

solvonisTHERAPEUTICS

LSE: SVNS

A clinical-stage psychedelics company developing therapeutics for addiction, PTSD and depression

An alcohol use disorder franchise across the UK, Europe and the U.S.
A de novo engine of proprietary new medicines.

Corporate presentation
October 2026



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KEY CHARACTERISTICS

Late-stage clinical assets, scientifically de-risked, with near-term milestones

01

Late-stage clinical assets

SVN-001 is in Phase 3 in severe alcohol use disorder (AUD), recruiting in the UK, with Portuguese sites being assessed. SVN-002, an esketamine oral thin film (OTF), has an FDA-agreed 505(b)(2) route to a U.S. Phase 2b.

02

Scientifically de-risked

Built on ketamine and esketamine, with established pharmacology and an approved precedent in SPRAVATO®. The published SVN-001 Phase 2a/b met one of its two co-primary endpoints. The FDA confirmed that no additional clinical data are needed before the SVN-002 Phase 2b.

03

Near-term milestones

SVN-015: further studies funded and conducted by the U.S. National Institute on Drug Abuse (NIDA). SVN-002: Investigational New Drug (IND) submission for the U.S. Phase 2b, planned for 2027. Non-dilutive funding being pursued for SVN-114 (U.S.) and SVN-015 in depression (UK).

04

Attractive entry point

Pharma is paying for psychedelic and rapid-acting neuropsychiatry assets: Lilly, \$2.8bn plus up to \$1.0bn for AtaiBeckley (2026); Otsuka, \$700m plus up to \$525m for Transcend's Phase 3 post-traumatic stress disorder (PTSD) asset (2026).

SVN-001, SVN-002, SVN-015 and SVN-114 are investigational; their safety and efficacy have not been established. FDA feedback is not an approval or a guarantee of any outcome. Timings are indicative and subject to the availability of capital, regulatory feedback and trial progress. Transactions are sector context, not valuation comparables. Sources: Grabski et al., Am J Psychiatry 2022;179(2):152–162; Solvonis RNS, 21 January 2025 and 13 October 2026; Eli Lilly, 11 September 2026; Otsuka, 12 June 2026; Company information.

WHY NOW

Pharma is paying for psychedelic and rapid-acting neuropsychiatry assets

Date	Acquirer	Asset / company	Transaction value	Clinical focus
SEP 2026	Eli Lilly	AtaiBeckley	\$2.8bn + up to \$1.0bn	Psychedelic-derived therapies; lead in depression
JUN 2026	Otsuka	Transcend (methyllone)	\$700m + up to \$525m	Phase 3 PTSD asset
OCT 2025	AbbVie	Gilgamesh (bretisilocin)	up to \$1.2bn	Phase 2 major depressive disorder (MDD) asset

Pharma has paid billions for depression and PTSD. Solvonis has programmes in both, and a Phase 3 ketamine-based treatment in alcohol use disorder (AUD), where only 1 in 6 people receive treatment.

Sources: Eli Lilly, 11 September 2026 (completion) and 16 July 2026 (terms); Otsuka, 12 June 2026; AbbVie, 17 October 2025. Values include contingent consideration where stated. Dates are completion dates. Sector context; not valuation comparables.

STRATEGY

A clinical-stage psychedelics company with two complementary development strategies

Scientific leadership: Professor David Nutt, Chief Scientific Officer, and Edmond J Safrá Professor of Neuropsychopharmacology at Imperial College London

Value-added medicines: the AUD franchise

Differentiated ketamine- and esketamine-based treatments built on established pharmacology, clinical experience and regulatory precedent.

SVN-001 | Phase 3 in severe alcohol use disorder (AUD) in the UK; EU sites proposed

SVN-002 | FDA-agreed 505(b)(2) route, with a scientific bridge to SPRAVATO®; esketamine OTF in Phase 2b planning for moderate-to-severe AUD (U.S.)

VALUE CREATION | Nearer-term clinical and regulatory catalysts

De novo drug development: the engine

Creating proprietary new chemical entities (NCEs) with composition-of-matter filings in high-burden addiction and psychiatric disorders.

SVN-015 StUD | A new stimulant use disorder candidate; encouraging initial NIDA screening, advanced into further NIDA-funded studies

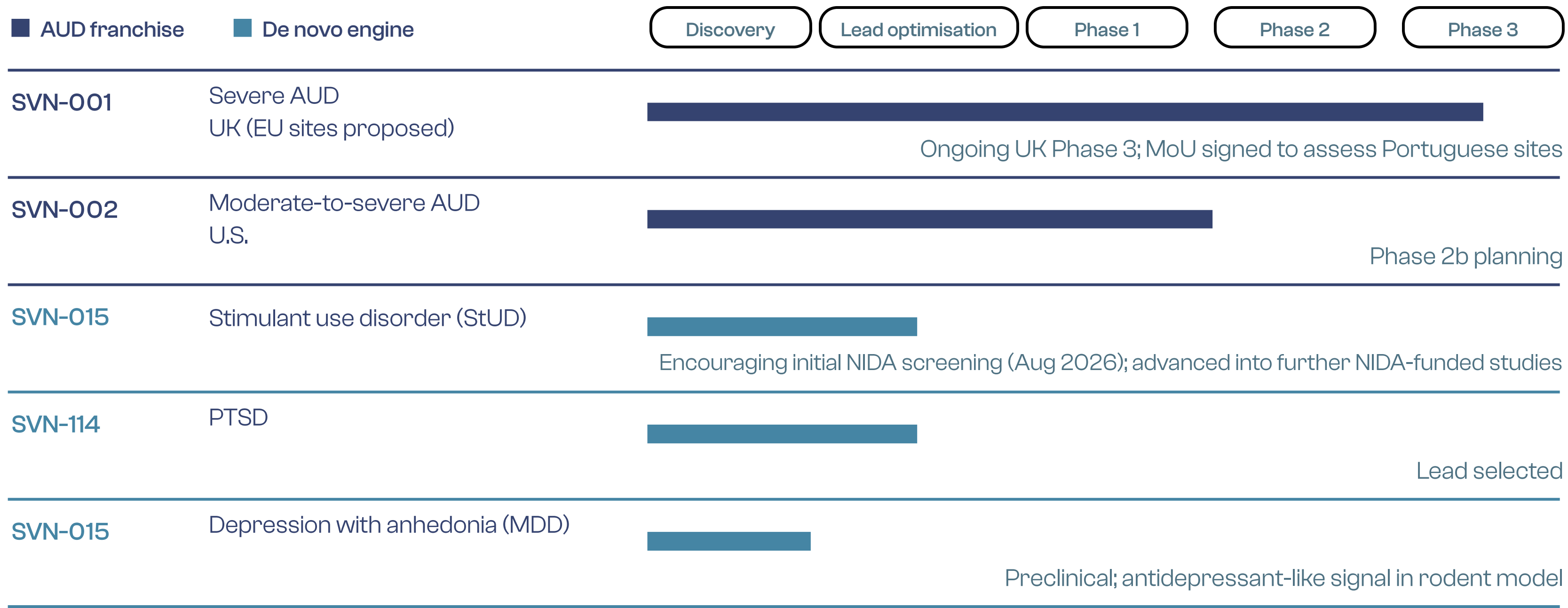
SVN-114 | A preclinical entactogen for PTSD

SVN-015 MDD | Depression with anhedonia

VALUE CREATION | Longer-duration IP and early-stage upside

PIPELINE

Five programmes: an alcohol use disorder franchise and a de novo engine



Sources: Company programme information; UK Health Research Authority (HRA), MORE-KARE study summary; Solvonis announcements dated 6 August, 25 August and 13 October 2026. MoU: memorandum of understanding with ICAD (Portugal's public addiction institute) and Panegreen (Lisbon clinical partner) to assess Portuguese trial sites. Status as at 13 October 2026.

CATALYST ROADMAP

Where each programme stands and what comes next

All milestones and timings are prospective, and subject to the availability of capital, regulatory feedback and trial progress.

Programme	Delivered (as at 13 Oct 2026)	Next milestone	Indicative timing
SVN-001	UK Phase 3 recruiting (280 planned); MoU signed to assess Portuguese sites (Oct 2026)	Phase 3 readout; EU site expansion (proposed)	2029–30 Readout (indicative)
SVN-002	Positive preclinical bridging data (Jun 2026)	Further FDA engagement; IND submission	2027 IND for the U.S. Phase 2b
SVN-015 StUD	Encouraging initial NIDA screening; advanced into further NIDA-funded studies (Aug 2026)	NIDA confirmatory dopamine transporter (DAT) studies; mouse locomotor study; 5-HT ₂ B follow-up	To be announced
SVN-114	Lead selected (Mar 2026)	Non-dilutive funding (U.S.); dose response, pharmacokinetics and brain exposure	To be announced
SVN-015 MDD	Antidepressant-like signal in a mouse model (Jan 2026)	Non-dilutive funding (UK); anhedonia-specific models	To be announced

Indicative Company planning dates as at 13 October 2026, subject to the availability of capital, regulatory feedback and trial progress.
Sources: ISRCTN85955128; Ulysses Neuroscience UNL094; Solvonis RNS, 21 January 2025, 3 December 2025, 28 January 2026, 10 March 2026, 22 June 2026, 6 August 2026, 25 August 2026 and 13 October 2026; Company data on file.

