Anxiety disorders and Attention Deficit Hyperactivity Disorder (ADHD) are highly prevalent childhood disorders and their symptoms can negatively interfere with general well-being, social life, academic performance, and development of social skills (Pine et al. 1998, Kendall et al. 2001, 2004). In the U.S, the prevalence of anxiety disorders has been estimated to be 5.7% in a cohort aged 9-13 yrs (Costello et al. 1996, 2003) and 13.0% in a cohort aged 9-17 yrs (Shaffer et al. 1996).

The neurobiology underlying the two disorders is only partially understood. An alteration of the reward system is supposed to be critical in ADHD leading to a bias toward approach behaviour, whereas perturbation in this system could contribute to the avoidance behaviour characteristic of anxious individuals.

Children with ADHD require stronger and more salient reinforcers to regulate their behavior than do healthy children, yet they respond excessively to novel stimuli as rewarding stimuli, i.e., generating approach behavior, often indiscriminately. They show less sensitivity to changes in reinforcement contingencies, and respond more strongly to negative than positive reinforcers (Ernst 2003). Neural correlates of behavioural control has been placed on neural circuits involving dorsolateral prefrontal (DLPFC) and anterior cingulated cortex (ACC). The ACC, especially the dorsal ACC, has strong connections to the DLPFC and is considered to play a critical role in complex cognitive processing (Bush et al 2000), particularly target detection, response selection, error
detection, and reward-based decision-making (Bush et al 2002), functions that are thought to be impaired in ADHD.

In contrast, anxiety disorders are characterized by a bias towards harm-avoidant behaviors. An obvious difficulty in the study of anxiety is the heterogeneity of disorders placed under the umbrella of anxiety disorders. Nonetheless, several theoretical models of generic anxiety have been proposed that focus on the interaction between cognition, affect, physiology, and behavior (for review, Wilken et al 2000). The association of stimuli with adverse affective experiences is a critical determinant of hyperarousal (Dowden and Allen 1997) and anxious apprehension, which occur across anxiety disorders. Accordingly, the neural substrates engaged in the processing of aversive stimuli have been implicated in the pathophysiology of anxiety. These include limbic (amygdala, ventral striatum) and paralimbic structures (orbitofrontal cortex, insula, ACC) (Ernst et al., 2003). Although a voluminous literature attributes a specialized role for harm avoidance to the amygdala (LeDoux, 2000) and for reward processing to the nucleus accumbens (Wise et al. 1992; Di Chiara, 2002), these structures support a number of additional functions, such as associative learning (Cardinal et al. 2002b; Salamone & Correa, 2002; Gabriel et al. 2003) and attention filtering (Pessoa & Ungerleider, 2004), which are perturbed in both ADHD and anxiety disorders.

Although anxiety appears to be biased toward avoidance and ADHD towards approach behaviour, these two disorders often coexist within individuals. Approximately 25% of children with ADHD have an anxiety disorder (Pliszka et al, 1999; Levy et al. 2004).

The aim of the present project is to explore alteration in the reward system in patients with ADHD and Anxiety disorder compared to healthy controls. Unique patterns of activity in the reward system could be helpful in understanding how this system contributes to motivated behaviour. In addition, a better understanding of the underlying neurobiological substrates of anxiety and ADHD should provide a priori hypotheses for future focused studies.
References


