biotechnology products, delivery systems, medical devices, and drug-device combinations.

► Former Head of Pharmacology at Synthelabo (Sanofi-Aventis, Paris, France) where he participated in research and development of drugs such as Xatral (alfuzosin), Ambien (zolpidem) and Mizollen (mizolastine).

Alan Weichselbaum, CPA, MBA
Chief Financial Officer

► Seasoned Financial Executive with 30+ years of experience in corporate finance, capital markets, and strategic advisory; currently CFO of both MIRA and Telomir Pharmaceuticals.

► Board and Advisory Leadership as Director of FinWise Bancorp (Nasdaq: FINW) and founder of The Wexus Group, advising growth-stage companies on capital structuring and exit strategies.

► Capital Markets Expertise gained through senior Wall Street roles, hedge fund management, and leadership in institutional transactions across public and private markets.

Alex Weisman, PhD
Scientific Advisor

► Occupied executive positions of VPR & D and Chief Scientist at numerous Israeli and international pharmaceutical companies.

► Currently serve as an advisor and management team member for companies developing new products for the chemicals, pharmaceuticals, and food industries.

► More than 30 years of experience in the development, characterization, scale-up, technology transfer, trouble shooting, production and registration of novel and generic drugs, and other pharmaceutical and chemical products.

Corporate Overview

We are MIRA Pharmaceuticals— advancing a differentiated pipeline of oral therapies for neurologic, neuropsychiatric, and metabolic disorders. Built on safety and efficacy, our programs target high-value markets with significant unmet need and strong clinical demand.

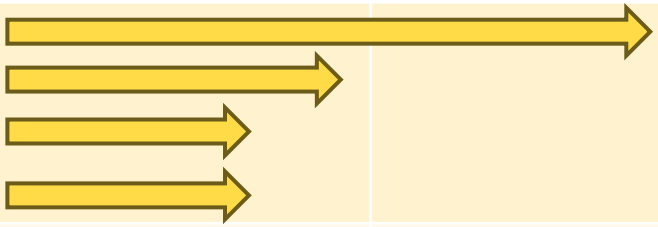
Our pipeline includes:

Ketamir-2 – a next-generation oral ketamine analog with improved bioavailability, selectivity, and safety profile. It is currently in Phase 1 clinical trials for neuropathic pain.

SKNY-1 – a THCV analog designed for obesity and smoking cessation. It modulates CB1, CB2, and MAO-B with receptor selectivity that minimizes psychiatric risk and supports metabolic balance.

MIRA-55 – a THC analog designed to reduce anxiety and improve cognition in aging populations, showing activity across models of memory, inflammatory pain, and behavioral regulation.

Our Pipeline

	Indications	Pre-Clinical	Phase-I	Phase-II
Ketamir-2	Neuropathic Pain (oral) Pain (topical) PTSD Treatment Resistant Depression (TRD)			
Mira-55	Inflammatory Pain			
SKNY-1	Obesity Smoking Cessation			

Ketamir-2: A Novel Oral Approach to Treating Neuropathic Pain

An advanced ketamine analog with fewer side effects, better safety, and clinical-stage momentum.

What is Ketamir-2?

- A next-generation oral ketamine analog with improved bioavailability, selectivity, and safety profile, specifically engineered for neuropathic pain, chemotherapy-induced peripheral neuropathy (CIPN) and treatment-resistant depression.

Neuropathic Pain: A Major Unmet Need:

- Affects 7–10% of the global population, including patients with diabetic neuropathy, postherpetic neuralgia, CIPN and MS-related pain. Current treatments are often ineffective or come with significant side effects and addiction risks.

Why It Matters to Pharma Leaders:

- Existing therapies (anticonvulsants, SNRIs, opioids) offer limited efficacy and poor tolerability. DEA review confirmed
- Ketamir-2 is not a controlled substance, making it a safer, rapid-acting oral alternative with clear regulatory and commercial advantages.

Current Status:

- Undergoing a Phase I clinical trial at Hadassah Medical Center in Israel, showing promising early tolerability and safety.

Key Therapeutic Area

Neuropathic Pain: High Prevalence, Low Satisfaction, and Urgent Demand

An underserved patient population where existing treatments fall short — and the risk of addiction runs high.

- Widespread and Costly Neuropathic pain affects 7–10% of the population globally, often linked to diabetes, CIPN, MS, shingles, and nerve injury. It's one of the most common forms of chronic pain.

Current Treatments Are Insufficient

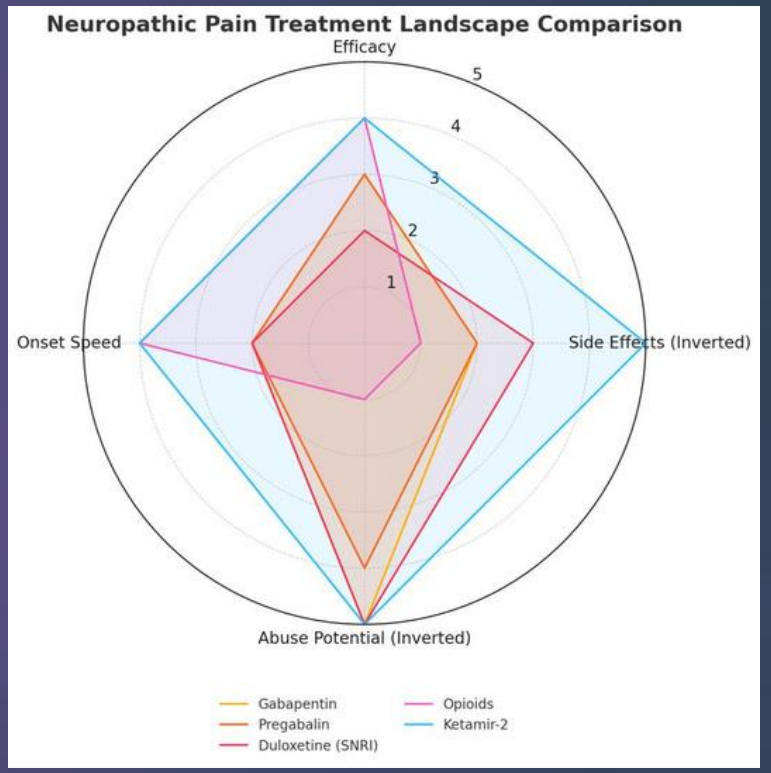
- First-line therapies like gabapentin, pregabalin, and SNRIs offer limited relief and come with CNS side effects, sedation, or tolerance concerns. Opioids are a last resort, burdened by addiction risk.

No Oral, Rapid-Acting, Low-Abuse Option Exists Today

- There's a significant gap in the market for a therapy that is oral, non-addictive, and provides fast-acting, targeted relief without psychomimetic or sedative effects.

Opportunity

- Entering this space positions any firm as a leader in next-generation non-opioid pain therapeutics, aligned with both public health priorities and long-term CNS portfolio expansion.



Key Therapeutic Area

Treatment Resistance Depression (TRD) & Post-Traumatic Stress Disorder (PTSD): High Prevalence, Limited Options, and Urgent Demand

An underserved patient population where existing treatments fall short — and safe, effective options remain scarce.

Widespread and Costly:

- TRD affects ~30% of major depression patients who fail ≥2 antidepressant therapies. PTSD affects 3.6% (past-year) and 6.8% (lifetime) of U.S. adults, with millions more worldwide.

Current Treatments Are Insufficient:

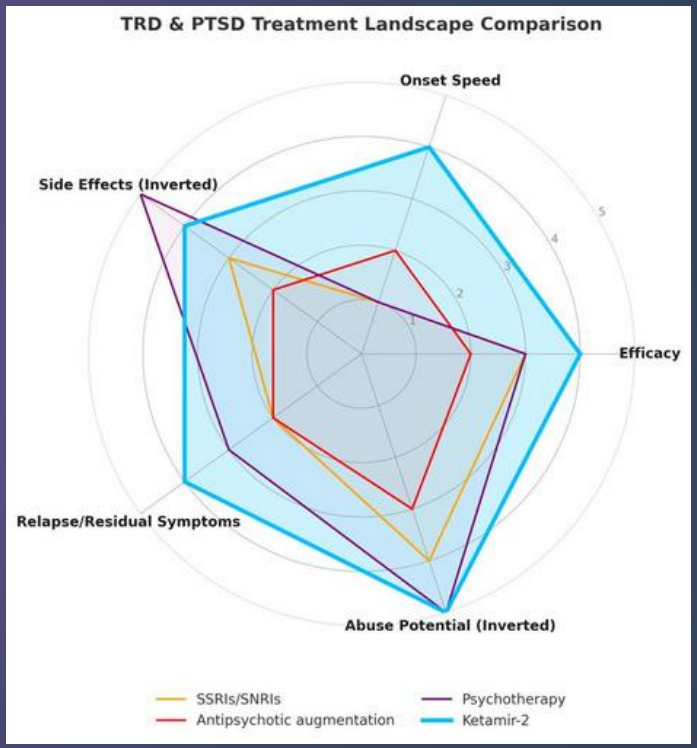
- TRD: Limited efficacy from SSRIs, SNRIs, and augmentation therapies; high relapse rates.
- PTSD: Standard options (SSRIs, therapy) leave many patients with residual symptoms or intolerable side effects.

No Oral, Rapid-Acting, Low-Abuse Option Exists Today:

- Market gap for therapies that are fast-acting, well-tolerated, and non-addictive, addressing both mood and trauma-related symptoms.

Opportunity:

- TRD global market: $1.3–2.2B (2024–2025), growing 4–9% CAGR.
- PTSD global market: $2.2–3.0B (2024), steady growth ~4% CAGR.
- Expanding into TRD & PTSD positions Ketamir-2 as a potential first-in-class oral option across high-need psychiatric indications.



Ketamir-2: CMC

Simple and scalable process for drug substance and drug product production.

Drug Substance Manufacturing Process

- Simple, high yield drug substance synthesis
- Source for Starting Material of high quality established and approved.
- No impurities over 0.10% originated from Starting material in the Drug substance
- High Drug substance quality attributes
- Particle size distribution controlled by precipitation process
- Scaled up to 1.7 kg /GMP batch

Physical Properties

- Complete characterization of crystalline form (FT-IR, Raman, SS-NMR, pXRD, SEM) performed.
- Reproducibility of crystalline form for all manufactured lots confirmed.

Stability Studies

- Drug substance was found stable (no trends in quality parameters) for 9 months.

Drug Product

- Hard gelatine capsules of 50mg and 300 mg developed Dissolution criteria in accordance with USP requirements
- Drug products were found stable (no trends in quality parameters) for 6 months.

Ketamir-2: Pharmacology Precision Targeting at the NMDA Receptor — Without the Collateral Effects

A novel oral NMDA antagonist with selective PCP-site binding and none of ketamine's psychomimetic baggage.

- Low-Affinity, Highly Selective NMDA Modulation Ketamir-2 acts as a low-affinity antagonist at the PCP-site of the NMDA receptor, with an $IC_{50}$of~100µM—avoiding the broader binding that drives ketamine's side effects.

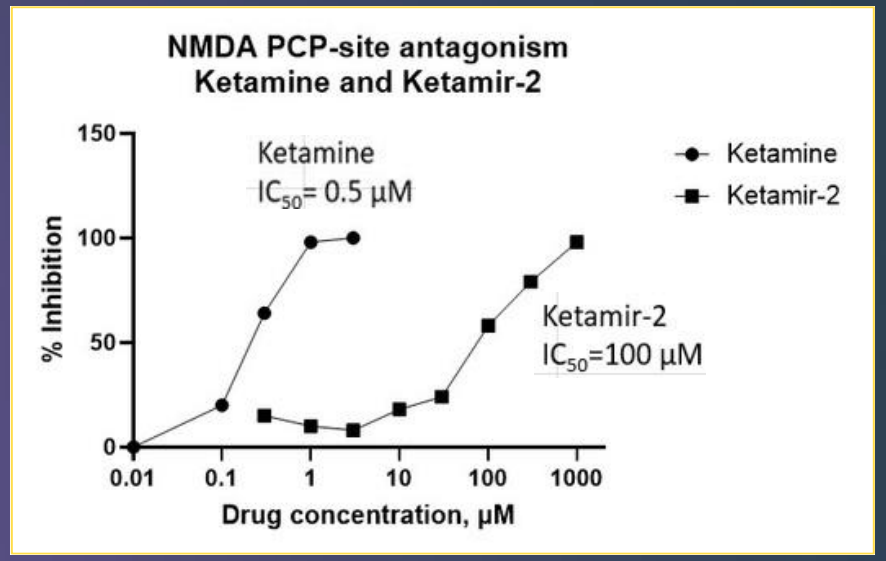
No Off-Target Binding to Problematic Sites

- Unlike ketamine, Ketamir-2 does shows no binding to: AMPA, Kainate, Sigma, Glycine or Glutamate receptors

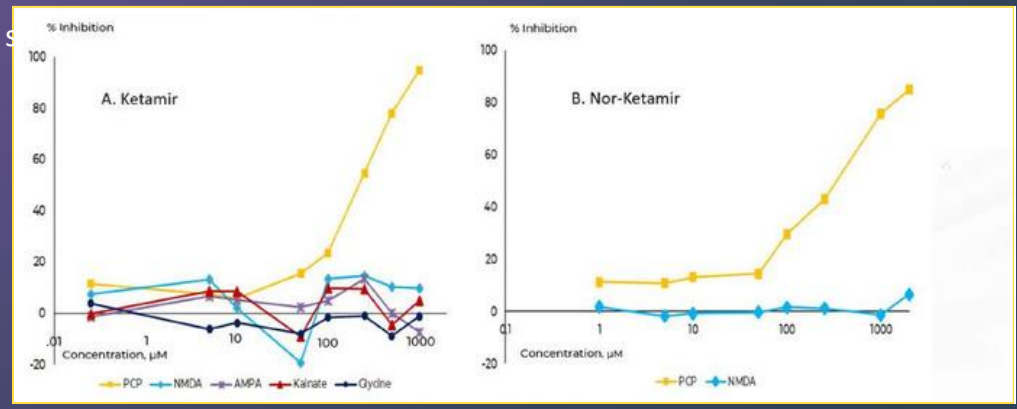
→ This greatly reduces psychosis-like effects, sedation, and misuse potential.

Improved Safety Profile Confirmed in Nor-Ketamir Metabolite

- Its active metabolite shows similarly selective NMDA antagonism (IC_{50} ~300 µM), reinforcing a consistent, predictable pharmacodynamic profile.
- Differentiated CNS Action Without Hyperactivity or Euphoria In vivo data confirms no hyperlocomotion or euphoria, highlighting Ketamir-2's differentiated CNS profile in schizophrenia-relevant behavioral models.



Binding and inhibition data for Ketamir-2 and Nor-Ketamir at the NMDAPCP-site. No significant activity at other glutamatergic receptor



Ketamir-2: Pharmacology CNS Differentiation Without Hyperlocomotion or Euphoria

Unlike ketamine, Ketamir-2 does not induce hyperactivity — a key signal of reduced psychomimetic liability.

- Clinical locomotor activity testing confirms no increase in spontaneous movement with oral or injected Ketamir-2 In contrast, ketamine (20 mg/kg, i.p.) significantly elevated horizontal locomotion
- Supports low risk of psychomotor activation, dissociation, or abuse liability
- Further differentiates Ketamir-2 from existing NMDA-targeting compounds

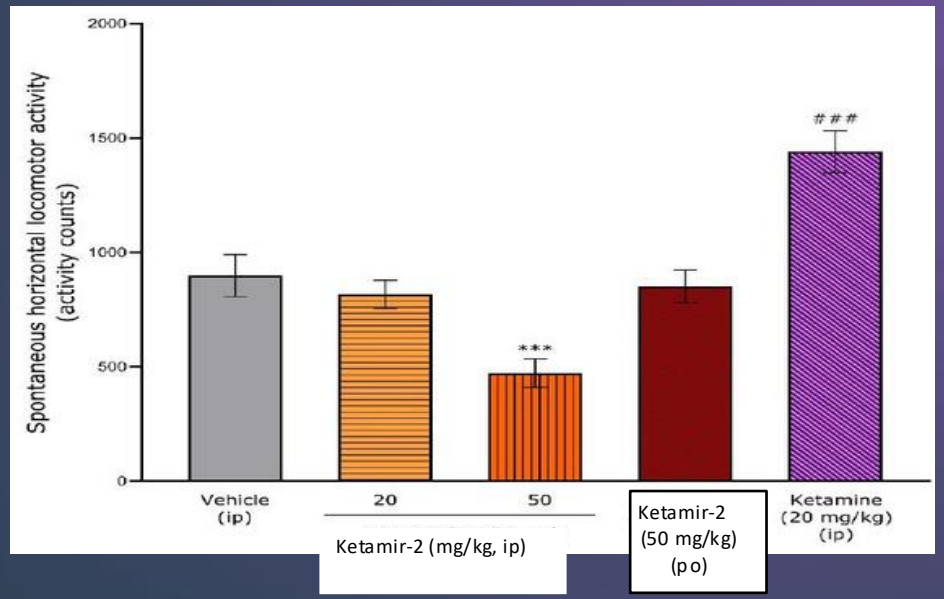
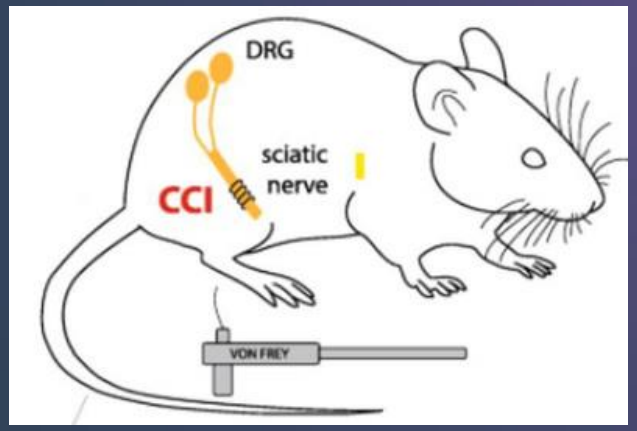


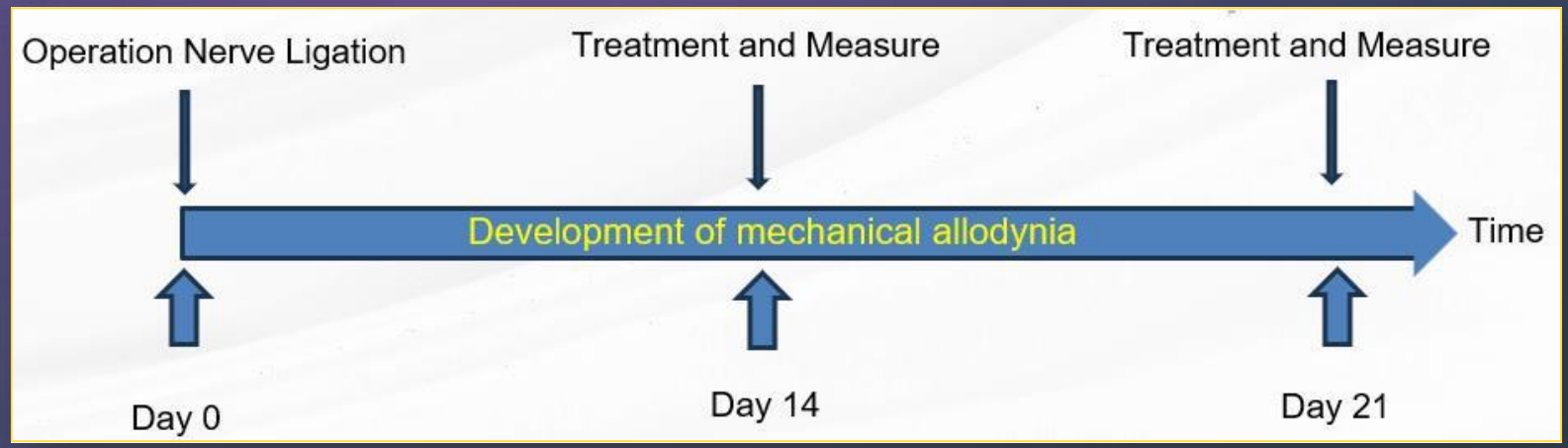
Figure: Spontaneous hyperlocomotion in mice (1 hour)
Results are expressed as mean ± SEM
*****:p<0.001 for ketamir (50mg/kg,ip) vs vehicle (ip) by one-way ANOVA test followed by a Dunnett's multiple comparisons test**
###:p<0.001 for ketamine (20mg/kg,ip) vs vehicle (ip) by two-tailed Student's t test
for

Ketamir-2: Pharmacology Validated Rodent Model for Neuropathic Pain Evaluation

The Chung Model simulates clinically relevant nerve injury to assess therapeutic effects in neuropathic pain.