AUD is a large, underserved market: worldwide, only 1 in 6 people with AUD receive treatment

UK AND EU4 | SEVERE AUD

12.5m

people aged 15+ with severe AUD (Company estimate)

UK 2.6m | EU4 9.9m

SVN-001 is developed for severe AUD

UNITED STATES | MODERATE-TO-SEVERE AUD

11.3m

people aged 12+ with past-year moderate-to-severe AUD, 2024

SAMHSA NSDUH: 6.0m moderate + 5.4m severe

SVN-002 is developed for moderate-to-severe AUD

Sources: Company estimates (July 2026) of DSM-5 severe AUD, ages 15+: WHO Europe 5.9% alcohol-dependence prevalence × 75.3% ICD-10-to-DSM-5 severe-AUD crosswalk (Degenhardt et al., Addiction 2019) × Eurostat 2024 and ONS mid-2024 population (EU4 = France, Germany, Italy, Spain). U.S.: Substance Abuse and Mental Health Services Administration (SAMHSA), 2024 National Survey on Drug Use and Health (NSDUH), Tables 5.2A and 5.2B, past-year moderate-to-severe AUD, ages 12+, civilian non-institutionalised. Treatment rate: Mekonen et al., Addiction 2021. Epidemiological estimates, not treated or addressable patients; thresholds differ by region, so the two figures are not additive. U.S. components rounded.

Building an AUD franchise: SVN-001 in the UK and Europe, SVN-002 in the U.S.

East and west: one mechanism, two products, two regulatory pathways

	SVN-001 UK / EUROPE	SVN-002 U.S.
PRODUCT	Ketamine IV + proprietary in-clinic relapse-prevention therapy	Esketamine oral thin film (OTF) + at-home digital psychosocial support
PATIENT	Severe AUD	Moderate-to-severe AUD
TREATMENT	Three IV ketamine sessions + in-clinic relapse-prevention therapy	Three esketamine OTF sessions + at-home digital psychosocial support
STATUS	UK Phase 3 ongoing; MoU signed to assess Portuguese trial sites	Phase 1 and placebo-controlled study in 28 problem drinkers completed
REGULATORY	Full mixed application strategy	505(b)(2) pathway agreed with FDA, with a scientific bridge to SPRAVATO®

Sources: UK Health Research Authority, MORE-KARE research summary (IRAS 1008179; ISRCTN85955128); Solvonis RNS, 21 January 2025; Gent et al., J Psychopharmacol 2024; Company data on file. SPRAVATO® is a trademark of Johnson & Johnson. 505(b)(2): an FDA pathway that can draw on data for an already-approved drug. Detail: slide 33.

How ketamine-based treatment works: it opens a window of plasticity, and psychosocial support is designed to work inside it

FROM NMDA BLOCK TO NEW LEARNING

- 01 Blocks NMDA receptors on inhibitory interneurons**
Releases the brake on excitatory neurones: a transient surge of glutamate.
- 02 Activates AMPA receptors and BDNF–mTOR signalling**
The glutamate surge switches on the pathways that drive synaptic plasticity.
- 03 Builds new synaptic connections**
New synaptic proteins, dendritic spines and stronger excitatory synapses form in the prefrontal cortex.
- 04 Opens a window of plasticity**
The cue–reward associations that maintain addiction become more open to change.

WHAT THIS MEANS IN AUD

Stronger prefrontal control

over limbic urges and cravings

Weakening of drug memories

reported in hazardous drinkers when ketamine follows memory retrieval (Das et al., 2019)

Lower negative affect

through ketamine's rapid antidepressant effect

THE COMBINATION

Ketamine opens the window; in-clinic relapse-prevention therapy uses it to build healthier responses to urges, triggers and relapse risk. The integrated treatment addresses both the biological and psychosocial dimensions of severe AUD. In SVN-002, at-home digital psychosocial support is designed to play the same role.

Sources: Das et al., Nat Commun 2019;10:5187; Grabski et al., Am J Psychiatry 2022;179(2):152–162. The mechanism summarises current understanding of ketamine pharmacology; the pathway in AUD remains under investigation. NMDA: N-methyl-D-aspartate; AMPA: alpha-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid; BDNF: brain-derived neurotrophic factor; mTOR: mammalian target of rapamycin. Esketamine, the active ingredient of SVN-002, is the S-enantiomer of ketamine and acts at the same NMDA receptor; detail: slide 33.

SVN-001 Phase 2a/b: a completed, academic-led, investigator-initiated trial in 96 patients with severe AUD

FOUR ARMS, 96 PATIENTS



96

adults with severe AUD, recently detoxified and abstinent at entry; randomised 1:1:1:1 (166 screened)

WHO RAN IT

Investigator-initiated trial: academic investigators at two UK sites (Exeter and London), funded by the Medical Research Council

A proof of concept, not designed for registration; Solvonis is now progressing SVN-001 in Phase 3

TREATMENT

Three 40-minute IV infusions, 1 to 3 weeks apart: ketamine 0.8 mg/kg or saline

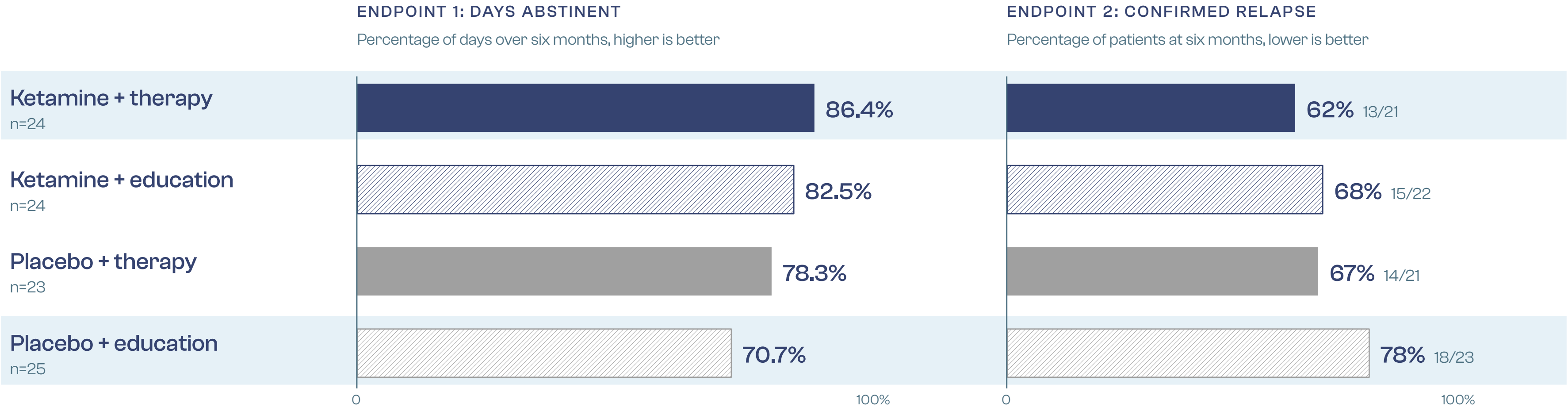
Seven 1.5-hour sessions of relapse-prevention therapy or alcohol education, timed around each infusion; follow-up to six months

ENDPOINTS, SIX MONTHS

- 1 Percentage of days abstinent: days with no alcohol (self-reported, Timeline Followback)
 - 2 Confirmed relapse: one or more heavy drinking days (more than 64.8 g a day for men, 52.0 g for women)
- Primary comparison: ketamine versus placebo, pooled across therapy arms

Source: Grabski et al., Am J Psychiatry 2022;179(2):152–162 (design, funding statement, Figures 1 and 2). Trial name: KARE (Ketamine for Reduction of Alcohol Relapse); NCT02649231, EudraCT 2015-000222-11; recruitment September 2016 to July 2019. Heavy drinking thresholds as defined in the paper; reporting time capped at 180 days.

What was published: one of two co-primary endpoints met, with more days abstinent on ketamine



Shaded rows: the two regimens Phase 3 tests. Its endpoint is heavy drinking days (next slide).

Source: Grabski et al., Am J Psychiatry 2022;179(2):152-162 (Table 2; Table S1). 96 adults, 2x2 design; arm means over six months, observed data. Confirmed relapse = one or more heavy drinking days within 180 days (n/N shown; only patients with at least 159 days of data). The co-primary analysis compared all ketamine with all placebo patients: days abstinent was met; confirmed relapse was not. The trial was not powered for arm-by-arm comparison.

