Clinical evidence shows that there is acute anxiety initiated by selective serotonin reuptake inhibitors (SSRIs). Acute SSRI administration also enhances fear learning in rats, shown through fear conditioning. This enhancement is mediated by neurons in the bed nucleus of the stria terminalis (BNST).

A previous study (Ravinder et al. 2013) determined that acute systemic administration of fluoxetine (SSRI) enhances conditioned fear memory and enhances Arc protein expression in the BNST and CeA. The study also demonstrated that direct local infusion of fluoxetine into the CeA does not enhance fear memory or Arc expression, but direct local infusion of fluoxetine into the BNST is sufficient to enhance conditioned fear memory and enhance Arc protein expression in the CeA. Their conclusions suggest that cells in the BNST are initiating the mechanism of the acute response to the SSRI. Thus, in our experiment we are looking at the cells in that brain region with the particular serotonin receptors that are known to be involved with SSRIs (5HT2CRs).

We used single-labeling and double-labeling immunohistochemical (IHC) techniques in the rat to address this question. First, we localized 5HT2CRs in the oval nucleus of the BNST. Then we showed their co-localization with up-regulated Arc expression induced by pairing an SSRI injection with fear conditioning.