- Surgical Procedure- Ligation of L5 and L6 spinal nerves induces partial nerve injury, mimicking human neuropathic pain.
- Behavioral Outcomes- Rats exhibit mechanical allodynia — a key symptom of neuropathic pain — measurable using Von Frey filaments.
- Assessment Timeline- Pain response is evaluated on Day 14 and Day 21 post-ligation to capture both development and Persistence of pain.
- Visual Timeline- Treatment overlays the period of mechanical allodynia, providing insight into drug efficacy during peak sensitivity

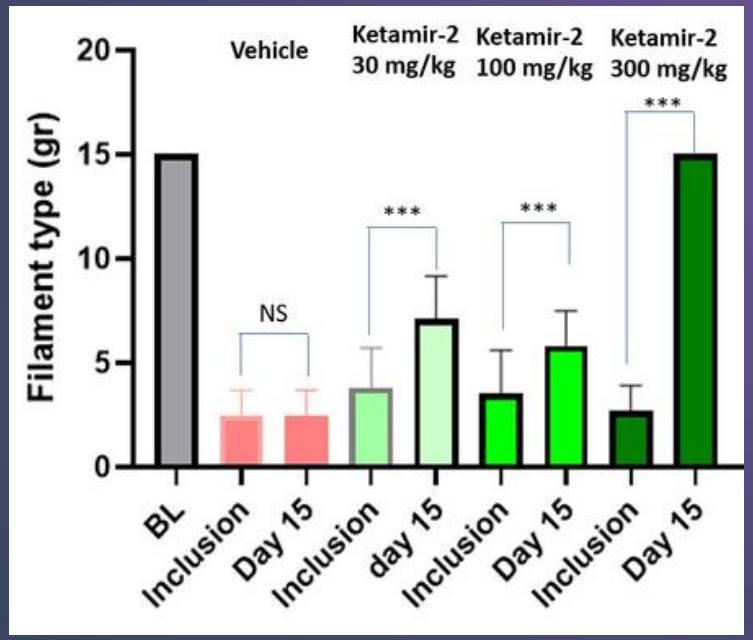




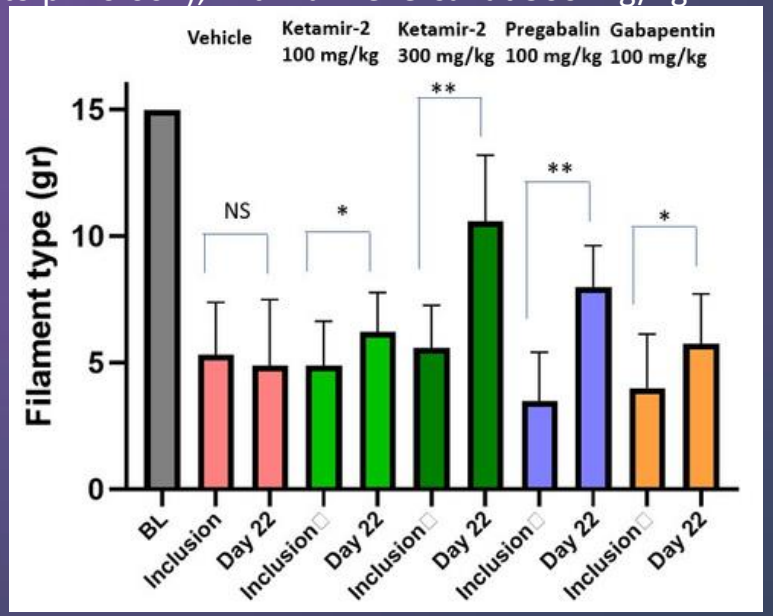
Ketamir-2 Demonstrates Potent Pain Relief in Validated Animal Models

Effective across sexes, models, and doses — with statistically significant reversal of neuropathic pain.

- Chung Model (Rats): - Male and female rats with spinal nerve ligation
- Ketamir-2 produced dose-dependent reversal of allodynia
- Stronger or equal to Ketamine; better than Pregabalin/Gabapentin
- Significant analgesic effects observed at 30–300 mg/kg ($p < 0.01$ to $p < 0.001$), with full reversal at 300 mg/kg.



Male

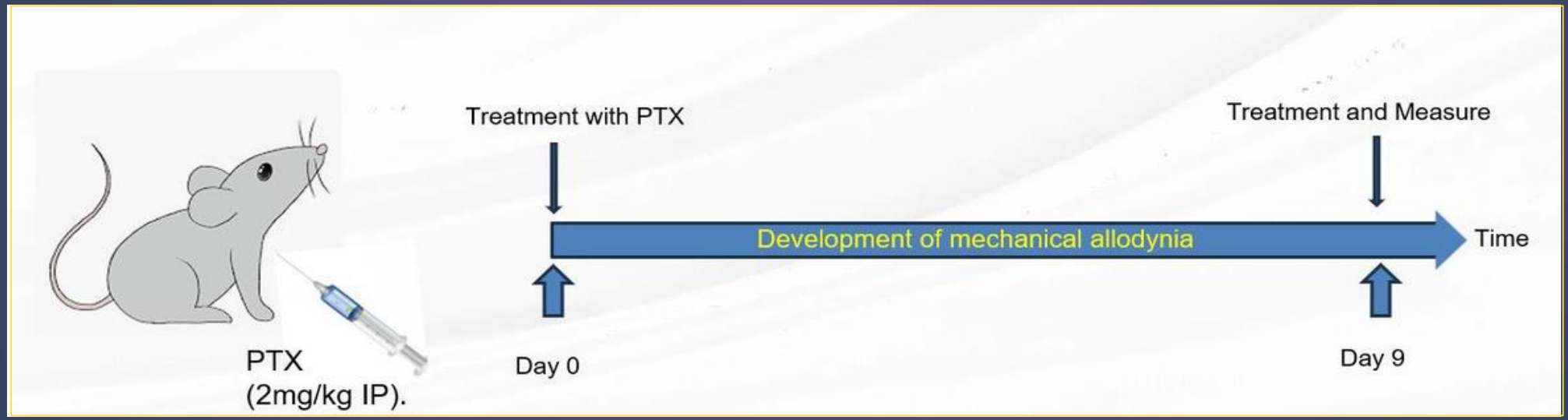


Female

Ketamir-2: Pharmacology PTX-Induced Neuropathy Model in Mice Validates Mechanical Allodynia Development

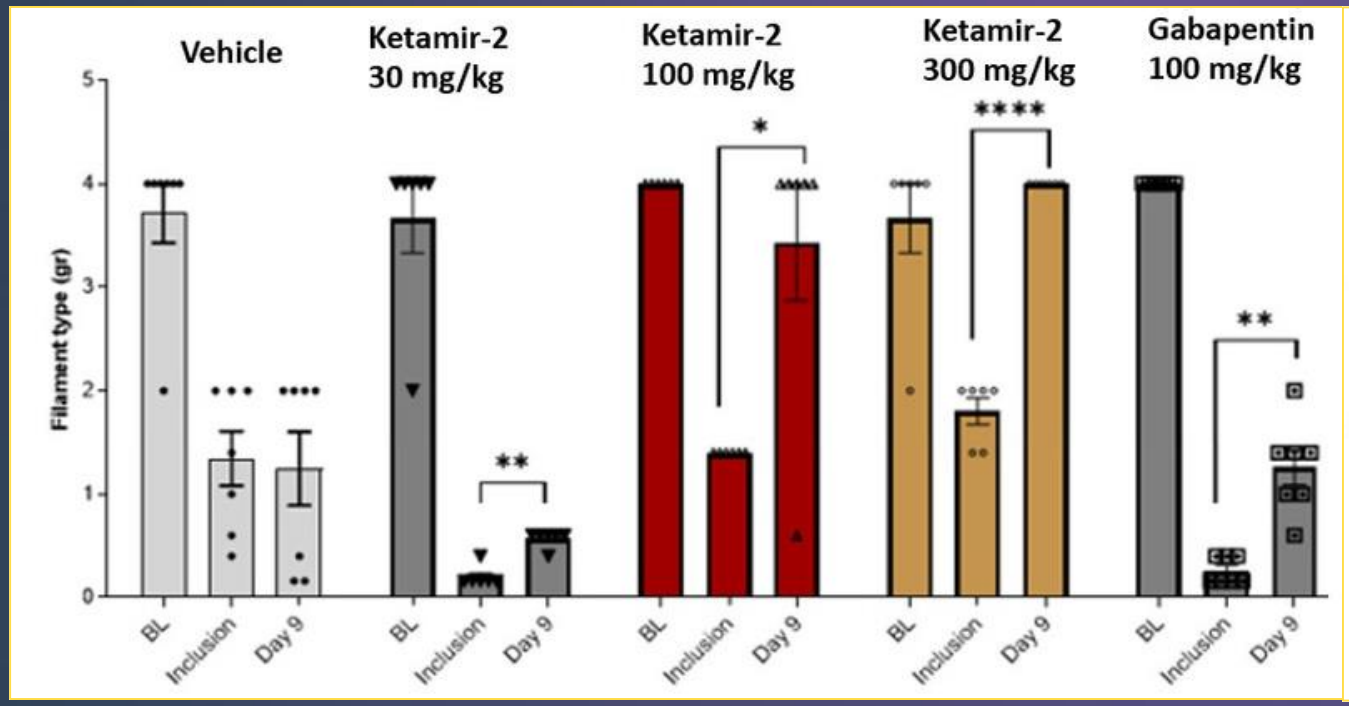
A chemotherapy-induced pain model mimicking clinical neuropathic symptoms—enabling evaluation of therapeutic interventions in a controlled, time-specific window.

- Model Overview: Paclitaxel (PTX) was administered intraperitoneally (2mg/kg) every other day for four doses to induce peripheral neuropathy.
- Assessment Timeline: Pain behavior measured by Von Frey filaments on Day 0 (baseline), Day 8, and Day 9.
- Readout: Mechanical allodynia evaluated 30 minutes post-test item administration on Day 9.
- Outcome Relevance: Provides a clinically relevant model for chemotherapy-induced neuropathic pain, supporting short-term pain reversal studies.

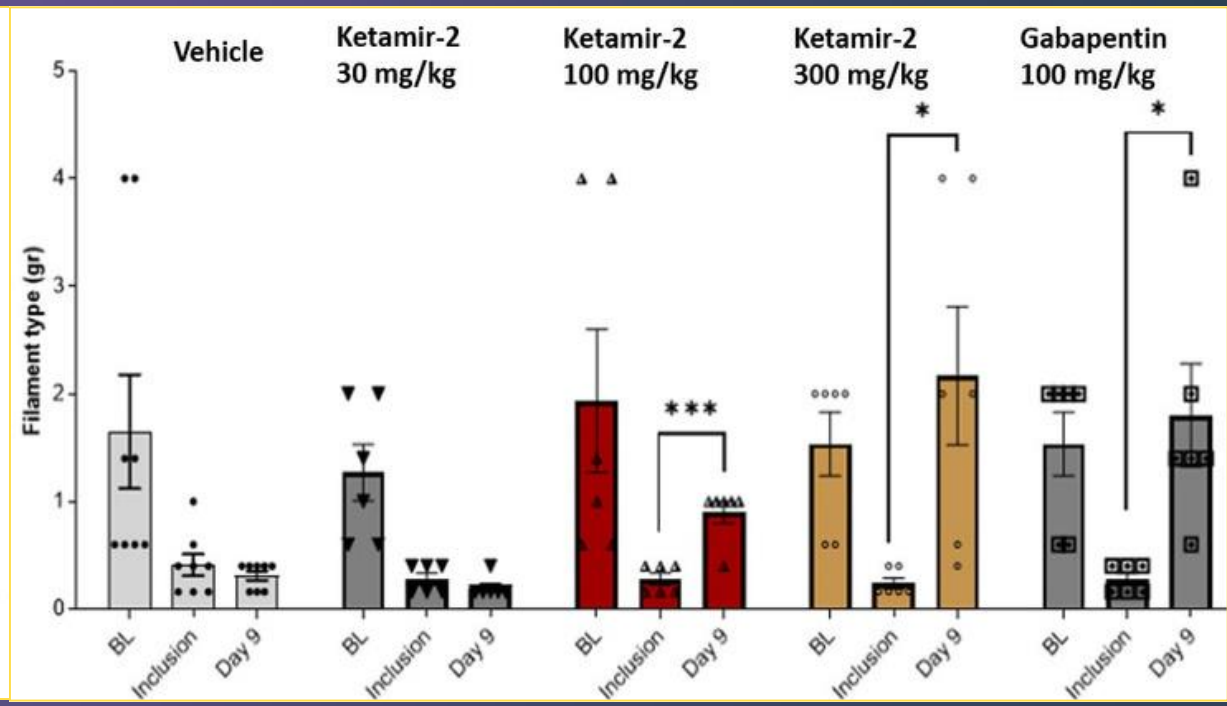


Ketamir-2 Produces Dose-Dependent Pain Reversal in Mice Post-PTX Treatment

Following paclitaxel-induced neuropathy, Ketamir-2 demonstrated significant and progressive pain relief starting at 30mg/kg PO, with complete reversal to baseline levels at 100–300mg/kg—outperforming Gabapentin in this validated pain model.



Male



Female

Ketamir-2: Pharmacology Promotes Antidepressant-Like Behavior Without Stimulant Effects

Demonstrates increased movement and velocity without hyperactivity — mirroring classical antidepressant responses in the Open Field Test

- Open Field Test is a validated model for screening antidepressant and anxiolytic activity.
- Ketamir-2treatmentledtoasignificant, dose-dependent increase in total distance moved (Panel A) and velocity (Panel B).
- This pattern aligns with classical antidepressant effects, suggesting improved mood and motivation.
- No significant effect was observed with ketamine (30 mg/kg PO) — underscoring Ketamir-2's differentiation.
- Trend toward increased time in the center of the field (Panel C) suggests potential anxiolytic activity.
- Behavioral effects observed with Ketamir-2 occurred without signs of psychomotor overstimulation.