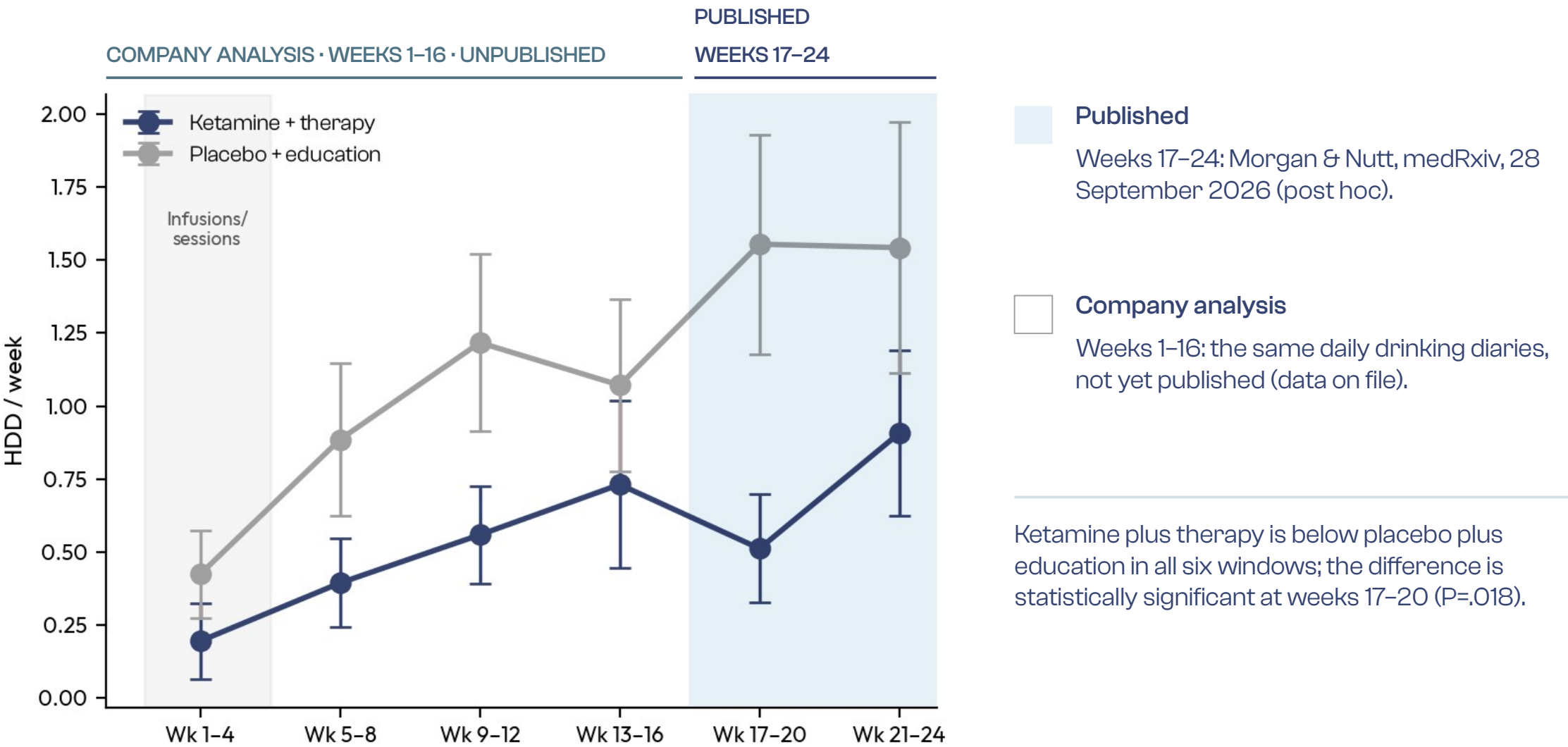
The same two arms on heavy drinking days (the Phase 3 primary endpoint): fewer than placebo in every four-week window, and 55% fewer over the last two months; post hoc

55%

fewer heavy drinking days over the last two months

Weeks 17–24, ketamine plus therapy versus placebo plus education: 0.71 vs 1.55 heavy drinking days a week (10.1% vs 22.2% of days); $P=.062$. Weeks 17–20 alone: 67% fewer, $P=.018$.

Phase 3 uses these two regimens; its control receives low-dose ketamine, not saline.



WHAT COUNTS AS A HEAVY DRINKING DAY

Phase 3 (European Medicines Agency, EMA): over 60 g of alcohol in a day for men, 40 g for women. Phase 2a/b re-analysis: 70 g (men) and 56 g (women).

WHY IT MATTERS

EMA lists reduction in heavy drinking days as a primary variable; it is the Phase 3 primary endpoint, tested prospectively over 180 days. These Phase 2a/b figures are post hoc.

Weeks 17–24: Morgan CJA, Nutt DJ, medRxiv, 28 September 2026 (post hoc secondary analysis of the Phase 2a/b trial; not peer-reviewed; nominal unadjusted P values; observed cases; ketamine plus therapy n=20 vs placebo plus education n=23). Weeks 1–16: company analysis of the same diaries, unpublished; data on file. Chart: University of Exeter. HDD: heavy drinking days. Professor Nutt is Solvonis' Chief Scientific Officer. Thresholds: EMA over 60 g (men), 40 g (women); re-analysis 70 g, 56 g. EMA, Guideline on the development of medicinal products for the treatment of alcohol dependence, section 4.2.1 (2010). Detail: slides 29–31.

The published trial was an academic proof of concept; Phase 3 is built around the EMA’s heavy drinking days endpoint

	Published Phase 2a/b	Phase 3 (Solvonis progressing)
Run by	Academic investigators at two UK sites; Medical Research Council funded	Exeter-sponsored; co-funded by NIHR, MRC and Solvonis; MHRA- authorised
Question	Proof of concept: does ketamine increase abstinence?	Does ketamine plus relapse-prevention therapy reduce heavy drinking days compared with control?
Comparison	Four arms; ketamine versus placebo pooled across therapy arms	Two arms: ketamine 0.8 mg/kg plus relapse-prevention therapy (the Phase 2a/b’s most intensive arm) versus ketamine 0.05 mg/kg plus alcohol education
Primary endpoint	Percentage of days abstinent; confirmed relapse (one or more heavy drinking days)	Heavy drinking days over 180 days
EMA guideline	Neither is an EMA primary variable	Heavy drinking days is an EMA primary variable for reduction in drinking (more than 60 g a day for men, 40 g for women)
Patients	96 (23 to 25 per arm)	280 planned

Sources: Grabski et al., Am J Psychiatry 2022;179(2):152–162 (design, funding statement); EMA, Guideline on the development of medicinal products for the treatment of alcohol dependence, section 4.2.1 (2010); MORE-KARE protocol version 9, 12 March 2025 (ISRCTN85955128). “EMA primary variable” refers to the primary variables the guideline specifies for each treatment goal. MHRA: UK Medicines and Healthcare products Regulatory Agency. NIHR: National Institute for Health and Care Research. MRC: Medical Research Council. Detail: slides 29 and 32.

Phase 3 tests the same two treatment regimens on heavy drinking days, the endpoint the EMA recognises

ONGOING TRIAL

280 planned participants

Two arms; double-blind

UK NHS sites; EU sites proposed

Exeter-sponsored; co-funded by NIHR, MRC and Solvonis

Primary endpoint: heavy drinking days over 180 days

MHRA-authorized; Innovation Passport (ILAP) granted

NOW	UK Phase 3 recruiting; MoU signed to assess Portuguese sites (13 Oct 2026)
2026–28	Recruitment; EU sites proposed
2029–30	Potential Phase 3 readout (indicative)
AFTER READOUT	Potential UK and EU marketing authorisation applications

THE TWO ARMS

Ketamine 0.8 mg/kg plus relapse-prevention therapy

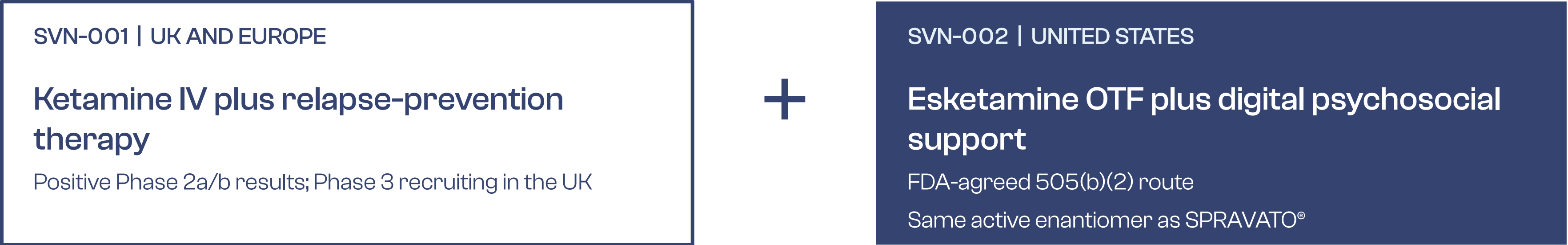
The most intensive Phase 2a/b arm

Ketamine 0.05 mg/kg plus alcohol education

The least intensive Phase 2a/b arm; low-dose ketamine replaces saline as an active control that protects the blind

Sources: MORE-KARE protocol version 9, 12 March 2025 (IRAS 1008179; ISRCTN85955128); MHRA clinical trial authorisation and HRA/REC approval, 2 November 2023; MHRA ILAP Innovation Passport; Solvonis RNS, 13 October 2026; Company information. Timings are indicative and subject to the availability of capital, regulatory feedback and trial progress. MoU partners: ICAD (Portugal's public addiction institute) and Panegreen. Portuguese participation is subject to sponsor and funder agreement and approvals. ILAP: Innovative Licensing and Access Pathway. REC: Research Ethics Committee. Detail: slide 32.

