Introduction

- **Emotional self-medication (ESM):** Some individuals consume anxiolytic substances to reduce negative emotions induced by stressful life events involving reward loss (Torres & Papini, 2016).
- **cSNC:** Loss-induced negative emotion was examined in rats using the consummatory successive negative contrast task (Papini et al., 2015).
- **Central amygdala (CeA):** Research investigating a hypothesized neural circuitry of reward loss identified the CeA as a brain nucleus involved in negative emotion (Kawasaki et al., 2015).

Method

- **Neural inactivation:** Inhibitory designer receptors exclusively activated by designer drugs (DREADDs) were used to silence CeA neurons via injection of the DREADD activator clozapine N-oxide (CNO).
- **Design:** Four groups of rats were exposed to the cSNC task. Two groups received a 32-2% sucrose downshift (32/CNO and 32/Veh), and two groups received only 2% sucrose (2/CNO and 2/Veh). Group 32/CNO experienced reward downshift under inactive CeA, whereas 32/Veh groups experienced reward downshift under normal CeA activity.
- **Preference test:** All rats were given an opportunity to voluntarily consume 10% ethanol or deionized water for 1 hour immediately following each cSNC session.

Results

- Relative lick frequency for sessions 11-13 was lower in 32/Veh than in 2/Veh: the cSNC effect.
- CeA inhibition eliminated the cSNC effect in 32/CNO vs. 2/CNO.
- A hint of ESM was found in 32/Veh, with increased ethanol consumption relative to water.
- The ESM effect was not found in 32/CNO.
- The cSNC and ESM effects found on sessions 11-13 were not present on sessions 14-15 after full recovery from reward downshift.

Conclusions

- CeA inactivation prior to reward devaluation eliminated the cSNC effect, confirming a function for the CeA in reward loss circuitry.
- Preference test results suggested a link between reward loss and anxiolytic intake, supporting the ESM hypothesis.
- Future research will explore the role of inputs to the CeA in reward loss using the DREADD approach to manipulate neural activity.

References