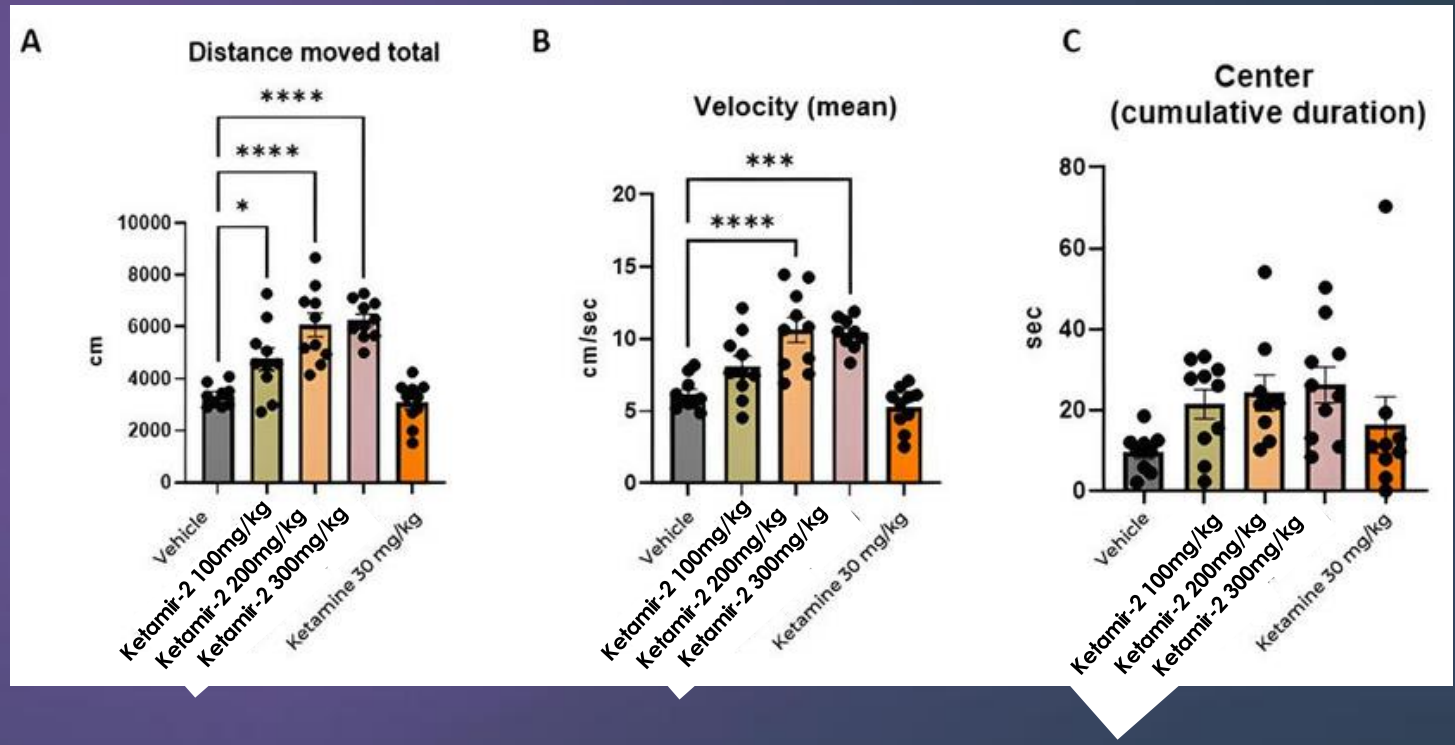


Figure: Open field parameters. (A) Distance moved(cm); (B) mean velocity (cm/seconds) and (C) cumulative time spent in center (seconds). Significance values were obtained through One-Way ANOVA.

Ketamir-2 Shows Anxiolytic-Like Effects in Elevated Plus Maze

Demonstrates increased movement and time in center — consistent with reduced anxiety and enhanced exploratory behavior

- Elevated Plus Maze is a classical assay used to assess anxiolytic and antidepressant activity based on exploratory behavior.
- Mice treated with Ketamir-2 moved significantly farther (Panel A) and faster (Panel B) than vehicle controls — reflecting increased mobility.
- Time spent in closed arms decreased(Panel C), suggesting reduced anxiety and greater willingness to explore open spaces.
- Ketamir-2 effects were dose-dependent, with increased activity seen at 100–300 mg/kg.
- Ketamine (30 mg/kg PO) showed no significant improvement, further differentiating Ketamir-2's anxiolytic potential.
- These findings reinforce Ketamir-2’s behavioral signature consistent with clinical antidepressant and anxiolytic profiles.

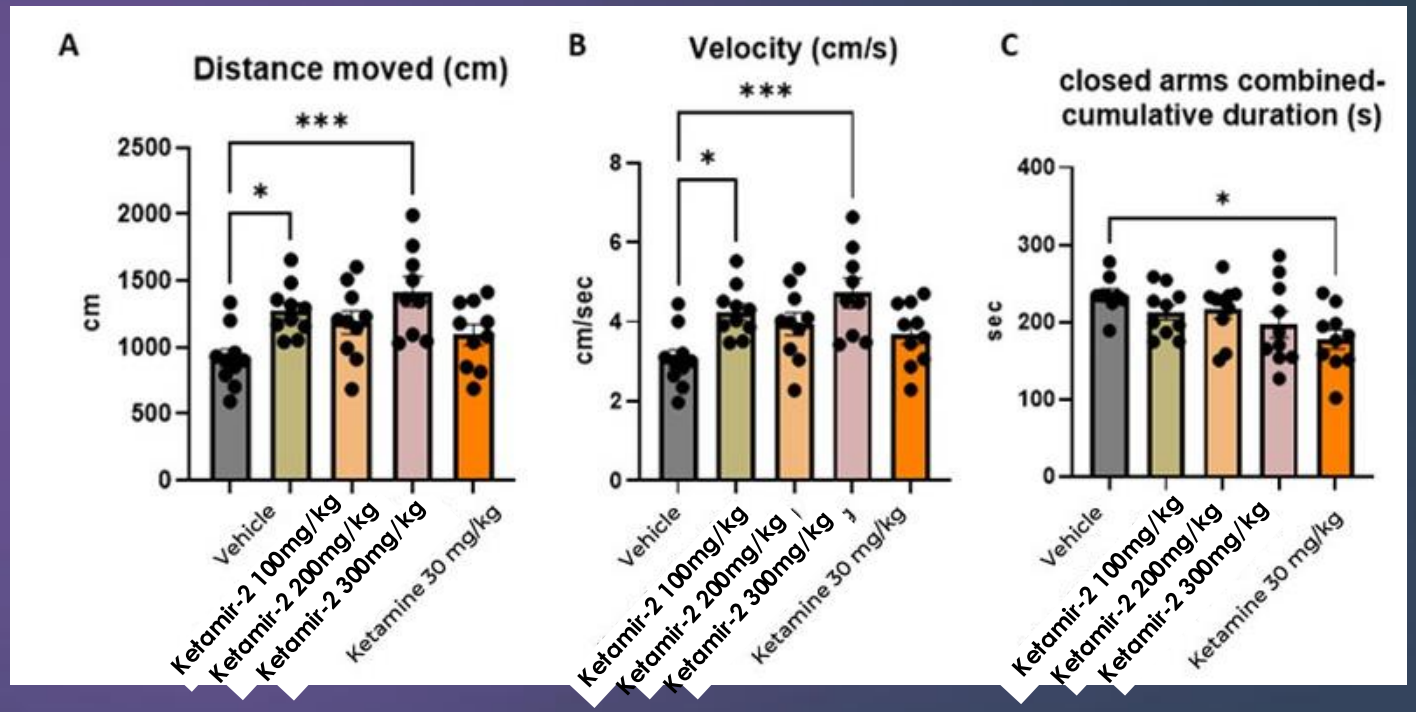
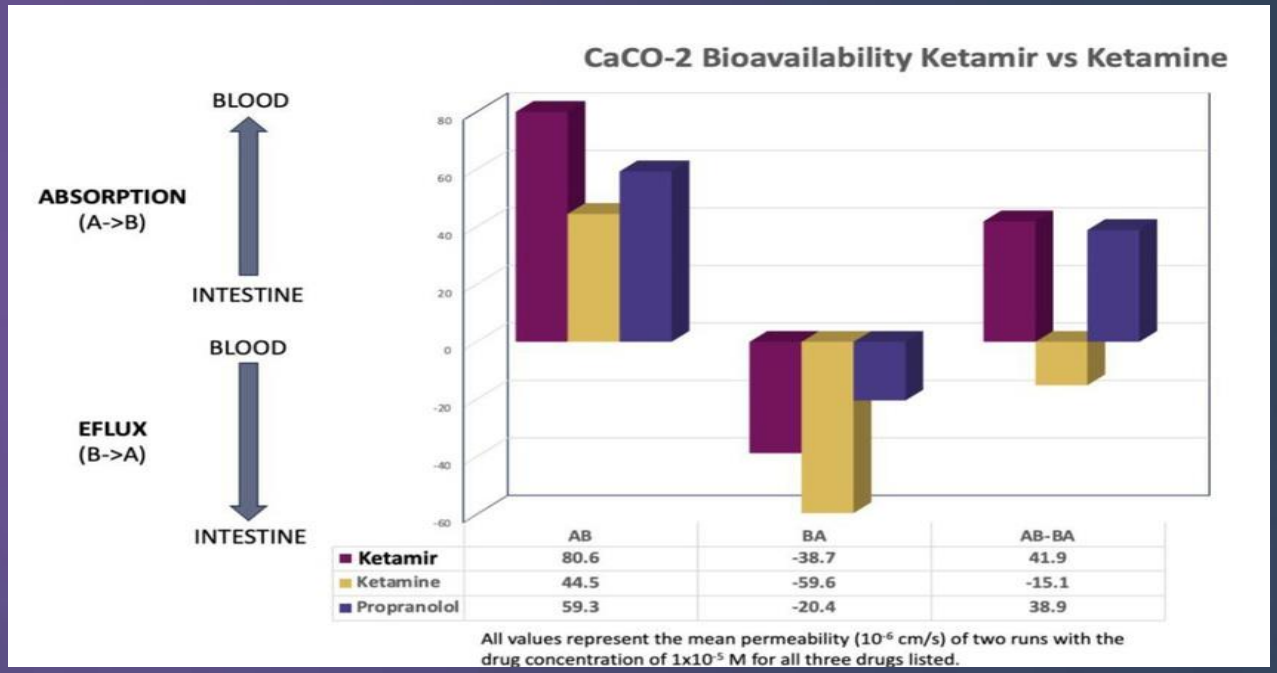


Figure Elevated plus maze (EPM) parameters. (A) Distance moved(cm); (B) mean velocity (cm/seconds), (C) cumulative time spent in open arms (seconds. Significant values were obtained through One-Way ANOVA.

Ketamir-2: ADME Shows Superior Oral Bioavailability and Brain Penetration Potential

Not a substrate of P-gp efflux transporter — enabling improved absorption and CNS exposure compared to ketamine

- Caco-2 assay results confirm Ketamir-2 is not a substrate for P- glycoprotein (P-gp), unlike ketamine.
- Ketamir-2 showed high intestinal absorption (AB: 80.6 × 10^{-6} cm/s) and low efflux (BA: –38.7), similar to the oral reference drug propranolol.
- In contrast, ketamine demonstrated higher efflux than absorption, a classic P-gp interaction profile.
- Net absorption(AB–BA)for Ketamir-2 was 41.9, compared to –15.1 for ketamine, supporting significantly better permeability.
- Predicted oral bioavailability of ~80%, exceeding that of ketamine's oral or intranasal routes.
- Supports potential for convenient, at-home, oral administration — improving treatment access and patient autonomy.



	AB	BA	AB-BA
Ketamir	80.6	-38.7	41.9
Ketamine	44.5	-59.6	-15.1
Propranolol	59.3	-20.4	38.9

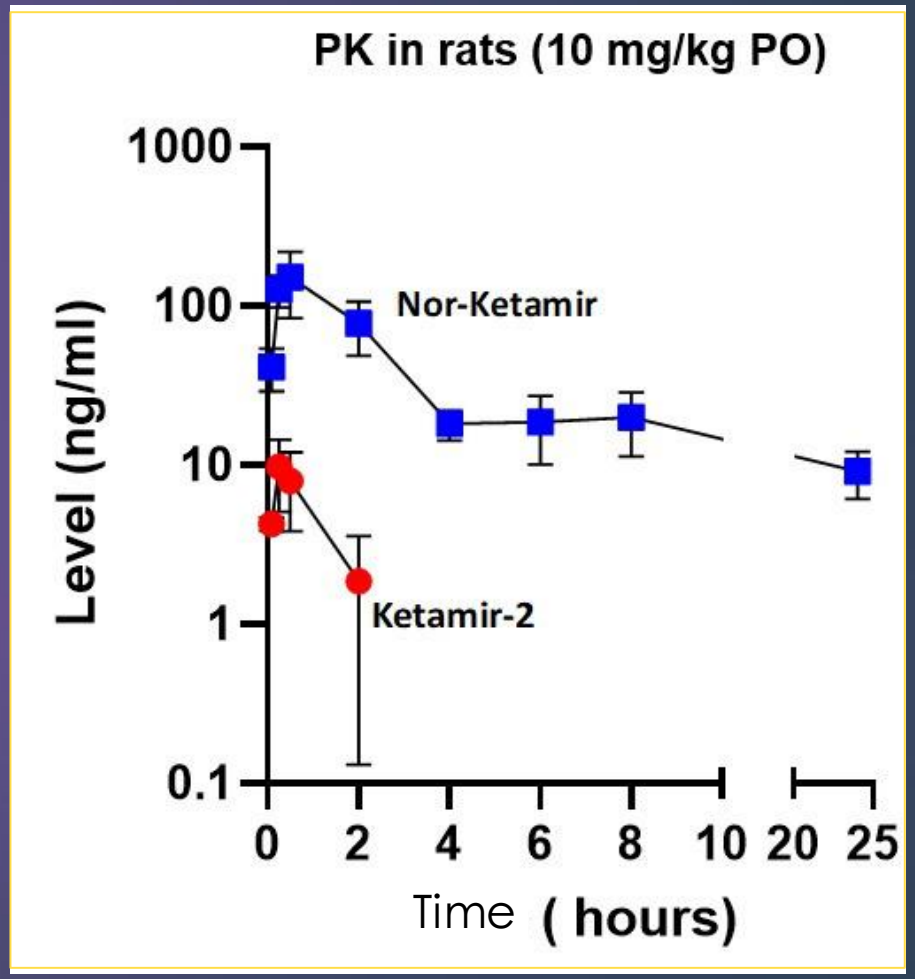
All values represent the mean permeability (10^{-6} cm/s) of two runs with the drug concentration of $1x10^{-5}$ M for all three drugs listed.

Figure: Data obtained from the CaCO-2 model of intestinal absorption. Propranolol, a commonly prescribed beta-blocker that is taken orally and used to treat hypertension, is included as a positive control. The intestinal absorption (AB), Intestinal efflux (BA) and net absorption (AB-BA) are shown.

Ketamir-2: ADME

Rapidly absorbed and metabolized to active Nor-Ketamir

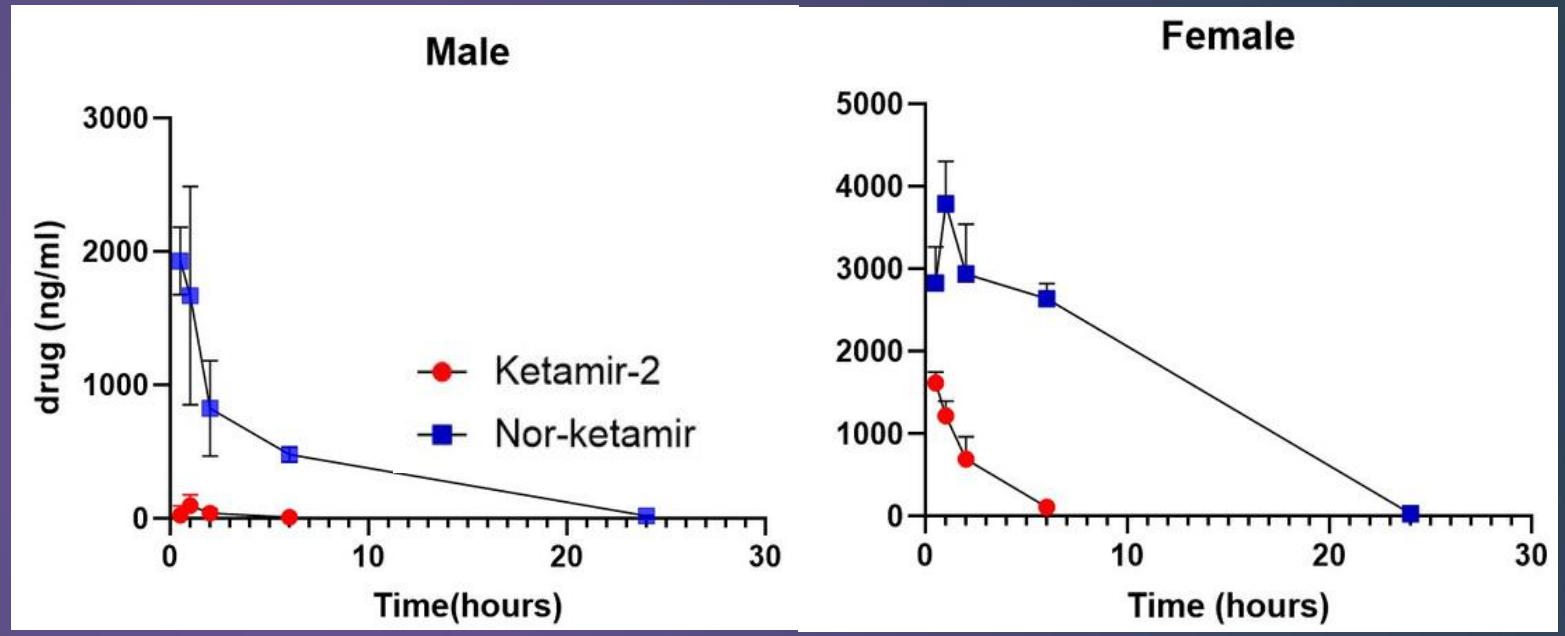
- Ketamir-2 pamoate in a specific 2-hydroxypropyl-beta-cyclodextrin(HP-β-CD formulation is rapidly absorbed
- It is metabolized to the Nor-Ketamir metabolite with has similar pcp-site activity and selectivity as Ketamir-2
- Nor-ketamir has a long elimination half life (4.3 hours, vs 0.25 hours for Ketamir-2) and high levels (Cmax of 250 and 22 ng/ml, respectively) , representing 100% bioavailability compared to IV levels.
- It is also present in the brain in several time points measured



Ketamir-2: ADME

In Rats, females have higher levels of Ketamir-2 and Nor-Ketamir. Not seen in dogs.