The same approach, built for the U.S.: SVN-002 brings esketamine to moderate-to-severe AUD



Established precedent

Supervised esketamine treatment is already established in the U.S. (SPRAVATO®).

A defined route

FDA agreed the 505(b)(2) pathway, with a scientific bridge to SPRAVATO®; Phase 2b planning is under way.

A large U.S. market

11.3m people aged 12+ with past-year moderate-to-severe AUD in the U.S.

Sources: Grabski et al., Am J Psychiatry 2022;179(2):152–162; Solvonis RNS, 21 January 2025; SAMHSA NSDUH (past-year moderate-to-severe AUD, ages 12+); MORE-KARE (ISRCTN85955128). SPRAVATO® is a trademark of Johnson & Johnson. Detail: slide 33.

Esketamine OTF: peak levels in line with SPRAVATO®, and a strong psychedelic-type experience

PEAK PLASMA LEVEL

96 vs 101

ng/mL: 50 mg OTF vs SPRAVATO® 84 mg, reached in about 19 minutes

PSYCHEDELIC-TYPE EXPERIENCE

95 vs 24

mystical-experience score (0–150): OTF vs placebo

PHASE 1 TOLERABILITY

0

serious adverse events; all 20 volunteers had dissociative effects

WHY THE MYSTICAL-EXPERIENCE SCORE MATTERS

It is the standard measure of the psychedelic experience in psilocybin and LSD research. In a randomised trial of ketamine in at-risk drinkers, mystical-type experiences mediated the reduction in drinking (Rothberg et al., 2021).

SVN-002 is investigational. Peak levels are model estimates (OTF: Phase 1, 20 healthy volunteers; SPRAVATO® 84 mg: typical patient aged 18–60). Placebo-controlled study: 28 problem drinkers. Sources: Simons et al., Front Pain Res 2022; Perez-Ruixo et al., Clin Pharmacokinet 2021; Gent et al., J Psychopharmacol 2024; Rothberg et al., J Psychopharmacol 2021. SPRAVATO® is a trademark of Johnson & Johnson.

FDA agreed the 505(b)(2) route, and no additional clinical data are needed before Phase 2b



FDA CONFIRMED

No additional clinical data are required before a Phase 2b trial in moderate-to-severe AUD.

FDA feedback is not an approval or a guarantee of any outcome. SVN-002 is investigational. IND: Investigational New Drug application; 505(b)(2): an FDA pathway that can draw on data for an already-approved drug. Sources: Solvonis RNS, 21 January 2025, 22 July 2025, 22 June 2026 and 30 September 2026. SPRAVATO® is a trademark of Johnson & Johnson.

Granted patents to ~2041, and potential Orange Book listing

Granted

Formulation patents

Covering the esketamine OTF formulation, granted in the U.S. and Europe; expected term to ~2041

Filed

Method-of-use applications

Esketamine OTF combined with psychotherapy in AUD and other substance use disorders, filed internationally

Potential

Orange Book listing

Eligible patents can be listed in the FDA Orange Book, adding patent-based protection alongside regulatory exclusivity

Patent grant, scope and Orange Book eligibility are not assured. SVN-002 is investigational.
Sources: patents US 11,992,561 B2 and EP 3995137 B1, and PCT/US2025/013057; Solvonis RNS, 21 January 2025.

A U.S. population about 4× that of treatment-resistant depression, and an established supervised-treatment model

PEOPLE WITH THE CONDITION, U.S.



Moderate-to-severe alcohol use disorder, ages 12+¹



about one quarter of AUD

Treatment-resistant depression, U.S. population²

\$1.7bn

SPRAVATO® worldwide sales in 2025³

+57%

SPRAVATO® sales growth, 2025 vs 2024³

REIMBURSEMENT MODEL

Supervised in-clinic dosing visit with structured at-home digital psychosocial support, designed to align with established SPRAVATO® supervised-visit coding.

Epidemiological population estimates. SVN-002 is investigational.
¹ SAMHSA, 2024 NSDUH, Tables 5.2A and 5.2B (ages 12+; civilian, non-institutionalised); ² Zhdanava et al., J Clin Psychiatry 2021 (U.S. adults: 30.9% of 8.9m with medication-treated MDD); ³ Johnson & Johnson Q4 and full-year 2025 results (SPRAVATO® worldwide sales \$1,696m in 2025 vs \$1,077m in 2024). SPRAVATO® is a trademark of Johnson & Johnson.

A de novo engine of proprietary new medicines across addiction and psychiatry

Proprietary new chemical entities from Solvonis' own discovery work, each with a composition-of-matter filing. NIDA funds and conducts the SVN-015 studies; Solvonis retains ownership and IP.

Programme	Compound	Delivered	Protection
SVN-015 StUD	A new dopamine and serotonin reuptake inhibitor, for cocaine and methamphetamine addiction	Encouraging initial NIDA screening (Aug 2026); advanced into further NIDA-funded studies	Composition-of-matter patent filed
SVN-114	A preclinical novel entactogen for PTSD, designed around MDMA's limitations	Lead selected from a six-analogue series (Mar 2026); rat microdialysis completed	Composition-of-matter patent filed
SVN-015 MDD	The same molecule as SVN-015 StUD, in depression with anhedonia	Antidepressant-like signal in a mouse model (Jan 2026)	Covered by the SVN-015 composition-of-matter filing

Preliminary preclinical findings do not establish safety or efficacy in humans. Patent grant and scope are not assured. Sources: Solvonis RNS, 3 December 2025, 28 January 2026, 10 March 2026 and 6 August 2026; Company patent data and data on file. Detail: slides 22–24, 34 and 35.

A new cocaine and methamphetamine addiction candidate in further NIDA-funded studies, after encouraging initial screening

THE DEVELOPMENT PROBLEM

59%

of U.S. overdose deaths (2021 to mid-2024) involved stimulants; there is no FDA-approved medicine for cocaine or methamphetamine addiction.

GBR-12909, an earlier dopamine transporter (DAT) inhibitor evaluated by NIDA, is known to interact with several cardiac ion channels.

FURTHER NIDA-FUNDED STUDIES

Confirmatory DAT binding and functional studies, a mouse locomotor-activity study of onset and duration of effects, and follow-up on serotonin 5-HT_{2B} receptor activity.

Preliminary preclinical findings do not establish the safety of SVN-015 in humans or whole animals. Sources: Tanz et al., MMWR 2025 (stimulant-involved overdose deaths, Jan 2021 to Jun 2024); Solvonis RNS, 3 Dec 2025 and 6 Aug 2026; Obejero-Paz et al., Sci Rep 2015;5:17623; Company patent data. Detail: slide 34.

THE SVN-015 RESPONSE

A new dopamine and serotonin reuptake inhibitor, evaluated by NIDA

WHAT HAS BEEN ANNOUNCED

Initial NIDA screening (Aug 2026)

Cardiac ion-channel and broader off-target screens, run by NIDA, gave encouraging results.

A more favourable profile than GBR-12909

On the cardiac ion-channel measures assessed to date.

NIDA-funded; Solvonis retains ownership and IP

NIDA conducts and funds the studies through its preclinical Addiction Treatment Discovery Program (ATDP).

Intellectual Property

Composition-of-matter patent filed.

Engineering a proprietary, potentially more scalable alternative to MDMA

THE OPPORTUNITY

MDMA-assisted therapy has generated clinical interest in PTSD.

But MDMA is not proprietary, and supervised treatment is resource-intensive.

The objective is a medicine designed for healthcare delivery — not simply another MDMA analogue.

THE SVN-114 PRODUCT THESIS

Retain the therapeutically relevant serotonergic profile while designing around MDMA’s limitations

ESTABLISHED	Proprietary	Composition-of-matter patent filed.
EARLY EVIDENCE	Moderated dopamine	Lower fitted dopamine release than MDMA in the same in-vitro assay.
COMPANY DATA	Cleaner metabolism	Preclinical metabolism indicates no MDA formation.
DESIGN OBJECTIVE	Scalable treatment	Soft-drug design aims for a shorter, more predictable treatment window.

