The influence of 5-HTTLPR, emotion regulation and parenting on Fear Conditioning

Background
A polymorphism within the serotonin transporter gene promoter region (5HTTLPR) is associated with anxiety-related personality traits (Canli & Lesch, 2007), amygdala hyperreactivity (Hariri & Holmes, 2006), increased startle response (Waters et al., 2008), and an increased vulnerability towards stress (Caspi et al., 2003). The short (s)-allele of 5-HTTLPR seems to be the risk allele, posing a risk for the carrier to develop anxiety disorders, as opposed to the long (l)-allele. However, Eley and colleagues (2011) revealed astonishing new insights: children with anxiety disorders attending cognitive-behavioral therapy (CBT) showed no differences during therapy and post-treatment in measurements of anxiety severity. In follow-up (6 months), carriers of the homozygotic (ss)-genotype showed significantly lower anxiety severity than carriers of at least one (l)-allele. Hence one could argue that the (s)-allele is associated with a better responding to CBT, specifically extinction as the major element of CBT in treating anxiety disorders (Eley et al., 2011). These results are in line with differential susceptibility theory, which assumes that people with specific predispositions are more sensitive to influences of the environment, both positive as well as negative influences (Bakermans-Kranenburg & van IJzendoorn, 2007). These findings of previous research lead to the question whether (ss)-genotype is associated with general vulnerability or differential susceptibility to environmental influences. Our expectations are threefold:

Hypotheses:
1. In the group of children with the short allele the extinction effects of day one and two differ significantly.
2. 5-HTTLPR moderates the effect of parenting behavior on fear acquisition and extinction.
3. The association of 5-HTTLPR expression and anxiety during acquisition, extinction and follow-up measures is moderated by characteristics in emotion regulation

Conclusion
Limitations
The experiment was executed with healthy children only. Next children with anxiety disorders should be included. The time between the two extinction phases is too small. In the therapy study of Eley and colleagues the follow-up measure was six weeks after therapy.

Method
Differential fear conditioning data was experimentally obtained from 69 children without a anxiety disorder, aged six to 14 years. The children attended laboratory sessions on two sequent days together with one of their parents. Both completed the „Zürcher Kurzfragebogen zum Erziehungsverhalten (ZKE)“ (Reitzle, Winkler Metzke, & Steinhausen, 2001) and the FEEL-KJ (Grob & Smolenski, 2005).

Conditioning phases were habituation, acquisition and extinction on day one (Figure 1), and just habituation and extinction on day two. As the outcome measure, startle responses were obtained via electromyographic (EMG) activity over the orbicularis oculi muscle. Children’s DNA was obtained from saliva samples.

Results
Dropouts
One child had to be excluded because of social phobia and five children interrupt the experiment because of excessive demand.

What we expect

Figure 1: Diagram of the conditioning phases on day one and the stimuli of the fear conditioning.