- Female rats have a higher level of Ketamir-2 and Nor-Ketamir than males (Cmax of 127 and 1620 ng/ml Ketamir-2, and 1930 and 3790 ng/ml Nor-Ketamir, for males and females respectively)
- No kinetic sex differences were observed in dogs
- Figure depicts plasma levels following 100 mg/kg of Ketamir-2 oral administration



Ketamir-2: Toxicology

Excellent safety and toxicology profile

- Ketamir-2 was found to be highly selective across a broad receptor, enzymes and channels panel
- Non-mutagenic, non-genotoxic in standard assays
- Not neurotoxic in NMDA specific neurotoxicity study
- NOAELs: 300 mg/kg/day in rats; 200 mg/kg/day in dogs

Ketamir-2 Preclinical Summary: Clinical Profile Supports Further Development

Mechanistically selective, orally bioavailable, and non-genotoxic — with consistent activity across models of depression, anxiety, and neuropathic pain

Chemistry & Formulation (CMC)

- **Ketamir-2 is a stable crystalline analog of ketamine, developed as a hemi-pamoate salt**
- Synthesized via a cost-efficient, high-yield process
- Formulated into stable 50 mg and 300 mg oral capsules

Pharmacology

- Acts as a low-affinity, selective PCP-site NMDA antagonist
- Demonstrates potent activity in models of neuropathic pain, depression, and anxiety

ADME & Bioavailability

- Not a P-glycoprotein substrate, supporting improved brain penetration
- Metabolized via N-demethylation (primarily CYP2B6 & CYP3A4) to Nor-Ketamir
- Shows rapid oral absorption and short half-life; metabolite has longer half-life and CNS exposure
- Male to female differences in rats but not in dogs
- Predicted oral bioavailability of Nor-Ketamir~100%

Safety & Toxicology

- Highly selective across a broad receptor panel
- Non-genotoxic in standard assays Not neurotoxic
- NOAELs: 300 mg/kg/day in rats; 200 mg/kg/day in dogs

Ketamir-2: Clinical

Ongoing Phase I Trial: Ascending Dose Safety Study of Oral Ketamir-2 Current SAD and MAD cohorts show favorable safety and tolerability across doses up to 600mg in healthy volunteers.

Design:

- Randomized, double-blind, placebo-controlled, single-center Phase | trial (MIRA-001) assessing safety, tolerability, and pharmacokinetics of oral Ketamir-2 in healthy volunteers

Dosing Overview:

- SAD cohorts (Cohorts 1–4): Single daily oral doses from 50mg to 600mg
- MAD cohorts (Cohorts 5–7): 5-day repeat dosing from 150mg to 600mg

Participants:

- 18 subjects dosed to date with no serious or dose-limiting adverse events reported.

Endpoints:

- Safety (AEs, vitals, ECG, labs) and psychomimetic evaluations (KSET, Bowdle VAS).

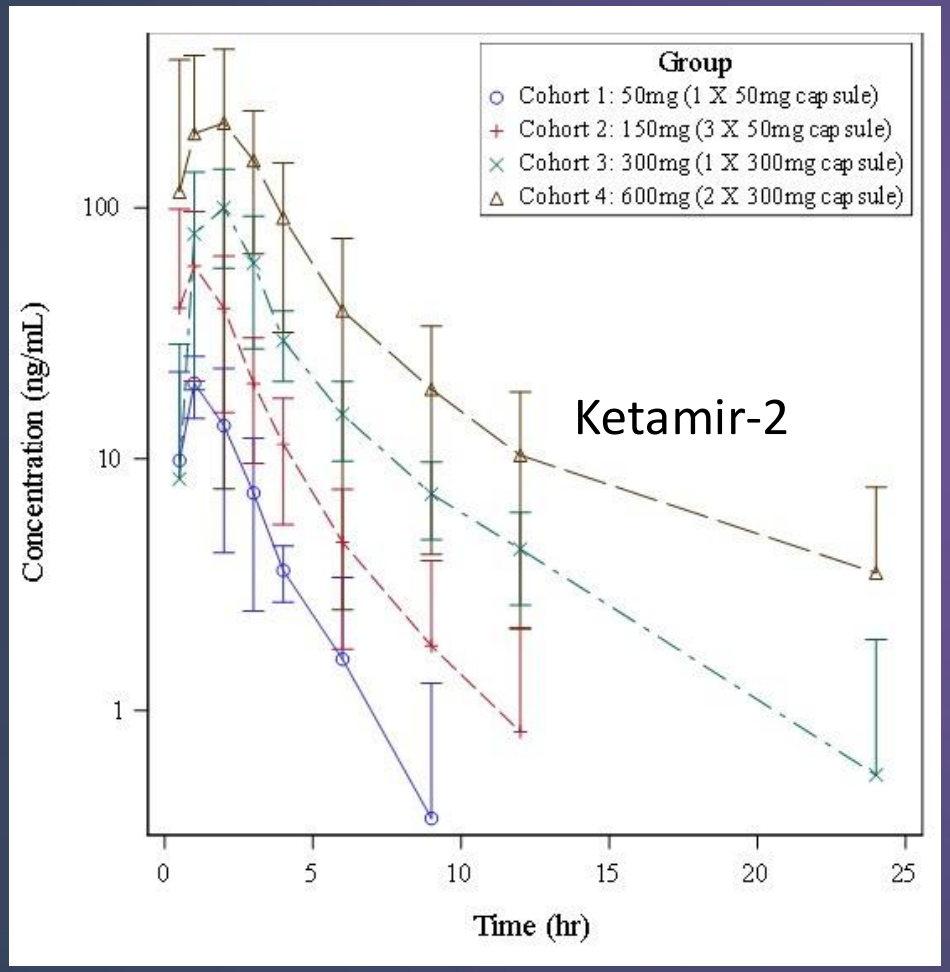
Status:

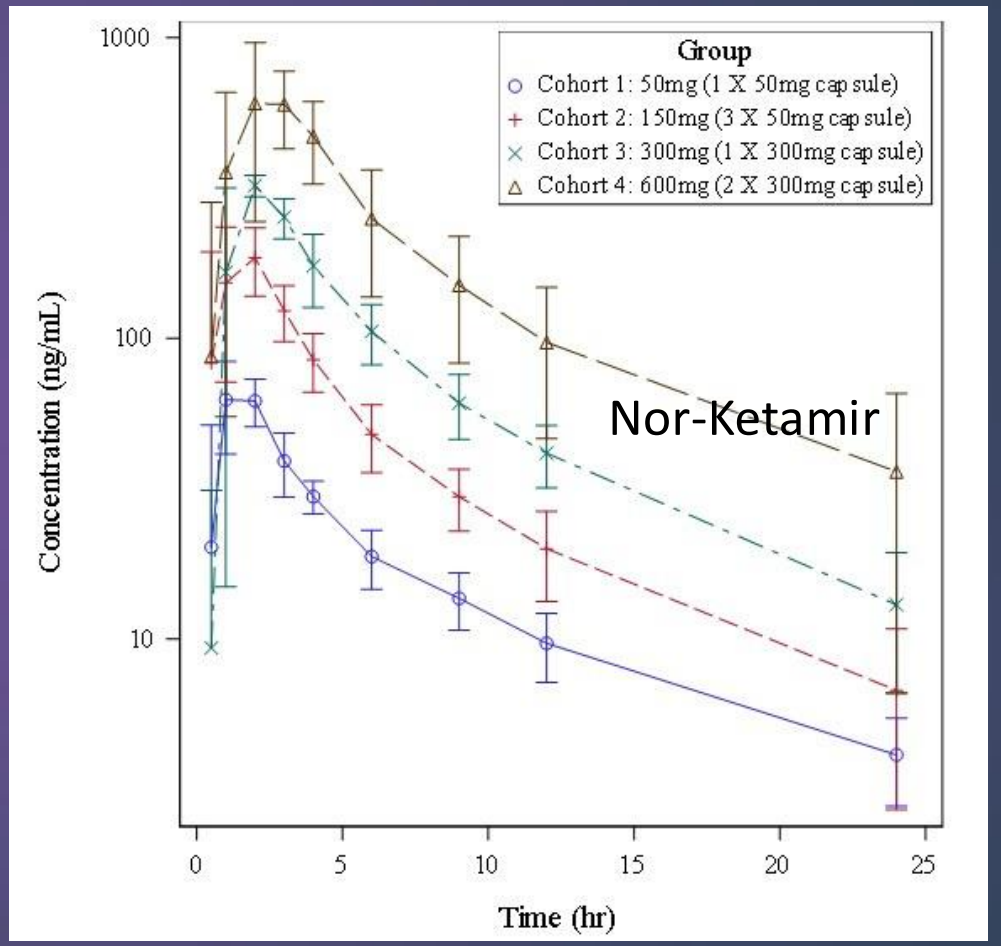
- SAD completed. Initiating MAD shortly.

SAD	Dose level, mg (once daily oral dosage)	Number of participants	
		Ketamir hemipamoate	Placebo
Cohort 1	50mg, 1X50mg capsule	6	2
Cohort 2	150mg, 3X50mg capsule	6	2
Cohort 3	300mg, 1X300mg capsule	6	2
Cohort 4	600mg, 2X300mg capsule	6	2
MAD			
Cohort 5	150mg, 3X50mg capsule for 5 days	6	2
Cohort 6	300mg, 1X300mg capsule for 5 days	6	2
Cohort 7	600mg, 2X300mg capsule for 5 days	6	2
Total Participants		**42**	**14**

Note: 32 subjects dosed to date; table shows planned totals.

Phase-I Results: Human Plasma Levels of Ketamir-2 and Nor-Ketamir





Phase-I Results: Key PK Parameters

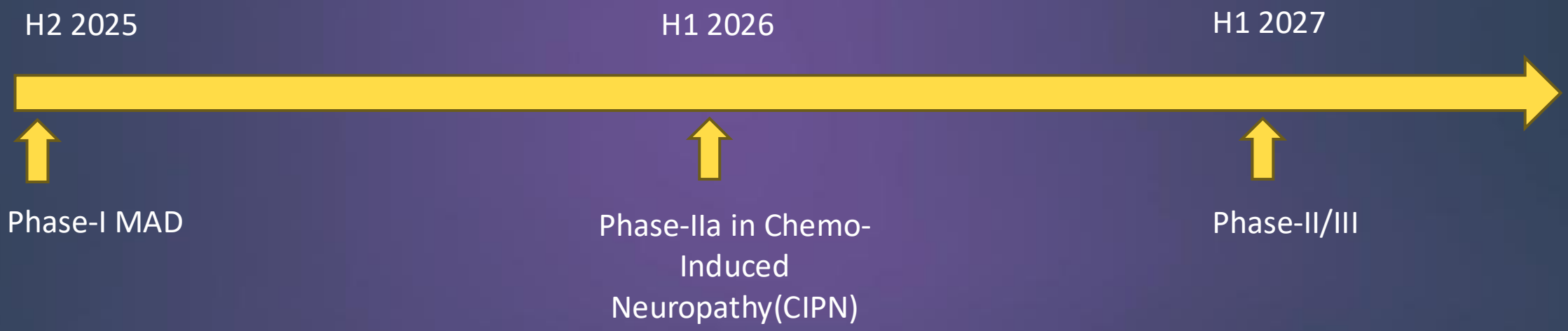
	Ketamir-2	Nor-Ketamir	Ketamir-2	Nor-Ketamir	Ketamir-2	Nor-Ketamir
Dose (mg/day)	Cmax (ng/ml)		Tmax (h)		T1/2 (h)	
50	22.7±2.72	73.9±2.1	1±0.2	1.1±0.2	2.1±.6	8.5±0.8
150	59.6±15.8	210.0±18.9	1.1±0.3	1.7±0.3	2.0±0.5	6.5±1.1
300	111.6±18.0	349±19.6	1.6±0.2	1.9±0.3	3.6±0.7	6.7±0.6
600	258.9±66.7	780±64.7	1.6±0.5	2.0±0.3	5.5±0.6	6.8±0.7

Phase-I Results: Conclusions

- No safety concerns Absorption of Ketamir-2 in human subjects is rapid and dose-proportional with relatively short half-life (2-5 hours)
- Metabolism into nor-Ketamir is rapid, with similar Tmax of the parent compound and the metabolite
- Plasma levels (Cmax) of nor-Ketamir are about 3-fold higher than Ketamir-2
- Plasma half life of nor-Ketamir is much longer than Ketamir-2 (6.5-8.5 hours)
- Exposure to nor-Ketamir (AUC) is 5-7-fold higher than Ketamir-2 Excretion of both Ketamir-2 and nor-Ketamir into the urine is rapid, dose-proportional and at high levels, which are proportional to plasma levels
- This indicates substantial systemic absorption and active renal elimination of the compound. This suggests that the compound is taken up into the bloodstream, circulates systemically, and is efficiently filtered or secreted by the kidneys
- Considering that nor-Ketamir is active, this justifies well a once daily administration

Ketamir-2: Clinical

Expected timeline for Ketamir-2 clinical development

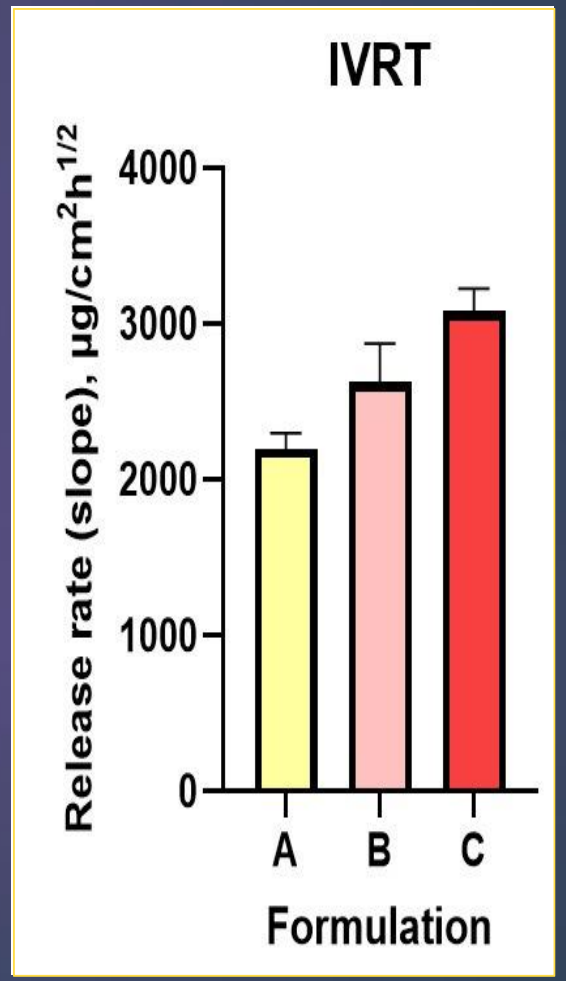


Ketamir-2: Topical Formulation

Topical hydrophobic formulation for Ketamir-2 were developed

- Ketamir-2 was developed in three different oily formulations for topical administration
- Formulations B and C had the best In Vitro Release testing (IVRT) values

Ingredient %w/w	Lot # 190225		
	A	B	C
Ketamir Hemipamoate	5	5	5
White Petrolatum	77	84	85
DMSO	10	5	0
GMS SE	3	1	0
Mineral Oil	5	5	10
sum	100	100	100



Ketamir-2: Topical Formulation

Topical hydrophobic formulation is effective in reducing pain behavior in the formalin test in mice

- Ketamir-2 cream in two formulations was applied to mice paws 30 minutes before and just prior to formalin administration into the paw

- Formalin administration produces a biphasic pain response: an early phase (0–10 minutes) attributed to direct nociceptor activation (neurogenic pain) and a late phase (15–60 minutes) often linked to inflammatory mechanisms and central sensitization

Two variables were evaluated:

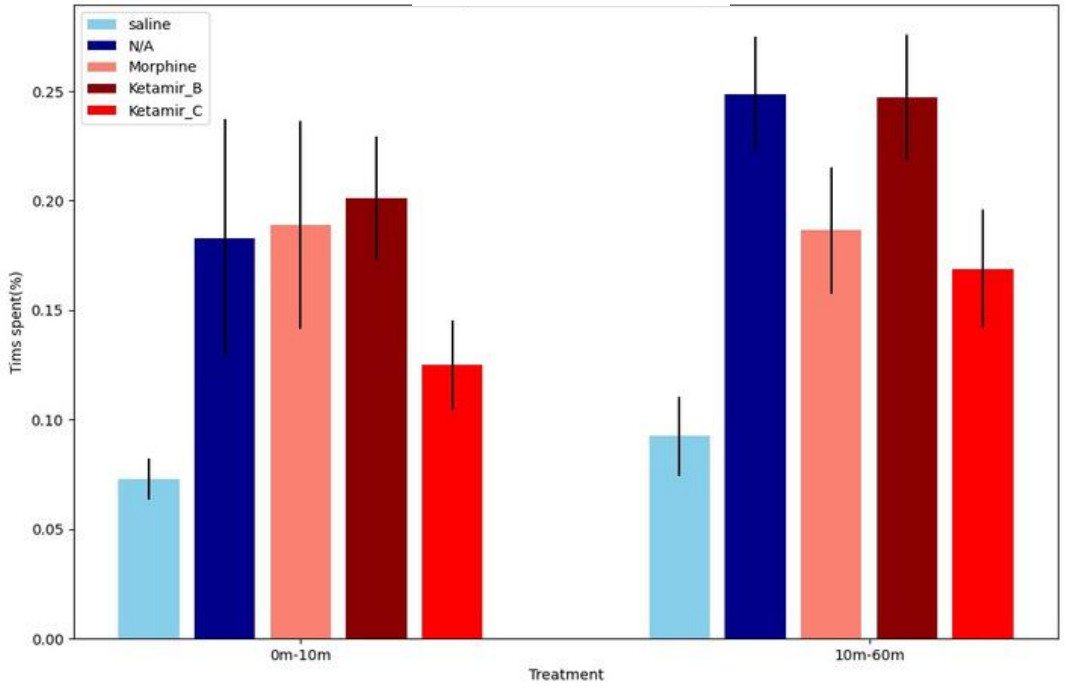
- Paw licking - Licking reflects a direct response to pain or irritation at the injection site Paw lifting - Lifting indicates an avoidance response to pain or discomfort, as the rodent attempts to minimize contact with the surface. It is prominent in both Phase I (acute pain) and Phase II (sustained pain).

- Ketamir-2 (formulation C) applied topically, reduced both the early stage and the late stages responses induced by Formalin, to near control values.