SVN-114 is preclinical; intended advantages remain to be established. Sources: Mitchell et al., Nat Med 2023; FDA Psychopharmacologic Drugs Advisory Committee, 4 June 2024; Company programme data. MDMA: 3,4-methylenedioxymethamphetamine; MDA: 3,4-methylenedioxyamphetamine, a metabolite of MDMA. Detail: slide 35.

Same molecule, second programme: an exploratory antidepressant-like signal

THE UNMET NEED

Anhedonia is an unmet need

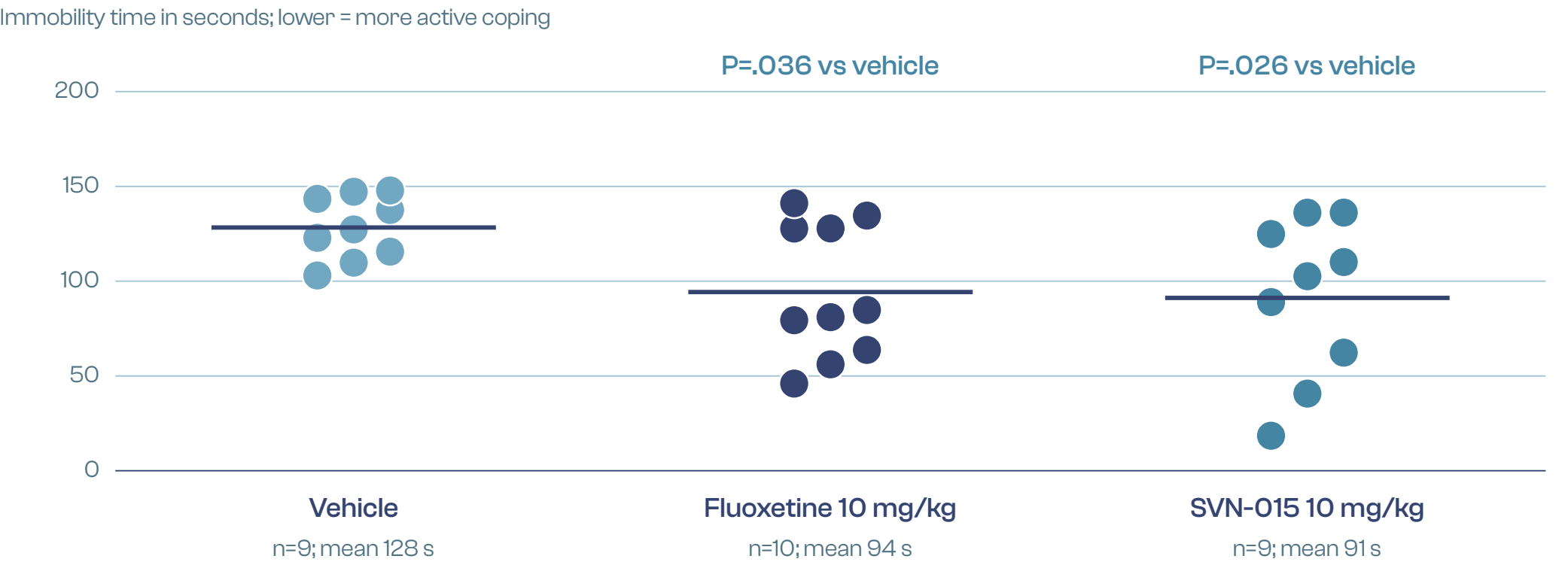
Anhedonia, the loss of pleasure and motivation, is a core symptom of major depressive disorder (MDD).

Many patients do not achieve adequate symptom control on selective serotonin reuptake inhibitors (SSRIs). SVN-015 is a serotonin-dopamine reuptake inhibitor, designed to engage pathways involved in mood, motivation and reward.

Preclinical animal data do not establish human safety or efficacy in depression, and the forced swim test does not itself measure anhedonia. Sources: Solvonis RNS, 28 January 2026; Ulysses Neuroscience study UNL094 (report dated 21 January 2026); Company data on file.

THE EARLY EVIDENCE

In one mouse study, treatment with SVN-015 reduced forced-swim immobility, as did fluoxetine, an approved SSRI antidepressant used as the positive control



One-way ANOVA P=.047 (Fisher LSD). 14 daily subcutaneous doses, tested 24 h after the last dose. Bars: means.

Single study, one dose, male mice. One vehicle outlier (182 s) and one under-dosed SVN-015 animal excluded. A screen for antidepressant-like activity, not for anhedonia.

SUMMARY

An alcohol use disorder franchise at the clinical core, with a de novo engine behind it

01

Late-stage clinical assets

The AUD franchise: SVN-001 in Phase 3 in the UK, with Portuguese sites being assessed; SVN-002 on an FDA-agreed 505(b)(2) route, with granted patents to ~2041.

02

Scientifically de-risked

Ketamine and esketamine pharmacology; one of two co-primary endpoints met in the published Phase 2a/b. NIHR and MRC co-fund the Phase 3, the FDA agreed the SVN-002 route, NIDA funds and conducts the SVN-015 studies. Scientific leadership from Professor David Nutt.

03

Near-term milestones

The de novo engine: SVN-015 in further NIDA-funded studies; SVN-114 and SVN-015 in depression, both with composition-of-matter filings. SVN-002 IND submission in 2027.

04

Attractive entry point

Pharma has paid billions for depression and PTSD. Solvonis has programmes in both, and a Phase 3 in alcohol use disorder.

SVN-001, SVN-002, SVN-015 and SVN-114 are investigational. FDA feedback is not an approval or a guarantee of any outcome; patent grant and scope are not assured. Sources and detail: slides 9–24.

LEADERSHIP

Leadership combines psychedelic science, public-company execution and commercial experience



Anthony Tennyson
Chief Executive Officer

Public-company leadership; biopharma capital markets, M&A and psychedelics strategy



Professor David Nutt
Chief Scientific Officer

Edmond J Safra Professor of Neuropsychopharmacology, Imperial College London; former Chair, UK Advisory Council on the Misuse of Drugs



Ryan Neates
Chief Financial Officer

Public-company finance, governance and capital-markets execution

Board



Dennis Purcell
Chair



Dr Renata Crome
Non-executive Director



Paul Carter
Non-executive Director



Nicholas Nelson
Non-executive Director



Experience across addiction, psychiatry and neurology; biopharma capital markets and M&A; drug development and commercialisation.

CAPITAL STRUCTURE

7.90bn shares are in issue, and 9.31bn on a fully diluted basis

As at 28 September 2026. The fully diluted total assumes exercise of all outstanding warrants and options.

Class	Number	% of fully diluted
Common shares	7,899,736,826	84.9%
Warrants	1,111,298,765	11.9%
Options	298,000,000	3.2%
Fully diluted capital structure	9,309,035,591	100.0%

COMPOSITION OF THE FULLY DILUTED CAPITAL STRUCTURE



Percentages are calculated from the figures shown and rounded.
Source: Solvonis Therapeutics plc, latest Total Voting Rights announcement; Company data on file.

Appendix

Supporting data, cross-trial context and references

SLIDE	APPENDIX CONTENT	SUPPORTS
29	SVN-001 Heavy drinking days and EU expansion	Slides 13–15
30	SVN-001 Heavy drinking days over six months, all four arms	Slide 13
31	SVN-001 Heavy drinking days against other AUD trials	Slide 13
32	SVN-001 From Phase 2a/b to Phase 3	Slides 14 and 15
33	SVN-001 and SVN-002 Two molecules, one mechanism	Slides 9 and 16
34	SVN-015 StUD The NIDA programme in detail	Slide 22
35	SVN-114 Early monoamine-release data	Slide 23
36–37	References Sources for all slides	All slides

Why heavy drinking days matter for EU expansion: the regulatory case and post hoc data behind slides 13–15

WHY HEAVY DRINKING DAYS MATTER IN THE EU

- 01

A recognised regulatory endpoint

EMA's guideline on alcohol dependence lists reduction in heavy drinking days (>60 g men, >40 g women per day) as a primary variable, alongside change in total alcohol consumption.
- 02

The Phase 3 primary endpoint

MORE-KARE's primary endpoint is the number of heavy drinking days over 180 days, using EMA thresholds. Proposed EU sites are intended to generate EU data on it.
- 03

Phase 3 is the prospective test

In Phase 2a/b, heavy drinking days were analysed post hoc (n=20 vs 23). Phase 3 tests the endpoint prospectively, using EMA thresholds.

Definitions differ: EMA >60/>40 g; Grabski et al. relapse >64.8/>52.0 g; Morgan & Nutt heavy drinking days ≥70/≥56 g.

