Background

- Corticosteroid hormones, in interaction with noradrenaline, enable the consolidation of emotionally arousing and stressful experiences.
- This is thought to occur mainly in the basolateral nucleus of the amygdala (BLA), which is crucially involved in emotional memory formation.
- Extensive evidence points to long-term synaptic potentiation (LTP) as a mechanism contributing to memory formation.

Aim

To determine the effects of stress on LTP in the LA-BLA pathway and the specific roles of corticosteroid and β-adrenergic receptor activation in this process.

Methods

**Exp 1:** effect of stress on *in vitro* BLA LTP (male C57/Bi6 mice)

- 20 min restraint stress or left undisturbed

**Exp 2:** effect of antagonist pretreatment on BLA LTP in controls

- *In vivo* i.p. pretreatment with antagonists 50 min prior to decapitation: 10 mg/kg propranolol (β-adrenoceptor), 10 mg/kg mifepristone (GR), 50 mg/kg spironolactone (MR) or vehicle (Fig. 1A)

**Exp 3:** effect of antagonist pretreatment on LTP in stressed mice

- *In vivo* pretreatment with antagonists prior to 20 min of restraint stress (Fig. 1A)

- field potential recordings in mouse brain slices (*in vitro*) via stimulation of the LA-BLA pathway (Fig. 1B, C)

- 1x100 Hz (1s) tetanization to evoke BLA LTP (Pu et al., 2009)

Conclusions

- Acute stress changes BLA electrical properties such that subsequent LTP induction is facilitated.
- Both β-adrenergic and glucocorticoid receptors are involved in the development of these changes.
- MRs are important for the maintenance of LTP in the BLA, irrespective of stress-induced changes in the circuit.
- The prolonged changes in BLA network function after stress may contribute to effective memory formation of emotional and stressful events.