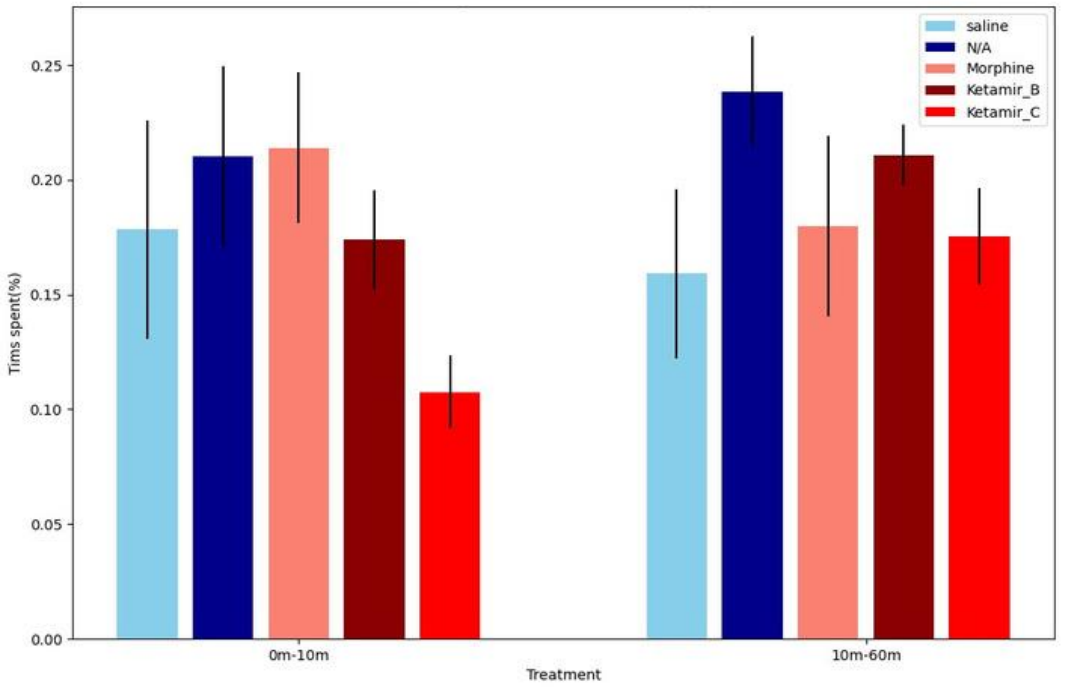
Ketamir-2: Topical Formulation

Topical hydrophobic formulation #C is effective in reducing pain behavior in the formalin test in mice. Efficacy on both early and late phases and in two tests with better or similar to injected morphine

Paw Licking

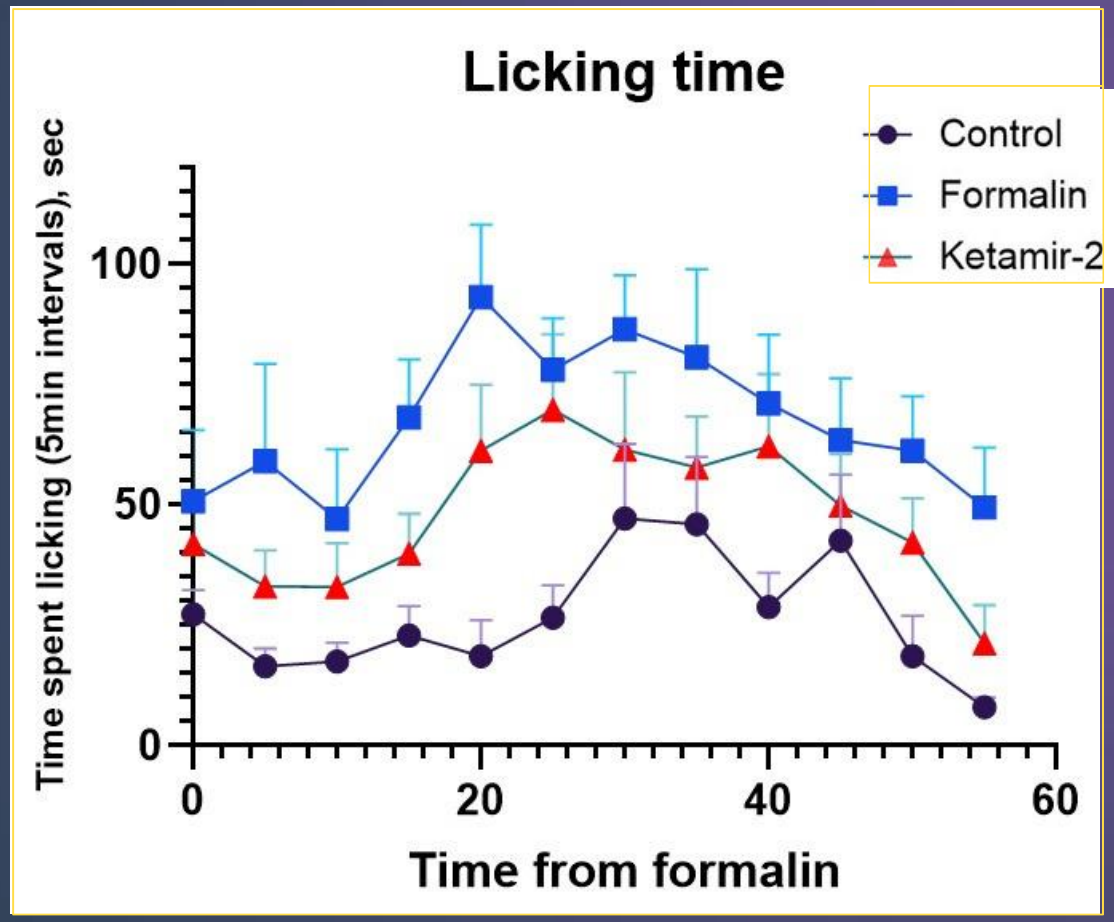


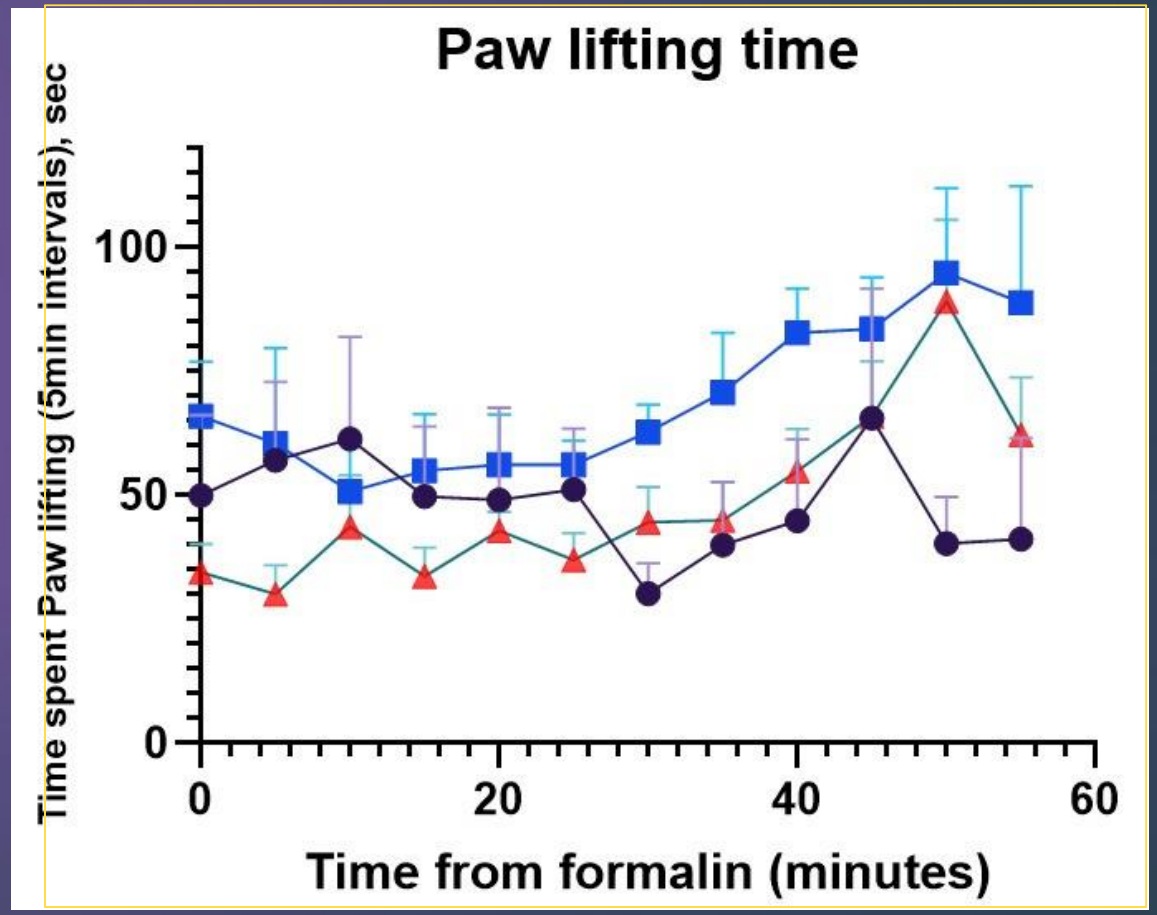
Paw Lifting



Ketamir-2: Topical Formulation

Topical hydrophobic formulation #C is effective in reducing pain behavior in the formalin test in mice – view of the whole-time profile



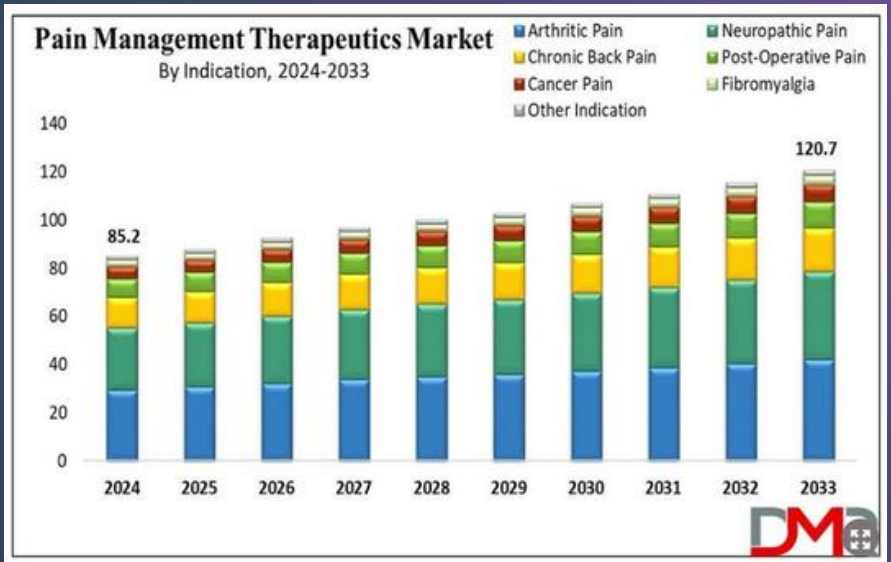


Ketamir-2 Development Pathway Toward Phase IIa

A strong foundation from clinical and Phase 1 data supports rapid advancement into neuropathy patients.

Market Opportunity (global pain):

- Pain therapeutics market expected to exceed $120B by 2033
- Neuropathic pain remains a large, underserved segment
- Ketamir-2 offers an oral, CNS-safe alternative with clear differentiation



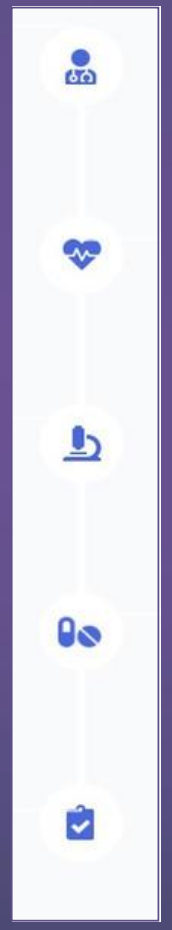
CIPN (chemotherapy-induced peripheral neuropathy):

- CIPN occurs in a substantial proportion of cancer patients, with estimates for incidence ranging from 19% to as high as 85%, depending on chemotherapy agent, dose, and assessment method.
- The highest rates—approximately 70%—are seen within one month of chemotherapy, with chronic symptoms persisting in about 41% of those initially affected.
- Market estimates for CIPN treatments in 2025 are between$1.06 billion and $2.5 billion, reflecting rapid growth (projected CAGR: 8–12%) driven by the large patient population, ongoing unmet needs, and increasing awareness.

Ketamir-2 Development Pathway Toward Phase IIa in CIPN

A strong foundation from clinical and Phase 1 data supports rapid advancement into neuropathy patients.

1. FDA IND USA IND obtained enabling Phase IIa trials
2. Phase 1 Mira-001 MAD study underway, completion Q4 2025
3. Phase IIa CIPN study initiation Q4 2025/Q1 2026 with MSK
4. Safety Endpoints AE/SAE frequency, vital signs, ECG monitoring
5. Patient Assessment Cognitive/CNS effects, daily pain via e-diary



Strategic Partnership Opportunity: Ketamir-2

Objective License, co-develop, or acquire Ketamir-2 to accelerate development in CIPN, neuropathic pain and unlock expansion across depression, PTSD and topical pain treatment.

Partner Value

- Access to > $120B global pain therapeutics market
- First-in-class oral, CNS-safe alternative with clear differentiation
- Phase IIa–ready in Q4 2025 with de-risked safety & PK profile
- Broad expansion potential into depression, PTSD, and topical inflammatory pain

Engagement Options

- Global licensing rights
- Co-development collaboration (shared costs & upside)
- Strategic acquisition opportunity

What is MIRA-55?

Key Differentiating Factors

THC

- Schedule 1, which means no accepted medical purpose
- Negative side effects
- Legal/regulatory hurdles Heightened competition
- Shipping/manufacturing issues

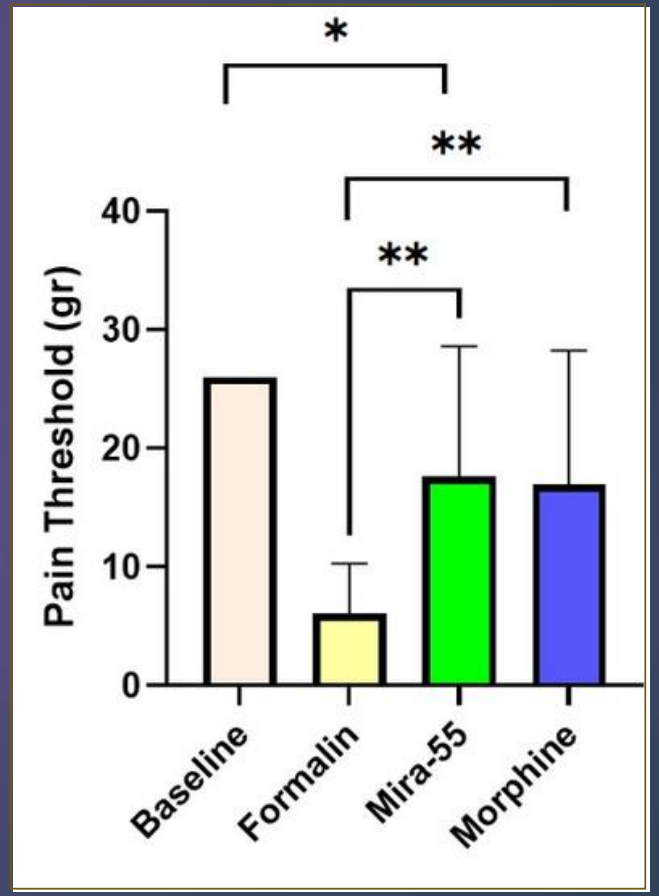
MIRA-55

- Pure Synthetic
- Based on animal model studies, better side effect profile (e.g. decreased anxiety across the dose range, improved rather than impaired cognition) Effective in inflammatory pain
- Being developed to be a prescription medication

MIRA-55 Reduces Inflammatory Pain

In the formalin test, rats treated with MIRA-55 had a lower sensitivity to pressure — a clear sign of reduced inflammatory pain

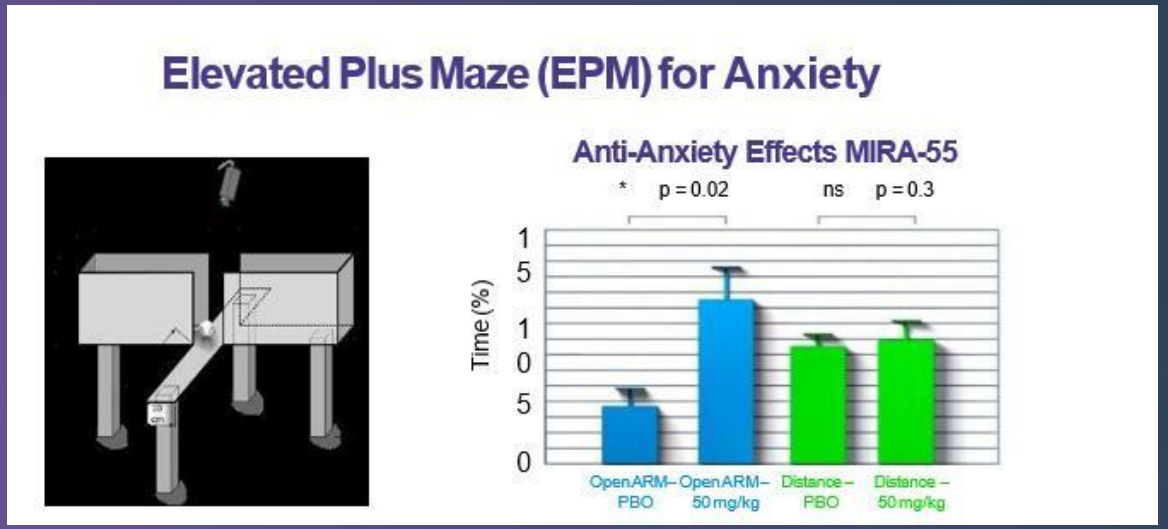
- Validated Model: The formalin test is a known inflammatory test in rodents
- Improved Behavior: MIRA-55 significantly reduced the sensitivity to pain after formalin treatment ($p < 0.01$)
- Similarity to Morphine: The effect was the same induced by morphine
- Clinical Relevance: Suggests potential as a treatment of inflammatory pain



MIRA-55 Reduces Anxiety Without Sedation or Intoxication

In the Elevated Plus Maze, mice treated with MIRA-55 spent more time exploring open areas — a clear sign of reduced anxiety — without showing signs of sedation.