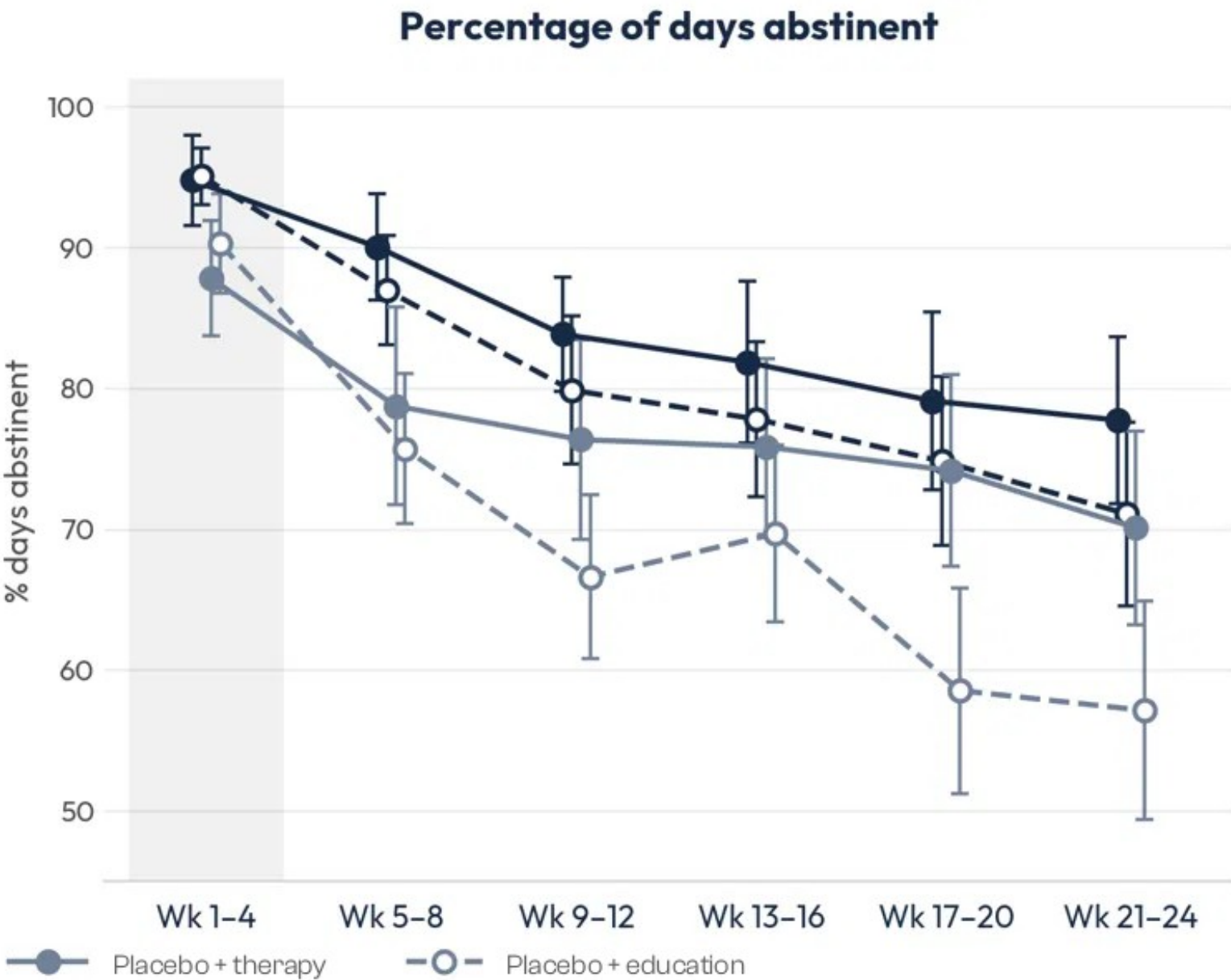
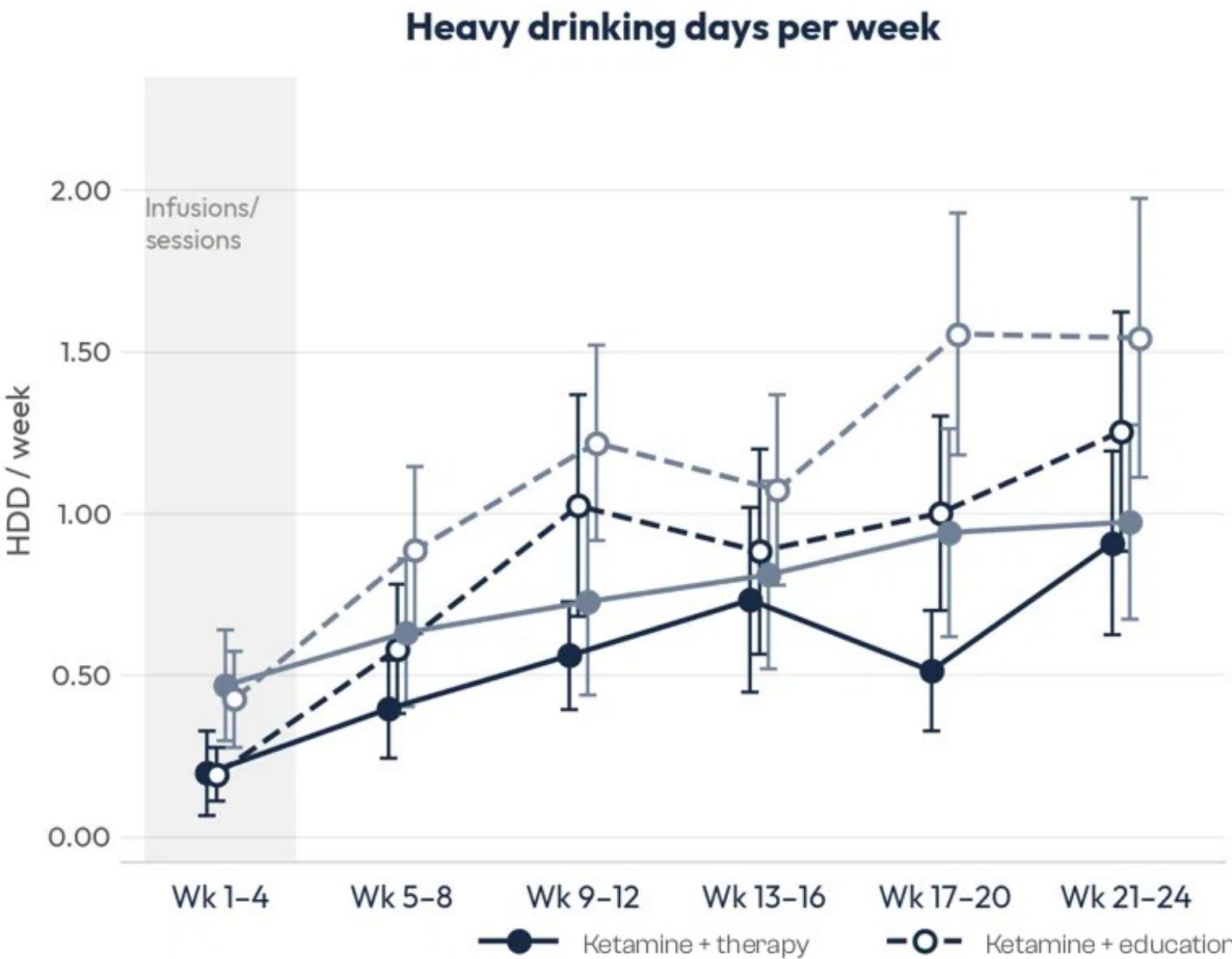
Sources: European Medicines Agency, Guideline on the development of medicinal products for the treatment of alcohol dependence, section 4.2.1; ISRCTN85955128; Morgan CJA, Nutt DJ (Nutt is Solvonis' CSO), medRxiv, 28 September 2026 (post hoc, not peer-reviewed); Grabski et al., Am J Psychiatry 2022;179(2):152–162.

PHASE 2A/B HEAVY DRINKING DAYS, % OF DAYS, BY WINDOW (POST HOC)

Phase 2a/b window	Placebo → active	Reduction	P
SVN-001, weeks 17–20 Phase 2a/b; ketamine + therapy	22.2% → 7.3%	67%	.018
SVN-001, weeks 21–24 Phase 2a/b; ketamine + therapy	22.0% → 13.0%	—	.226 (n.s.)
SVN-001, pooled 17–24 Phase 2a/b; ketamine + therapy	22.2% → 10.1%	55%	.062 (n.s.)

Post hoc analysis of the Phase 2a/b: ketamine + therapy (n=20) vs placebo + education (n=23), observed cases, nominal unadjusted P values, not peer-reviewed. Patients had completed detoxification and were measured 4–6 months after the last infusion. Other AUD treatments are shown for indirect context in the cross-trial appendix slide (slide 31); they are not head-to-head comparisons.

