Functional MRI studies in disruptive behaviour disorders

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Aggressive or antisocial behaviours with violations of social rules are the main features of disruptive behaviour disorders (DBDs), which are developmental diseases and include conduct disorder and oppositional defiant disorder. In the last decade, several efforts have been made to shed light on the biological underpinnings of DBDs. In this context, the main findings of functional magnetic resonance imaging studies in DBD are reported here. There are indications of neural dysfunctions in response to affective stimuli, especially regarding medial and orbitofrontal prefrontal cortex and connected subcortical structures.

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Disruptive behaviour disorders (DBDs) are common and severe developmental disorders include conduct disorder (CD) and oppositional defiant disorder (ODD). They are characterized by aggressive and antisocial behaviours associated with violations of social rules (e.g. thefts and violent behaviour) and are considered a predictor of antisocial/borderline personality disorder and substance abuse (Schutter et al. 2011). Also, they are more common in boys than in girls and are associated with a lower quality of life (Bot et al. 2011). High comorbidity is usually observed with attention deficit hyperactivity disorder (ADHD) (Spencer, 2006; Bellani et al. 2011). It should be noted that DBD children and preadolescents with comorbid ADHD show higher use of mental health services than those with mood or anxiety disorders and that ODD diagnosis is associated with the highest likelihood of the use of services at the age of 14–15 years (Ezpeleta et al. 2009). Recently, interest was addressed to a sub-group of DBDs with the so-called callous-unemotional traits (i.e. reduced empathy and guilt, poor emotional response), which may be associated with increased risk of antisocial outcomes (Marsh et al. 2008; De Brito et al. 2009).

Several functional magnetic resonance imaging (fMRI) studies have been conducted so far to explore the biological bases of DBDs (Table 1). Specifically, adolescents with DBD exposed to emotional pictures (i.e. negative/painful situations and fearful/sad faces) showed enhanced activation of amygdala, striatum, mid-cingulate and orbitofrontal cortex (OFC) (Herpertz et al. 2008; Decety et al. 2009; Passamonti...
et al. 2010) and reduced activation of anterior cingulate cortex (ACC) (Sterzer et al. 2005). Also, they had reduced functional connectivity between amygdala and medial prefrontal cortex (Marsh et al. 2008; Decety et al. 2009). In two studies, reduced activation of amygdala was instead observed in DBD adolescents with high callous-unemotional traits (Marsh et al. 2008; Jones et al. 2009). Interestingly, a reduced activation of amygdala, insula and PFC was also present in response to angry faces in CD (Passamonti et al. 2010). Such functional abnormalities are consistent with structural alterations showing volume reduction of amygdala, hippocampus, insula, OFC, dorsomedial PFC, temporal cortex and caudate nucleus, particularly in CD children (Huebner et al. 2008; Fairchild et al. 2011). However, increased sizes of medial OFC and dorsal ACC, with preserved amygdala and insula morphology, have also been found in boys with callous-unemotional traits and conduct problems (De Brito et al. 2009).

Based on the imaging literature briefly summarized above, a dysfunctional network including amygdala, PFC, striatum and insula may underlie affective dysregulation reported in subjects with DBD. In this context, preliminary findings suggest that DBD patients with callous-unemotional traits, particularly those with CD, may represent a specific sub-group with peculiar functional and structural brain maturation. Future fMRI studies should explore whether such abnormalities are specific to CD or ODD (Barker et al. 2011) and whether they persist over time during development. They should also further investigate the role of callous-unemotional traits for brain development in these children and should differentiate the specific features of DBD and ADHD (Rubia, 2011).

### References