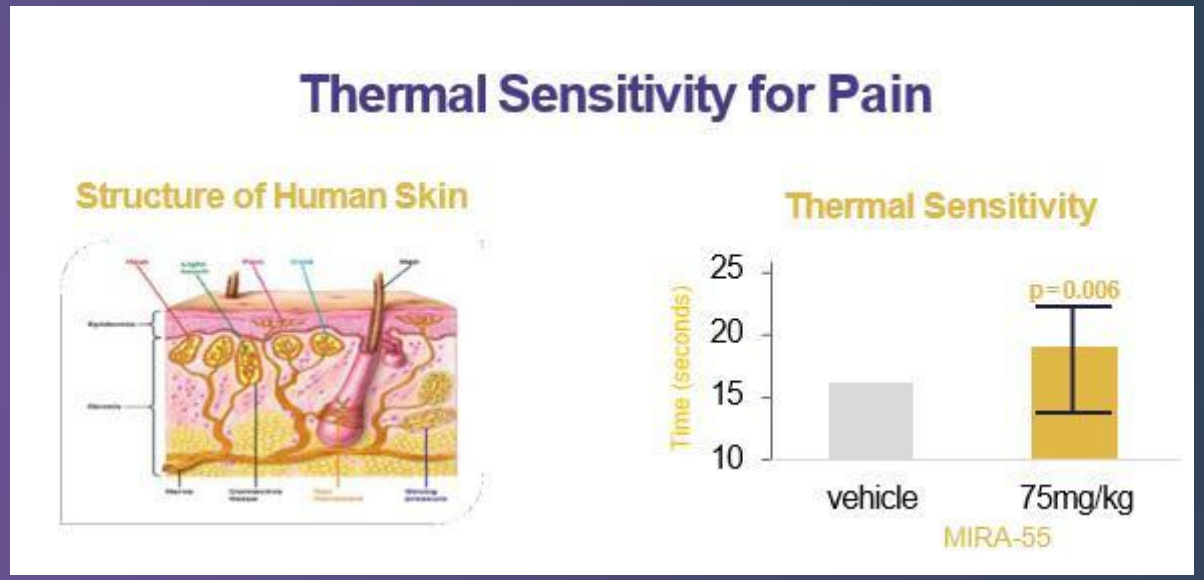
- Validated Model: The Elevated Plus Maze tests anxiety by measuring time spent in open vs. closed arms
- Improved Behavior: MIRA-55 significantly increased time in open arms vs. placebo (p = 0.02)
- No Sedation: Mice showed no decrease in mobility or exploratory activity
- Clinical Relevance: Suggests potential as a non-intoxicating treatment for anxiety disorders



MIRA-55 Shows Significant Pain Relief in Thermal Sensitivity Model

Mice treated with MIRA-55 took longer to react to heat — a clear sign of reduced pain sensitivity compared to placebo.

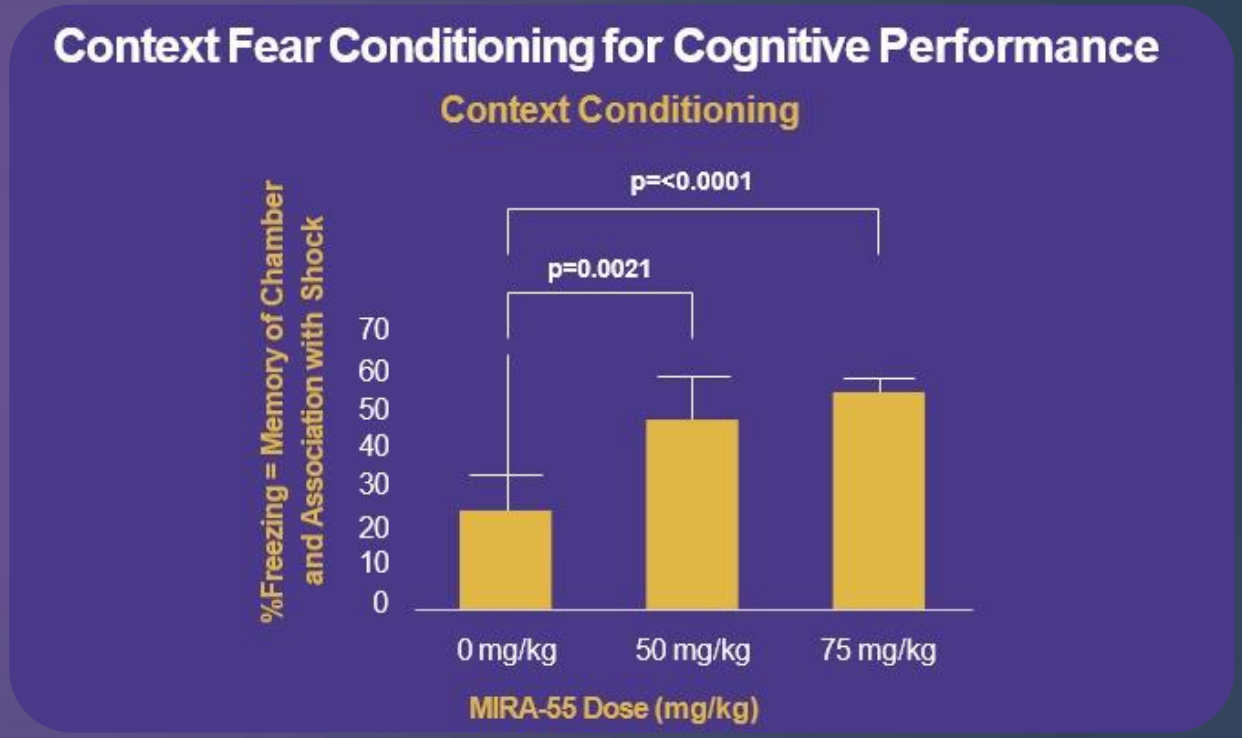
- Validated Model: Mice were placed on a heated plate and observed for paw-lift response time
- Reduced Pain Sensitivity: MIRA-55 increased response latency significantly (p = 0.006)
- Mechanism-Free Advantage: Relief occurred without sedation, intoxication, or opioid-like effects
- Therapeutic Implication: Supports MIRA-55's potential as a safe, non-opioid option for managing inflammatory or sensory pain



MIRA-55 Enhances Memory Recall in Contextual Fear Conditioning

In a validated cognitive model, MIRA-55 significantly improved memory performance — suggesting enhanced associative learning with no sedative tradeoffs.

- Model Overview: Mice were trained to associate a chamber with a mild shock; freezing behavior was used to assess memory
- Stronger Recall: MIRA-55 significantly increased freezing time vs. placebo ($p = 0.0021$, $p < 0.0001$)
- Unique Effect: Enhancement occurred independently of anxiety reduction — uncommon for cannabinoid-based compounds
- Cognitive Potential: Highlights MIRA-55's differentiated value as both an anxiolytic and cognitive enhancer



Regulatory Pathway to Commercialization

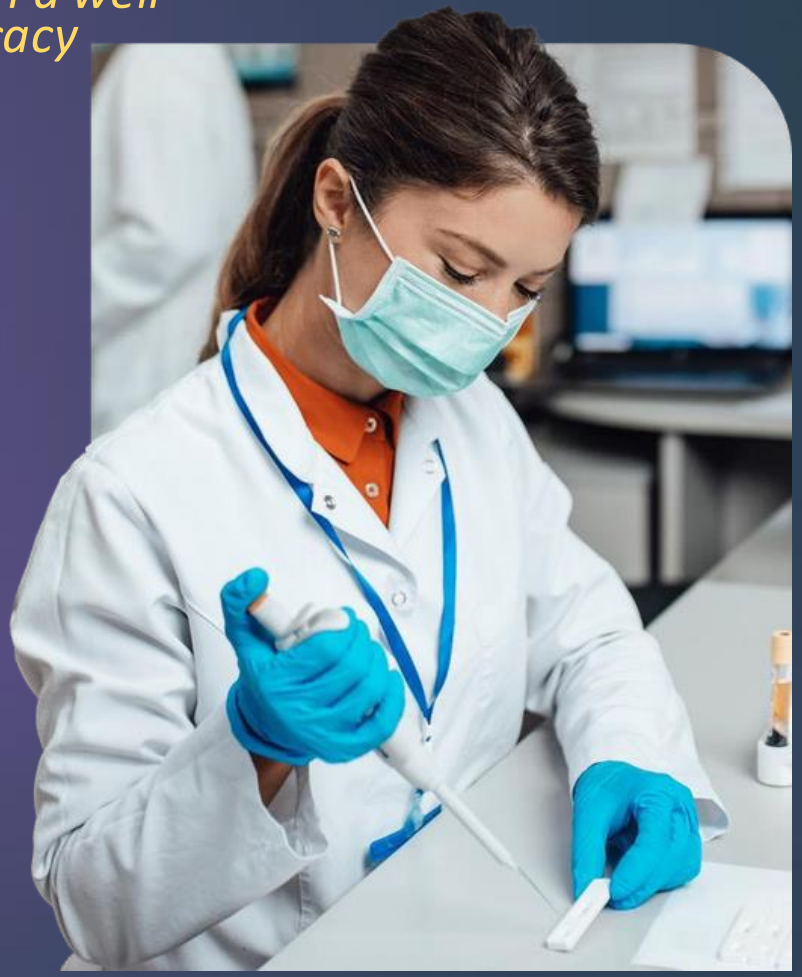
To develop, MIRA-55 as a commercialization asset, we are proceeding on a well-established regulatory pathway designed to establish its safety and efficacy

1

Clinical Testing
Focus on clinical testing including genetic toxicology, safety pharmacology and general toxicology testing to enable the filing of an IND application with the USFDA.

2

Clinical Trials
Clinical testing and trials based on guidance from FDA with a focus on our initial prioritized indications while preserving optionality to add 1 or 2 more indications with strategic partners.



A Next-Generation Approach to Obesity & Smoking Cessation

Unlocking metabolic and behavioral health innovation through SKNY-1

- Oral small molecule THCV analog with non-controlled status
- Multi-mechanistic profile: CB1 antagonist, CB2 partial agonist, MAO-B inhibition Designed to address obesity, smoking cessation, insulin resistance, and other metabolic disorders
- Differentiated safety and receptor bias avoids CNS liabilities of earlier CB1 drugs
- IND planned for Q3 2026, development-ready for strategic partnerships

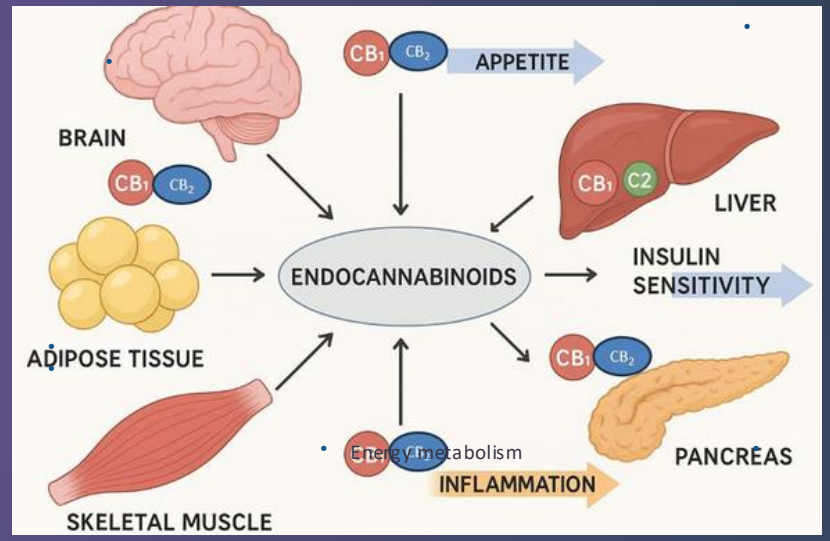
The Endocannabinoid System: A Master Regulator of Metabolism and Craving

CB1 and CB2 receptors influence appetite, insulin sensitivity, lipid metabolism, and reward — making them ideal targets for obesity and addiction.

- CB1 and CB2 receptors are found throughout the brain and peripheral tissues
- CB1 activity in the brain regulates appetite, craving, and reward
- CB1 in adipose tissue and muscle drives lipogenesis and energy metabolism
- CB2 is involved in immune modulation, inflammation, and insulin sensitivity
- Targeting both CB1 and CB2 offers a systemic strategy for treating obesity, diabetes, and addiction

Appetite Body Weight Reward

Insulin Regulation Lipid Levels



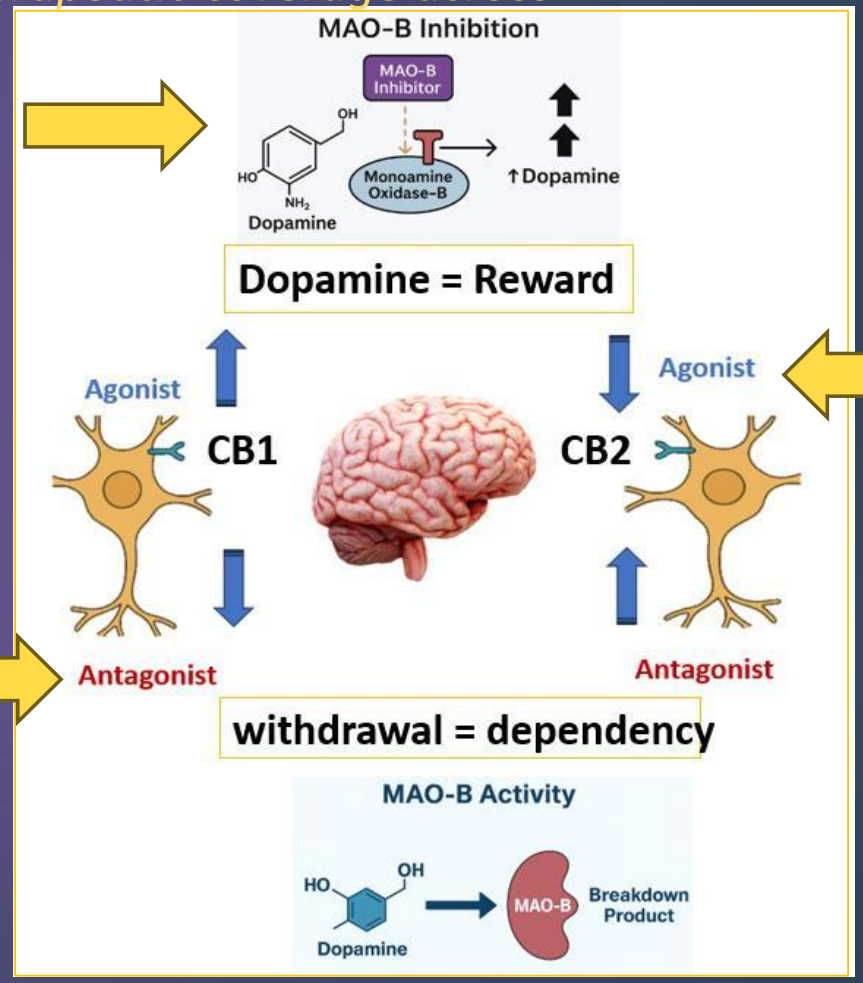
Lipogenes is Adipocyte size

Insulin Homeostasis

Multi-Target Mechanism to Address Complex Metabolic and Behavioral Conditions

SKNY-1's unique combination of activities enables broad therapeutic coverage across obesity, addiction, and metabolic dysregulation.

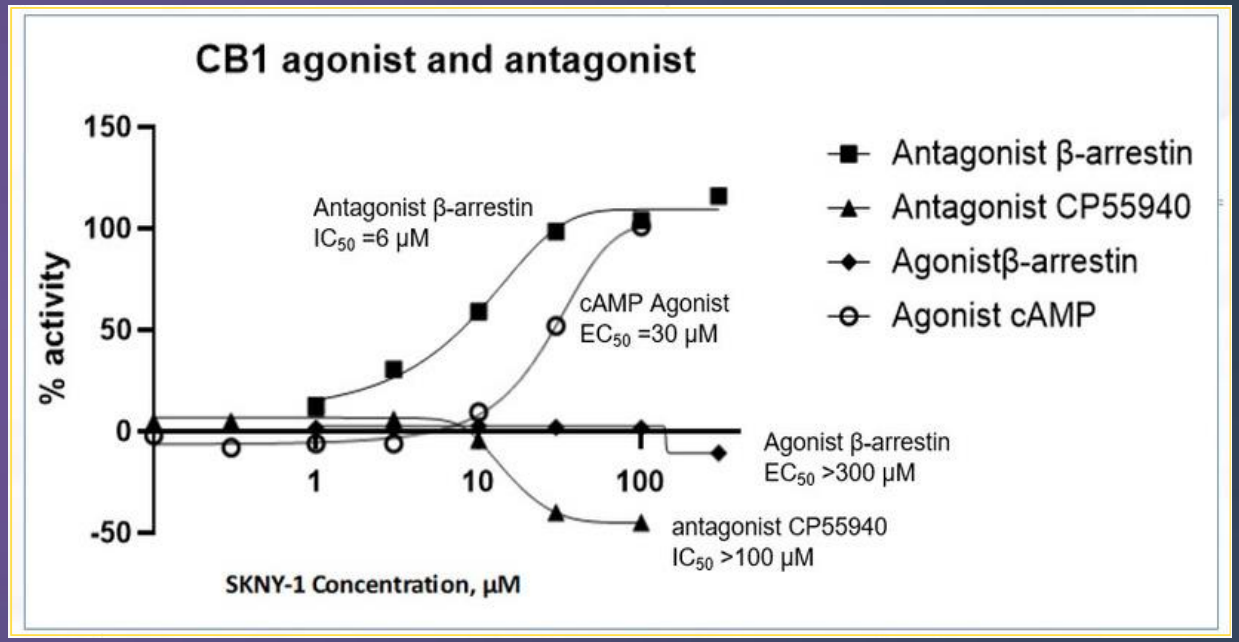
- CB1 Antagonism (biased) : Reduces appetite, body weight, and craving by dampening central reward signaling
- CB2 Partial Agonism: Modulates inflammation, improves insulin sensitivity, and reduces peripheral immune activation
- MAO-B Inhibition: Enhances dopamine tone to support mood and motivation without dopaminergic overstimulation
- NoCB1 Agonism or G-protein bias: Limits CNS liability, hallucinations, or psychomotor side effects



SKNY-1: A Low Potency, Biased CB1 Antagonist

SKNY-1 exhibits pathway-selective CB1 modulation, offering a differentiated mechanism versus traditional cannabinoid compounds.

