As of early 2026, the discussion around **CSL** and **JRT** in the context of schizophrenia represents one of the most promising frontiers in neuropsychiatry.

While **JRT** is not yet a "cure," it is a groundbreaking experimental compound that shifts the focus from managing symptoms to **repairing the brain's physical architecture.**

What is JRT?

JRT (named after researcher Jeremy R. Tuck) is a synthetic molecule derived from **LSD**. It was developed by Dr. David E. Olson and his team at the **University of California, Davis**.

Unlike traditional psychedelics, JRT is a **non-hallucinogenic psychoplastogen**. This means it retains the brain-healing benefits of LSD without causing the "trip" or hallucinations, making it safe for patients with schizophrenia who are traditionally barred from psychedelic therapy.

How It Works

- **Neural Repair:** Schizophrenia is characterized by "brain atrophy," specifically the loss of connections (synapses) in the prefrontal cortex. In preclinical studies, JRT increased **dendritic spine density by 46%**, essentially "rewiring" damaged areas.
- **Targeting the "Negative" Symptoms:** Most current drugs target dopamine to stop hallucinations. JRT targets **serotonin (5-HT2A)** to treat the "negative" and cognitive symptoms—such as social withdrawal, memory loss, and the inability to feel pleasure—which have previously been nearly impossible to treat.
- **Potency:** Early tests suggest JRT is significantly more potent than current fast-acting antidepressants like ketamine.

The CSL Connection

The link between **CSL** (a global biotechnology leader) and JRT stems from CSL's **Research Acceleration Initiative (RAI)**.

In late 2025 and early 2026, CSL has been actively scouting and funding "high-risk, high-reward" biotherapies. While JRT began as a UC Davis academic project, CSL's involvement typically signals the transition from **academic discovery to large-scale clinical development.** > **Current Status (2026):** JRT is currently in the **preclinical to Phase 1 transition phase.** This means it is moving from animal models into the first stages of human safety testing.

JRT vs. Traditional Treatments

The excitement around JRT exists alongside the recent approval of **Cobenfy**

(xanomeline/trospium), which was the first drug in decades to treat schizophrenia without using dopamine.

Feature	Traditional Antipsychotics	Cobenfy (Approved 2025)	JRT (Experimental)
Mechanism	Blocks Dopamine (D2)	Targets Muscarinic Receptors	Promotes Neuroplasticity
Primary Benefit	Stops Hallucinations	Reduces symptoms with fewer side effects	**Repairs neural connections**
Cognitive Impact	Often causes "brain fog"	Neutral to Positive	**Potentially Restorative**

Is it a Cure?

In medical terms, "cure" is a heavy word. However, JRT is part of a new class of **disease-modifying** treatments. If human trials mirror the animal results, JRT wouldn't just mask the disorder; it would help the brain return to a more functional, healthy state by physically regrowing the connections lost to the disease.

Would you like me to find the latest updates on the first human clinical trials for JRT, or would you prefer a deeper look at how it differs from other new drugs like Cobenfy?