It is well documented that social animals cooperate and communicate with each other, which influences their behavior and emotional state. Solitary confinement was found to be a severe stressor for social mammals. On the other hand, social support has positive influence on both psychological and physiological condition of social animals as well as humans. Moreover, in some social species such as rats, guinea pigs, non-human primates and also humans, the stress level is lowered and stress endocrine and autonomic responses are dampened by the mere presence of a conspecific during or right after the exposure to the aversive stimulus. It also improves recovery from aversive experiences. This effect is termed social buffering. Several studies showed a decrease in stress in previously stressed animals in the presence of a non-fearful conspecific. Most of the research concerning social buffering effect utilizes fear conditioning to model trauma. In this paradigm the animal is presented with a neutral stimulus (conditioned stimulus, CS, usually a tone or light), followed by an aversive stimulus (unconditioned stimulus, US, usually a mild electric footshock). After several presentations of CS and US together the animal learns to associate CS with US. The strenght of this association is then tested by subsequently exposing the animals to CS alone. In such case animals exhibit fear response manifested by a freezing response. Conditioned fear memory can be extinguished by several sessions of presentation of CS without US.

Fear is usually beneficial in terms of avoidance of danger and survival in the natural environment but pathological fear memory may cause phobias and post traumatic stress disorder (PTSD). Behavioral therapies are utilized to cure, or at least minimize the symptoms of these disorders. Such therapies rely on a paradigm very similar to fear extinction protocols used in animal studies, and are quite effective in the most of cases. Unfortunately, the effect of such treatment is very often transient. The once extinguished fear memory tends to be renewed with time, either spontaneously or due to the change of context. Therefore fear learning and extinction are still extensively studied. Recently, it was shown that during fear extinction, fear memory trace is not removed but only inhibited. Almost nothing, however, is known about the impact of social interaction on fear memory extinction and renewal. The only study concerning this topic comes from our laboratory (Nowak et al. 2013). It shows that previously extinguished mice tested in the presence of fearful cagemate undergo fear memory renewal. Since mouse behavior is often different from rat behavior, especially in terms of fear learning and extinction, we adapted this model to experiment on rats. In our model rats are housed toegether for at least two weeks and next habituated to an experimental cage devided into two compartment by a perforated wall. On the first day of the experiment both rats from each pair are separately fear conditioned by presentation of 5 CS (tone) terminated with a brief electric footshock (US). During the next three days one rat from each pair (called the ”observer”) is exposed to 15 presentations of CS alone (a procedure reliably extinguishing the learned fear response), while the other rat (called the “demonstrator”) is only exposed to the experimental cage (no extinction). In the control group both animals undergo fear extinction procedure.

Our preliminary results confirm the existence of a strong social buffering effect. Namely, in the experimental group we observed no fear response in either the extinguished observers or in the fearful, non extinguished demonstrators. However, the demonstrators tested alone exhibited a strong freezing response.

In this proposal we would like to investigate the mechanisms of social buffering in our model of socially modulated fear memory extinction. This goal will be achieved in two steps. First, we will investigate which modality of the information causes social buffering effect. We will also investigate the importance of familiarity and physical similarity between the rats for social buffering. Finally, we will look at the corticosterone levels in the blood taken from rats tested together or separately.

In the next step we will indentify brain strucures important for creating the social buffering effect in our model. We will look at the c-Fos protein level, commonly used marker of neuronal activity, in several structires including: the lateral nucleus of the amygdala (LA), basal nucleus of the amygdala (BA), medial (CeAm) and lateral (CeAl) parts of the central nucleus of the amygdala, as well as the prefrontal cortex (divided into prelimbic, PL, and infralimbic, IL, parts), the cingulate cortex and the insular cortex in brain slices from animals tested toegether or separately. Next, we will use electrophysiological single units recording technique to measure neuronal activity during behavioral testing in structures previously selected on the basis of c-Fos protein level analysis. Finally, we will support the afromentioned experiments using optogenetics approach which allows to selectively activate or inhibit single structures or even singles groups of neurons.

Data obtained during realization of this project will help to understand the mechanism of social buffering and its impact on fear extinction process. As it was shown that social interactions modulate conditioned fear learning it seems likely that also fear extinction undergoes social modulation. Understanding the mechanisms of social buffering during fear memory extinction is crucial for creating new therapeutical approaches.