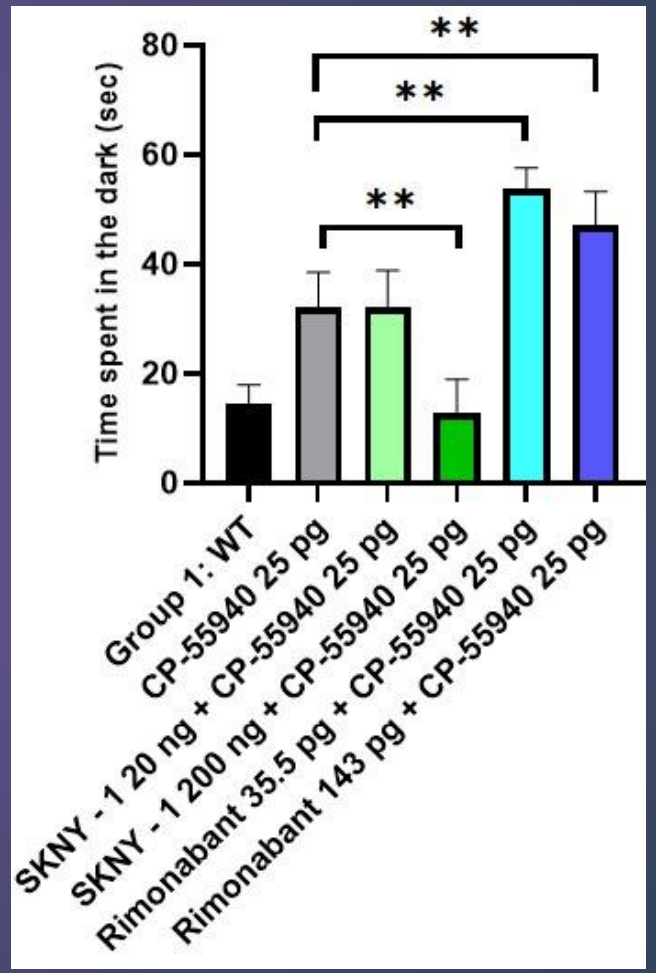
- No β-arrestin activation: SKNY-1 does not stimulate β-arrestin pathways, reducing potential side effects.
- G-Protein activity: Exhibits low-affinity agonist activity on cAMP signaling ($EC_{50} \approx 30$ µM).
- Selective β-arrestin antagonism: More potent as a β-arrestin antagonist ($IC_{50} = 6$ µM), suggesting pathway bias.
- No CB1 agonist antagonism: SKNY-1 does not interfere with standard CB1 agonist activity, preserving natural endocannabinoid signaling.



SKNY-1: A Low Potency, Biased CB1 Antagonist

SKNY-1 blocks CB1-agonist induced anxiogenic effect in Zebrafish, while Rimonabant potentiates it

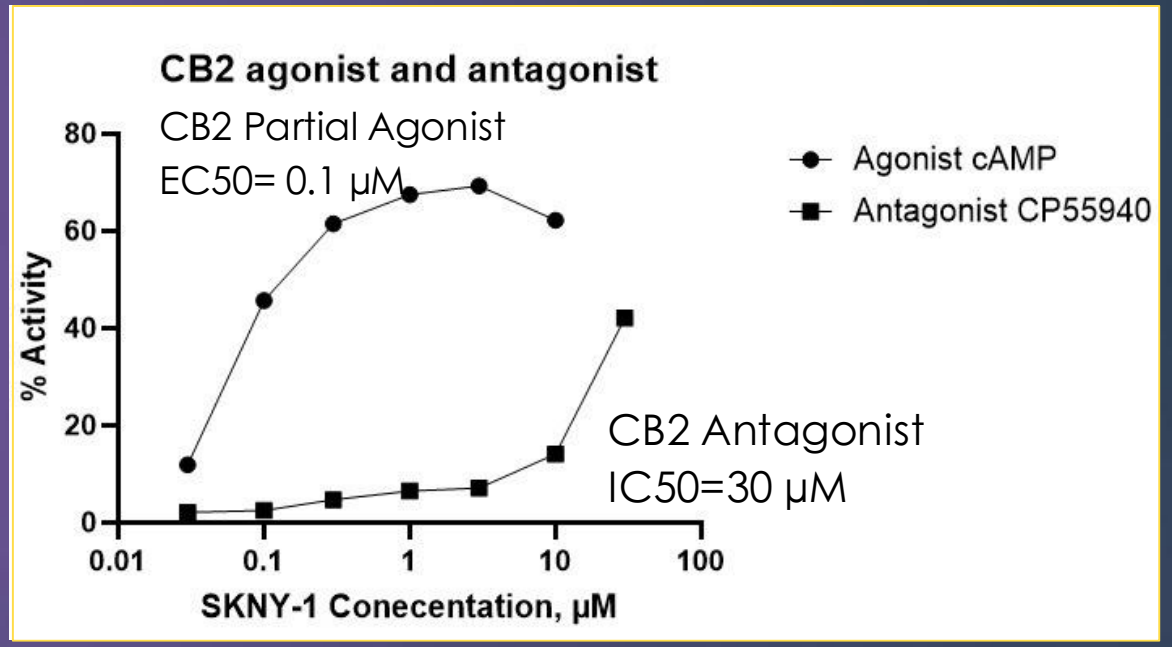
- CB1 antagonist activity: At high concertations, the CB1 agonist CP-55940 induces anxiogenic activity in zebra fish, expressed as an increase in the time spend in the dark compartment.
- SKNY-1 completely antagonizes this effect at the high dose used.
- Rimonabant further potentiates this effect of the CB1 agonist



SKNY-1: Tackling Obesity with a Differentiated Mechanism

Designed to modulate appetite and metabolism without the psychiatric baggage of first-generation CB1 blockers.

- Selective modulation of CB1 receptor without β-arrestin activation — may reduce CNS-related side effects
- Demonstrates partial agonist behavior at CB2 (~60% maximal effect)
- At higher concentrations, SKNY-1 shows CB2 antagonism — indicating a dose-dependent dual profile
- MAO-B inhibition (without affecting MAO-A) supports dopaminergic balance — key in appetite, craving, and addiction
- Combined activity offers a novel strategy for treating obesity, especially in patients with metabolic and behavioral complexity



Avoiding the Pitfalls of First-Generation CB1 Blockers

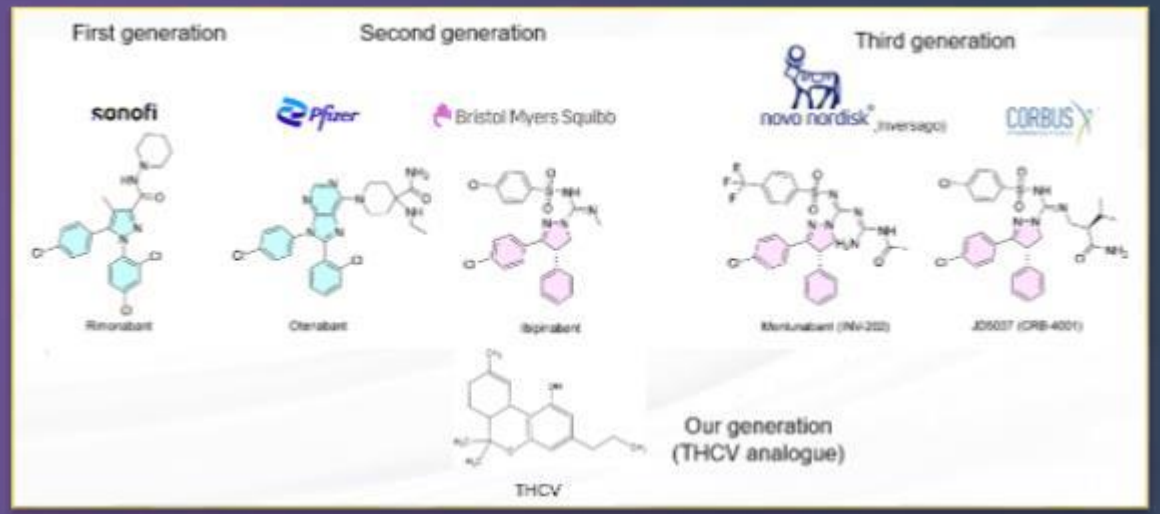
Designed to retain metabolic benefit while minimizing psychiatric burden

Earlier CB1 blockers like Rimonabant were effective in reducing weight and improving metabolic parameters — but failed due to CNS-related psychiatric side effects (e.g., depression, anxiety)

SKNY-1 avoids this fate through:

- No G-protein antagonism — avoids full CB1 shutdown Selective β-arrestin antagonism — preserves key metabolic signaling Peripheral action — limits CNS penetration and reduces psychiatric risk

- MRI-1891, a later-generation CB1 antagonist, also showed β-arrestin selectivity — but with greater G-protein antagonism and CNS liability compared to SKNY-1 SKNY-1 stands apart as a moderate β-arrestin antagonist with no G-protein antagonism and enhanced CB2 activity, positioning it as a next-generation metabolic modulator with improved safety margins



SKNY-1 represents a next-generation evolution in CB1/CB2 modulation — advancing the THCV class with improved safety and metabolic targeting.

MAO-B Inhibition: Enhancing Motivation and Reducing Craving

SKNY-1 selectively and weakly inhibits MAO-B — supporting dopaminergic tone with minimal CNS risk.

Low-Affinity, Selective MAO-B Inhibition:

- SKNY-1 inhibits MAO-B (EC_{50} ≈ 300 µM) with minimal effect on MAO-A (EC_{50} > 1000 µM), avoiding off-target serotonergic effects

Supports Dopamine Balance:

- Sustained dopamine levels may enhance motivation, reduce craving, and improve mood without overstimulation

Relevance for Addiction:

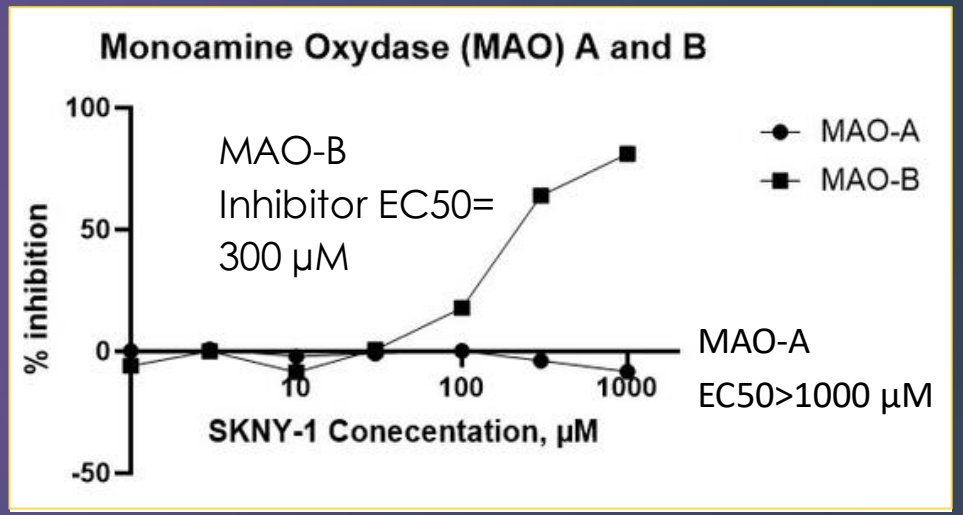
- MAO-B modulation is linked to reduced nicotine and reward-driven behaviors, making SKNY-1 a strong candidate for smoking cessation

Minimizes CNS Liabilities:

- Selective inhibition avoids the adverse events associated with non-selective MAO inhibitors

Integrative Mechanism:

- Enhances SKNY-1's CB-targeting strategy by adding dopaminergic support for metabolic and behavioral indications



Preclinical Profile of SKNY-1: Concentration-Dependent Pharmacology

SKNY-1 exhibits distinct activity across CB1, CB2, and MAO-B targets depending on dose level, supporting its role as a multifunctional, biased modulator.

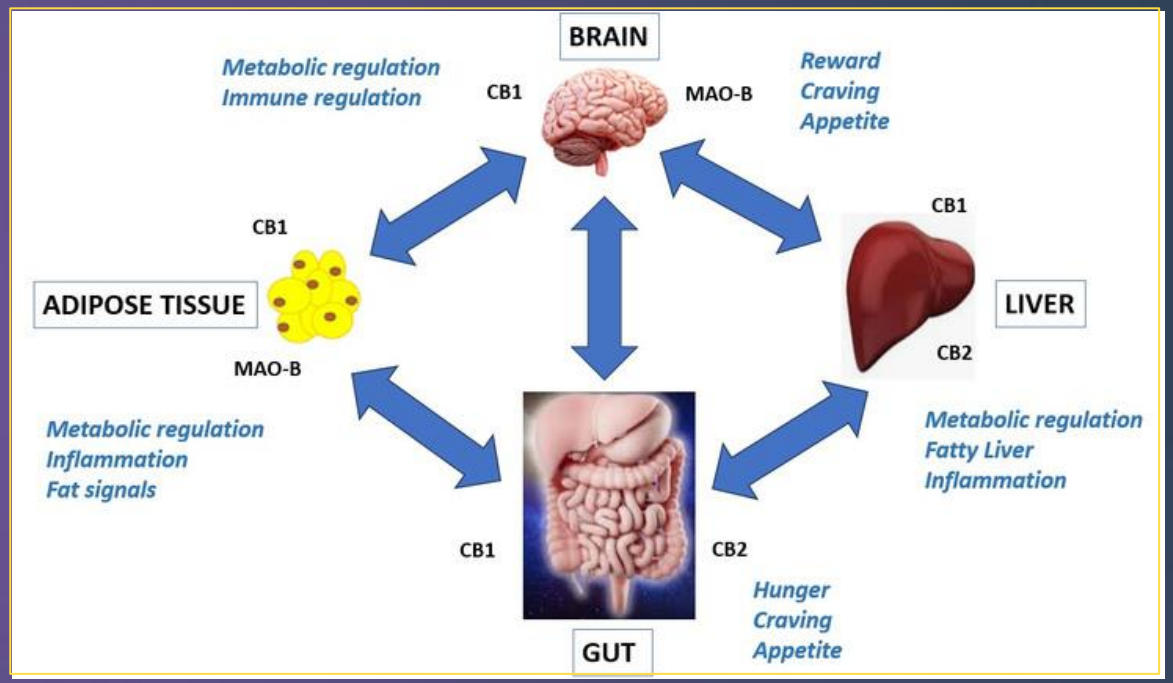
- CB2 Agonism: Strong activation at low concentrations (1–10µM), with reduced partial agonist activity at mid-range doses.
- CB1 β-arrestin Antagonism: Potent, pathway-selective antagonism observed at 10–100µM, maintained at higher levels.
- CB2 Antagonism: Emerges at moderate concentrations, indicating dual modulatory behavior.
- MAO-B Inhibition: Minimal at low doses; significantly increases at high concentrations (up to +++ at 100– 1000 µM).
- Selectivity: No inhibition of MAO-A, reducing potential for off-target effects.

Concentration range	Low (1-10µM)	Medium (10-100µM)	High (100-1000µM)
CB2 Agonist	+++	+-	
CB1 β-arrestin Antagonist	++	+++	++
CB2 Antagonist		+-	
MAO-B inhibition		+	+++

SKNY-1's Multi-Tissue Action: A Coordinated Strategy for Obesity and Addiction

A single compound influencing central and peripheral targets — uniting metabolic, behavioral, and inflammatory control.

