Psilocybin slows binocular rivalry switching through serotonin modulation.


**Previous findings**

**Psychosis** - People with bipolar disorder show slower than normal binocular rivalry switching (Pettigrew & Miller, 1998).

**Psilocybin** - The hallucinogenic psilocybin slows down binocular rivalry switching in parallel with increases in drug induced psychosis-like symptoms (Carter et al., 2005).

**Ketanserin** - Is a drug that has been found to block psilocybin’s subjective psychosis-like symptoms (Vollenweider et al., 1998).

**Project Aims**

- **To investigate the proposed link between switching rate and symptoms of psychosis (e.g., bipolar disorder & schizophrenia).**
- **Determine whether ketanserin can block psilocybin’s effect on binocular rivalry or if the subjective psychosis-like effects can be dissociated from the binocular rivalry switching rate.**
- **To use this pharmacological manipulation to tease out the relative contribution of the serotonin (5-HT) 1A and 2A receptors in any observed effects on rivalry rate and conscious state.**

**Methods**

10 Healthy human subjects - 4 conditions: placebo; psilocybin (215 µg/kg); ketanserin (50mg/kg); psilocybin + ketanserin.

**Subjective effects**

Psilocybin produced a variety of subjective changes in conscious state.

Ketanserin blocked some of the psilocybin’s psychosis-like effects, but NOT the attention/arousal effects.

**Summary**

- Hallucinogenic doses of the 5-HT1A agonist psilocybin slows down the rate of rivalry switching & the duration of the transition phase.
- Pre-treatment with the 5-HT1A antagonist Ketanserin blocked the “positive” psychosis-like symptoms (hallucinations) but not psilocybin’s effect on binocular rivalry or the “negative” psychosis-like symptoms (attention & arousal).
- These findings link binocular rivalry switching rate to subjective measures of attention and arousal.
- Suggest the involvement of serotonin & the 5-HT1A receptor in this association.
- This result raises the possibility that rivalry may have diagnostic implications for conditions characterised by attentional deficits such as autism and ADHD.

**References**