All four arms over six months: heavy drinking days and days abstinent, behind slide 13

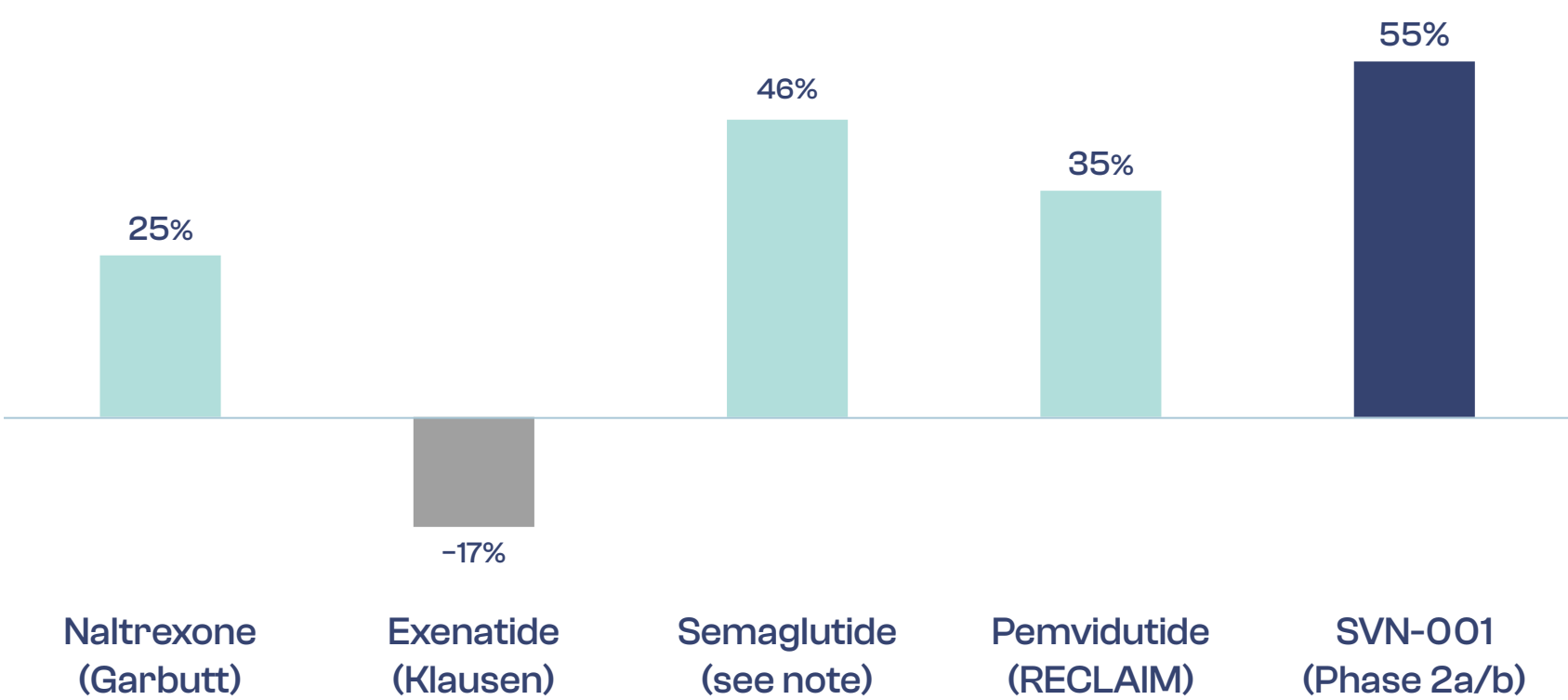


Company analysis of the KARE daily drinking diaries, all four arms, observed cases; data on file. Of these, only weeks 17–24 for ketamine plus therapy versus placebo plus education are published (Morgan CJA, Nutt DJ, medRxiv, 28 September 2026; post hoc, not peer-reviewed). The trial was not powered for arm-by-arm comparison. Chart: University of Exeter. HDD: heavy drinking days.

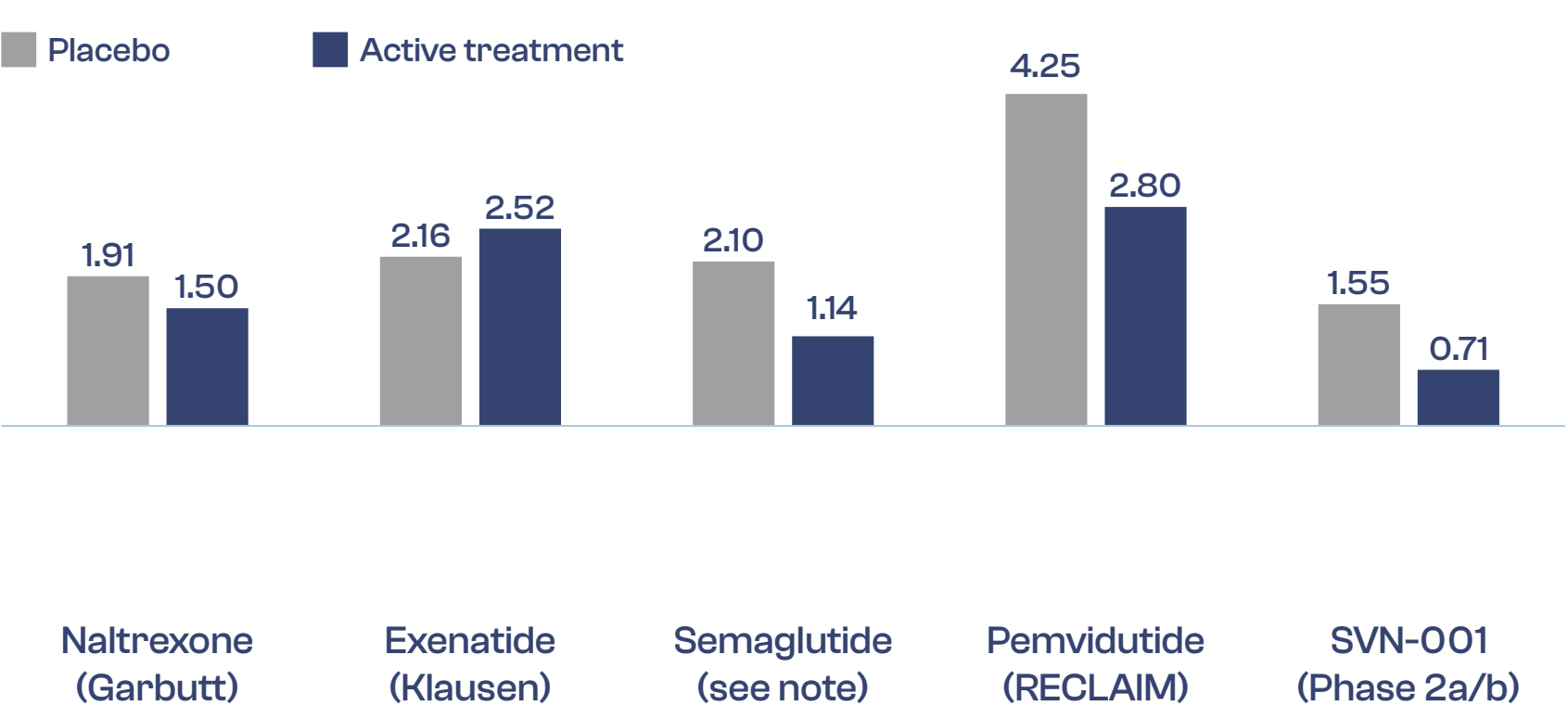
Context for slide 13: the post hoc heavy drinking days result alongside other AUD trials (indirect comparison)

Heavy drinking days in the Phase 2a/b versus established and emerging AUD pharmacotherapies

A Relative reduction in heavy drinking days



B Absolute heavy drinking days per week



SVN-001’s post hoc reduction in heavy drinking days (55%, weeks 17–24 pooled) compares with reported relative reductions of 25% for naltrexone, 35% for pemvidutide and 46% for semaglutide. Differences among the studies and patient populations mean these comparisons do not establish comparative efficacy.

These are indirect, cross-trial comparisons; dosing schedules reflect approved/reported regimens, not head-to-head. Phase 2a/b bar = ketamine + therapy vs placebo + education (most vs least intensive arm), post hoc, pooled weeks 17–24, P=.062 (not significant). Panel A: relative reductions as reported. Panel B: rates derived from published baselines and change scores; the two can differ by a few points. Data: Morgan & Nutt (medRxiv, 28 September 2026; not peer-reviewed; Nutt is Solvonis’ CSO). Naltrexone: Garbutt 2005 (long-acting injectable); 25% as reported by Morgan & Nutt. Semaglutide: derived by Morgan & Nutt from Hendershot 2025 and Klausen 2026. Pemvidutide: topline press release, July 2026; absolute values assume a baseline of 7 heavy drinking days/week.