- Brain: Modulates CB1 and inhibits MAO-B, impacting appetite regulation, reward signaling, and dopaminergic tone Liver & Adipose
- Tissue: Affects CB1 and CB2 to regulate fat storage, lipid metabolism, and reduce inflammation
- Gut: CB1 and CB2 activity influence hunger cues, cravings, and appetite suppression
- Systemic Integration: SKNY-1's profile links central mechanisms with downstream effects in peripheral tissues
- Therapeutic Implication: A promising candidate form ultifactorial conditions like obesity, metabolic syndrome, and substance dependence

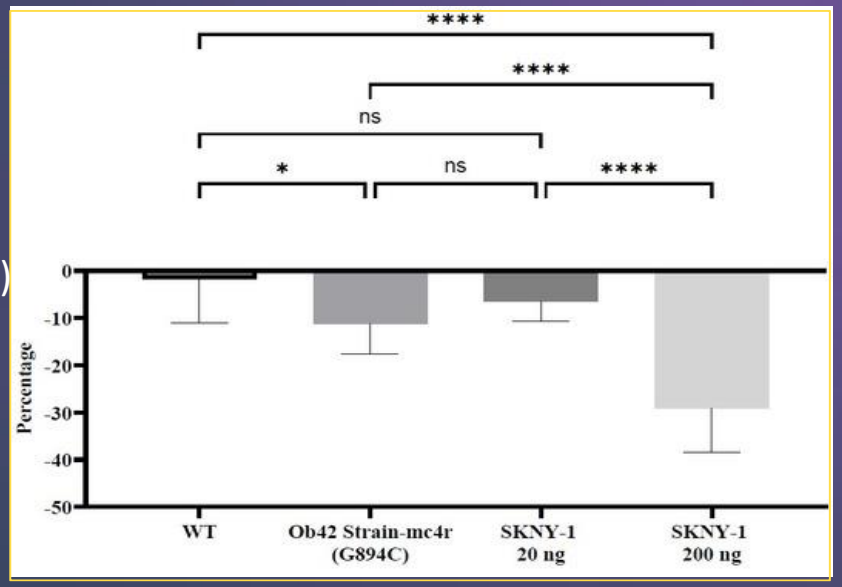


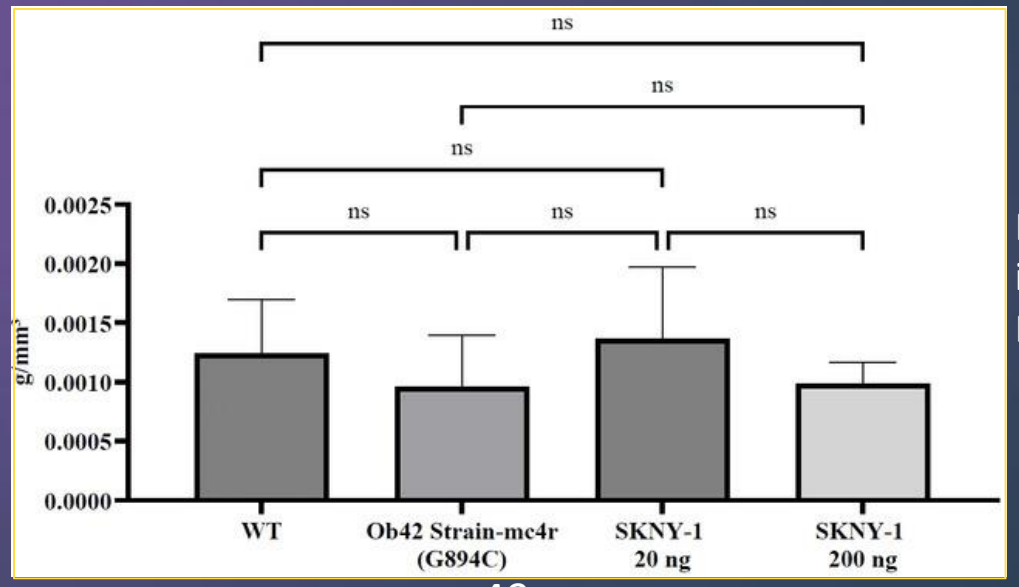
Obesity and Craving Model in Zebra Fish

Body weight changes without muscle density changes

- SKNY-1 was recently studied in an obesity and craving model in Zebra fish in collaboration with Pentagrit using MC4R strain -ObZebrafish [Ob42 Strain-mc4r(G894C) was used
- Adult Zebrafish have been exposed orally to the test compounds for 6 days followed by screening on day 7
- A marked decrease in body weight is observed with the high dose (200 ng)
- No significant changes in body density (indicative of muscle mass) was observed

Reduction in Body weigh (%)





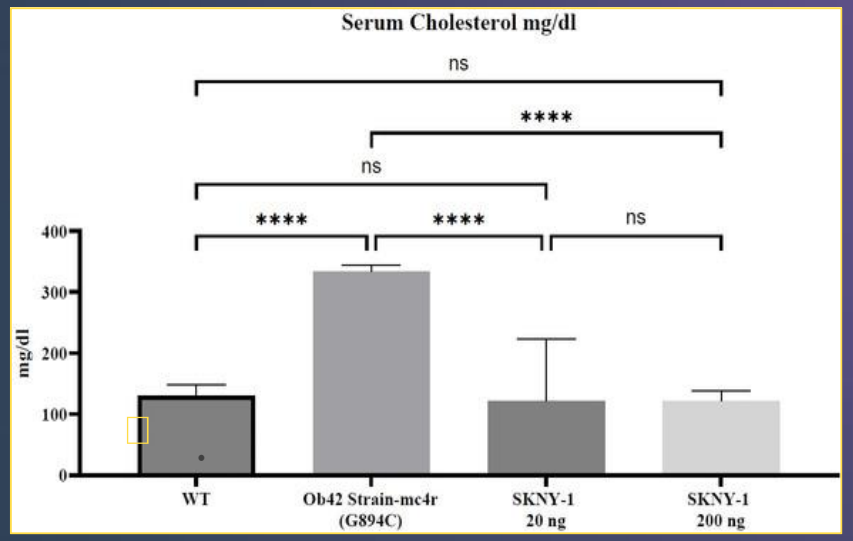
No Changes in Muscle Density

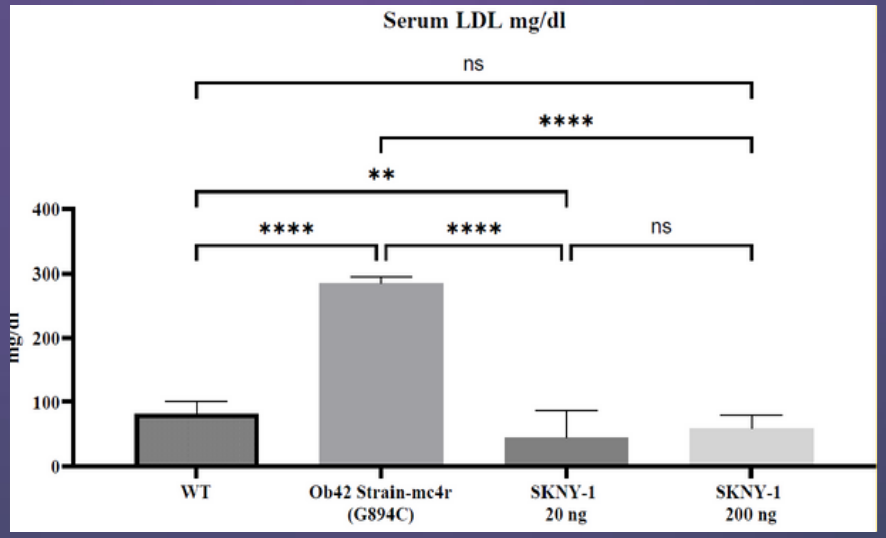
Obesity and Craving Model in Zebra Fish

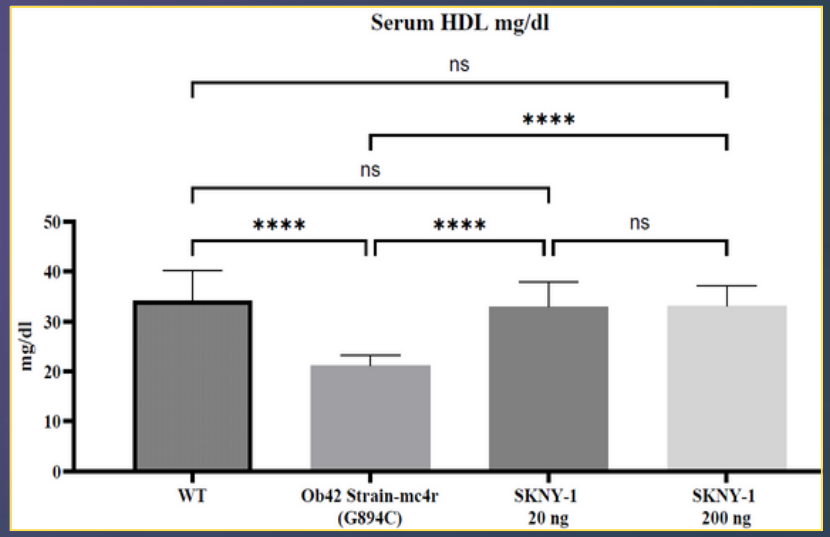
Cholesterol regulation

- At both studied doses, a marked decrease in cholesterol and LDL levels and a marked increase in HDL levels were observed.

- These changes brought the respective levels towards wild-type levels





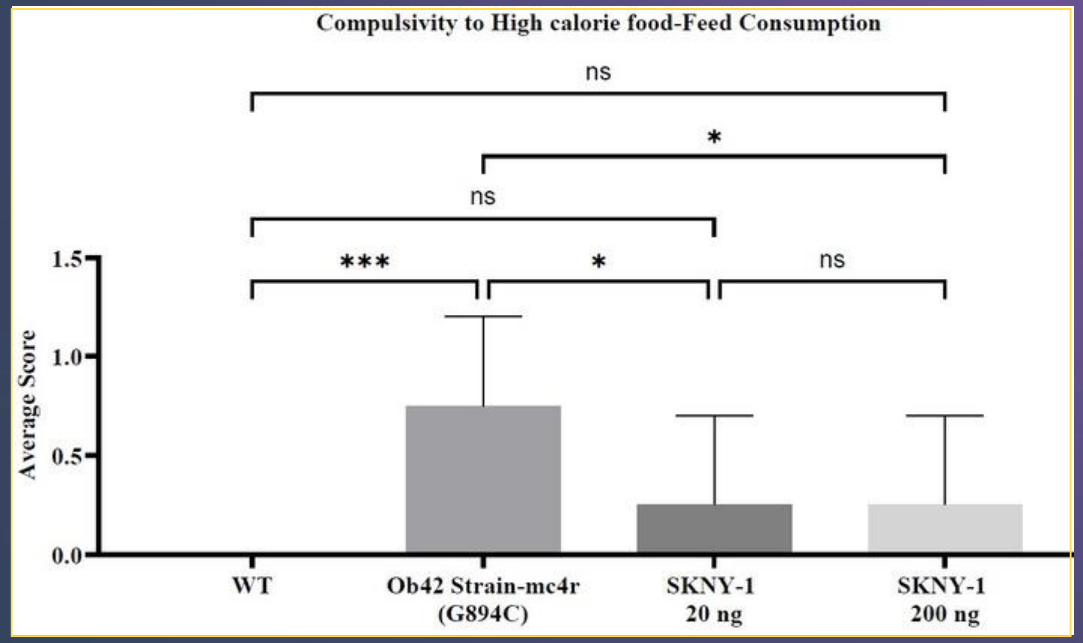


Obesity and Craving Model in Zebra Fish

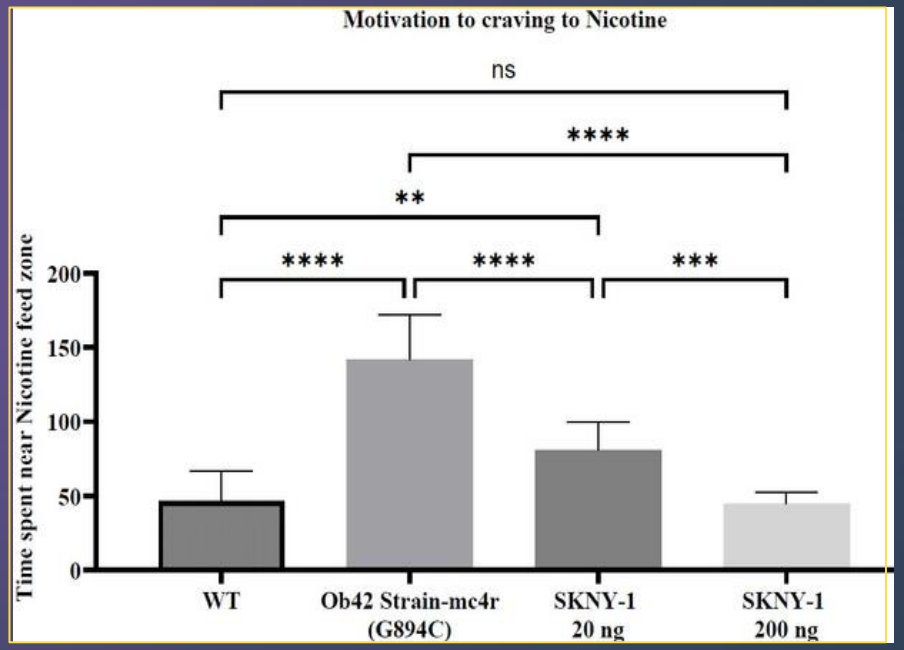
Appetite and craving for food and nicotine regulation

- Several parameters related to high calorie food and nicotine craving were evaluated.
- A marked, dose dependent decrease of high calorie and nicotine craving was observed, with highly significant decrease at the high dose

Reduction of High Calorie Food Craving



Reduction of Nicotine Craving



Advancing SKNY-1 Toward Strategic Partnership and Clinical Validation

Differentiated science, druggable profile, and commercial opportunity converge in SKNY-1

- Highly differentiated THCV-analog small molecule with CB1/CB2/MAO-B selectivity Multi-mechanistic action addresses both metabolic and behavioral drivers of obesity and addiction Peripheral selectivity and receptor bias avoid CNS risks that limited prior-generation CB1-targeting drugs
- Validated targets with strong precedent in large markets (e.g., rimonabant, Monlunabant)
- Clinical package supports IND in 2026 with optionality for metabolic, smoking cessation, or CNS pathways
- Positioned for partnering to advance SKNY-1 into the clinic and unlock value in multiple indications

KETAMIR-2 IP PROTECTION

Intellectual property protection for KETAMIR-2

- Worldwide patent protection is pending for Ketamir-2 and its therapeutic uses
- WO 2024/191676 A1, “Antidepressant Compounds, Pharmaceutical Compositions, and Methods of Treating Depression and Other Disorders”
- MIRA is the exclusive licensee of patent rights to Ketamir-2 and its therapeutic uses in the US, Canada and Mexico, for which corresponding national stage applications will be filed in September 2025

Investment Highlights

KETAMIR-2

Ketamir-2 is a novel oral ketamine analog with improved bioavailability, CNS selectivity, and a clean safety profile.

- Demonstrated potent activity in validated models of neuropathic pain, CINP, depression, and anxiety
- No psychomimetic effects, no hyperlocomotion, and minimal off-target activity
- Currently in Phase 1 trials with ongoing MAD portion
- Good oral bioavailability and activity far exceeding ketamine's oral/intranasal performance
- Not a DEA-controlled substance — favorable for regulatory and commercial pathways

MIRA-55

MIRA-55 is a THC analog designed to treat inflammatory pain, reduce anxiety, and enhance cognition — without THC-like intoxication.

- Demonstrated potent anti-inflammatory pain relief, reduced anxiety, and improved memory
- Acts without sedation or euphoria — a rare behavioral profile among cannabinoid analogs
- Addresses a large and underserved population suffering from anxiety, cognitive decline, and chronic inflammatory pain
- Non-scheduled compound — enabling streamlined development for age-related CNS and pain indications

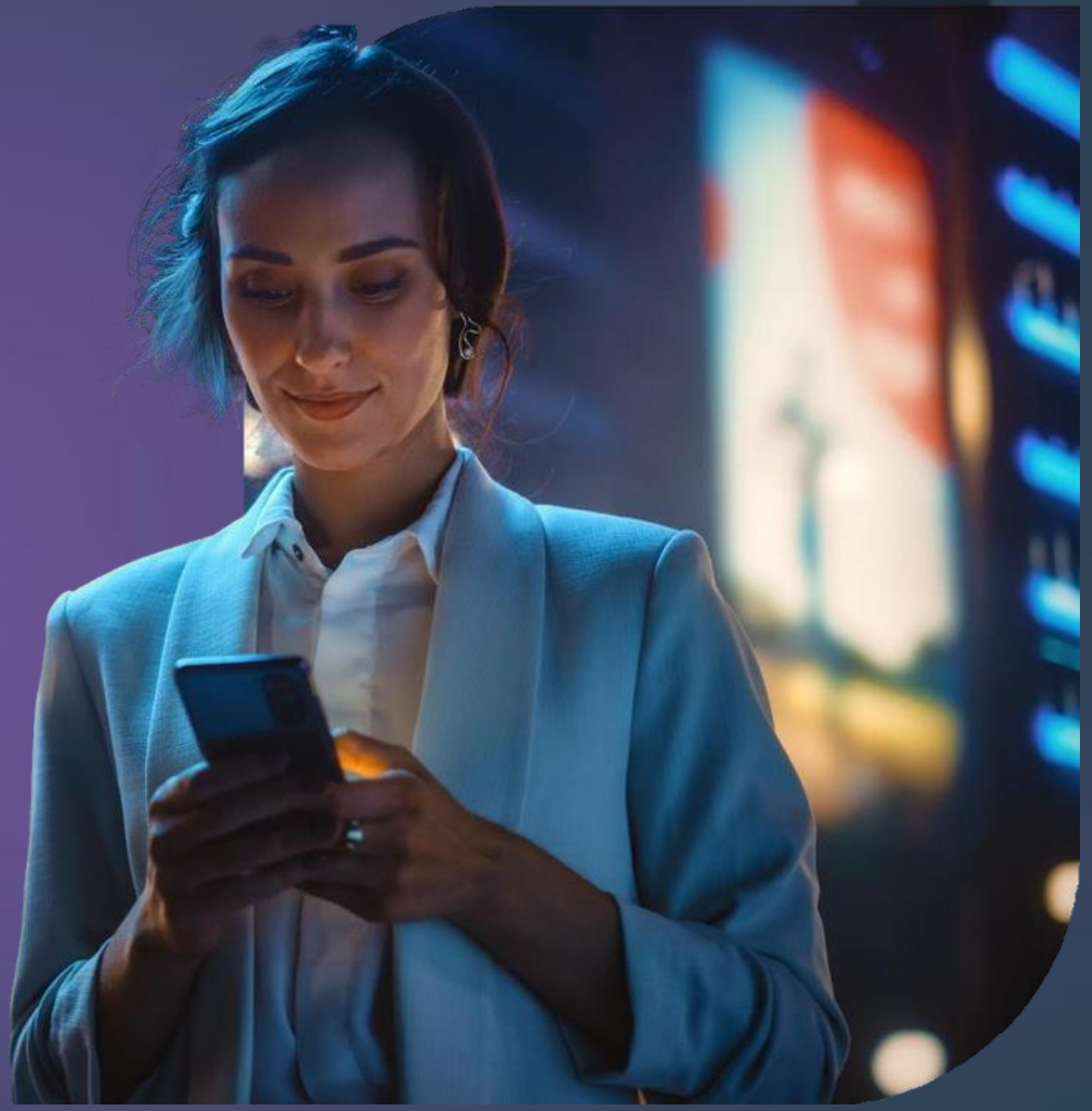
SKNY-1

SKNY-1 is a THCV analog with CB1 antagonist, CB2 partial agonist, and MAO-B inhibition activity — targeting metabolic and behavioral disorders.

- Designed to treat obesity, smoking cessation, and Metabolic syndrome
- Demonstrates peripheral selectivity and receptor bias — avoiding psychiatric side effects seen in earlier CB1-targeting drugs
- Distinct from Rimonabant and MRI-1891: No G-protein antagonism, only moderate β-arrestin modulation
- IND expected in Q3 2026, with strong partnering potential for metabolic or addiction pipelines

MIRA Pharmaceuticals

Thank you.



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