How Phase 2a/b leads into Phase 3: the regimen, results and endpoint carried forward on slides 14 and 15

PHASE 2A/B SECONDARY: KETAMINE + THERAPY (N=24) VS PLACEBO + EDUCATION (N=25)

As published (Grabski et al.)	Ketamine + therapy vs placebo + education
% days abstinent, 6 months (secondary comparison)	+15.9 points (95% CI 3.8 to 28.1): significant
Confirmed relapse, 6 months (secondary comparison)	62% (13/21) vs 78% (18/23); OR 0.46 (0.12 to 1.74): not significant
Confirmed relapse, 3 months	57% (12/21) vs 71% (17/24); descriptive

POST HOC, SAME TWO ARMS (N=20 VS 23)

Morgan & Nutt preprint, not peer-reviewed	Ketamine + therapy vs placebo + education
Heavy drinking days, weeks 17–20	7.3% vs 22.2% of days (–67%): P=.018
Heavy drinking days, weeks 21–24	13.0% vs 22.0% of days: P=.226, not significant
No heavy drinking days, weeks 21–24	50.0% vs 26.1%; OR 2.83 (0.79 to 10.17): P=.127, not significant
Days abstinent, weeks 17–20	79.1% vs 58.5%; +20.6 points (1.1 to 40.0): P=.039

WHAT PHASE 3 CARRIES FORWARD

Treatment: the most intensive arm’s regimen: three 40-minute IV ketamine infusions (0.8 mg/kg) with seven sessions of manualised relapse-prevention therapy.

Control: two arms only, matching the most and least intensive Phase 2a/b arms. Double-blind, with low-dose ketamine (0.05 mg/kg) plus alcohol education; Phase 2a/b used saline.

Scale: 280 planned participants, against 96 randomised in Phase 2a/b and 49 in this two-arm contrast.

Endpoint: heavy drinking days over 180 days (EMA thresholds: >60 g men, >40 g women). In Phase 2a/b this was post hoc; the published co-primaries were % days abstinent and confirmed relapse.

Setting: UK NHS sites, University of Exeter sponsor, with proposed EU expansion.

Sources: Grabski et al., Am J Psychiatry 2022;179(2):152–162 (Table 2; Table S1 as cited in text); Morgan CJA, Nutt DJ (Nutt is Solvonis’ CSO), medRxiv, 28 September 2026 (post hoc; nominal unadjusted P values; ketamine + therapy vs placebo + education); MORE-KARE protocol version 9, 12 March 2025 (IRAS 1008179; ISRCTN85955128); Company information. Phase 3 timings are indicative and subject to the availability of capital, regulatory feedback and trial progress.

Why two molecules: one shared mechanism, each built for its own market (slides 9 and 16)

01

A racemic molecule

Ketamine is manufactured and administered as a 50:50 mixture of two mirror-image enantiomers — R-ketamine (arketamine) and S-ketamine (esketamine) — both acting primarily as NMDA-receptor antagonists.

02

A more potent enantiomer

S-ketamine binds the NMDA receptor with about 4–5× greater affinity than R-ketamine (K_i 0.30 vs 1.4 μM, rat cortex)¹. Esketamine is the active ingredient of SPRAVATO®.

03

Two differentiated products

SVN-002, an esketamine OTF, leverages esketamine's FDA precedent (SPRAVATO®) for a U.S. 505(b)(2) pathway, while SVN-001 develops racemic ketamine's own UK/EU route.

SVN-001 and SVN-002 are not sequential — they are differentiated molecules, each built for its own market and regulatory pathway. SVN-002's efficacy and safety in AUD remain to be established in clinical studies.

¹ Zanos P, et al. "Ketamine and ketamine metabolite pharmacology: insights into therapeutic mechanisms." *Pharmacol Rev.* 2018;70(3):621–660. doi:10.1124/pr.117.015198 (K_i values from Ebert et al., 1997). Bonaventura J, et al. "Pharmacological and behavioral divergence of ketamine enantiomers: implications for abuse liability." *Mol Psychiatry.* 2021;26(11):6704–6722 (about 5-fold). Company information. SPRAVATO® is a trademark of Johnson & Johnson.

The NIDA programme in detail: encouraging initial screening; in further NIDA-funded studies (slide 22)

WHAT HAS BEEN ANNOUNCED

- 01

Accepted into NIDA's Addiction Treatment Discovery Program (Dec 2025)

NIDA funds and conducts early preclinical evaluation for methamphetamine and cocaine addiction.
- 02

Initial NIDA screening (Aug 2026)

Initial cardiac ion-channel and broader off-target screens, run by NIDA, gave encouraging results.
- 03

More favourable profile than GBR-12909

On the cardiac ion-channel measures assessed to date, versus the earlier DAT inhibitor.

FURTHER NIDA-FUNDED STUDIES (ANNOUNCED 6 AUGUST 2026)

Study	What it assesses
Mouse locomotor-activity study	Time course: onset and duration of effects
Confirmatory DAT studies	DAT binding and functional activity
5-HT ₂ B follow-up	Activity at the 5-HT ₂ B receptor

NIDA conducts and funds these studies. Timelines have not been announced.

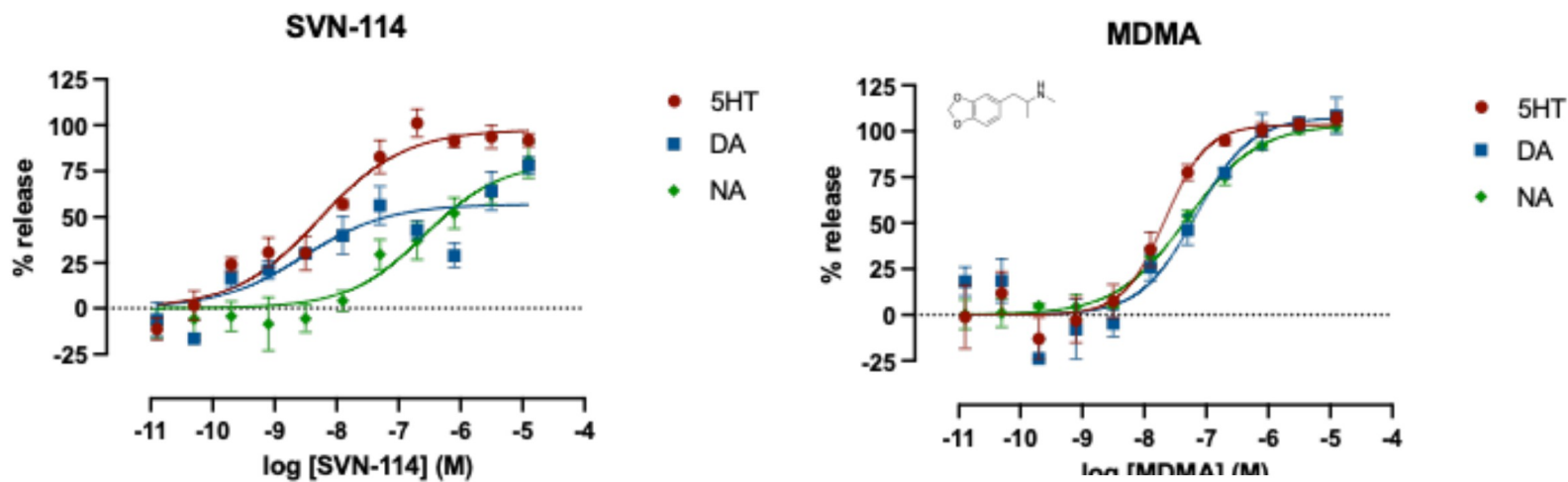
TERMS OF THE NIDA PROGRAMME

NIDA funds and conducts the studies; Solvonis retains ownership of SVN-015 and its intellectual property.

Preliminary preclinical findings do not establish the safety of SVN-015 in humans or whole animals; further safety pharmacology, toxicology and clinical evaluation will be required. NIDA's involvement does not necessarily represent the official views of NIH or NIDA. Sources: Solvonis RNS, 3 December 2025 and 6 August 2026; GBR-12909: Obejero-Paz et al., Sci Rep 2015;5:17623.

The early data behind slide 23: serotonin release comparable to MDMA with a lower dopamine maximum

CONCENTRATION-RESPONSE CURVES



Both plots use matched axes. Differentiation is the lower maximum dopamine response — not lower DAT potency.

Same-assay in-vitro monoamine-release data

PROGRESS TO DATE | Lead selected from six-analogue series • Rat nucleus-accumbens microdialysis completed • Composition-of-matter patent filed

Preliminary preclinical findings do not establish human efficacy, improved safety or reduced abuse liability. Source: Company same-assay release data and programme information.

FITTED MAXIMUM RELEASE, EMAX (% OF CONTROL)

Serotonin release comparable to MDMA; lower fitted dopamine maximum

Serotonin	SVN-114 98	MDMA 104	near-comparable
Dopamine	SVN-114 57	MDMA 108	47% lower (fitted)
Noradrenaline	SVN-114 87	MDMA 103	16% lower (fitted)

A lower fitted dopamine maximum supports a lower-reinforcement hypothesis; formal abuse-liability testing is required.

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