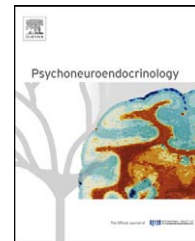




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Cortisol levels in response to starting school in children at increased risk for social phobia

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Summary

Background: Research on depression has identified hyperactivity of the HPA axis as a potential contributory factor to the intergenerational transmission of affective symptoms. This has not yet been examined in the context of social phobia. The current study compared HPA axis activity in response to a universal social stressor (starting school) in children of 2 groups of women: one with social phobia and one with no history of anxiety (comparison group). To determine specificity of effects of maternal social phobia, a third group of children were also examined whose mothers had generalised anxiety disorder (GAD).

Method: Children provided salivary cortisol samples in the morning, afternoon and at bedtime across 3 time-blocks surrounding the school start: a month before starting school (baseline), the first week at school (stress response), and the end of the first school term (stress recovery). Child behavioural inhibition at 14 months was assessed to explore the influence of early temperament on later stress responses.

Results: All children displayed an elevation in morning and afternoon cortisol from baseline during the first week at school, which remained elevated until the end of the first term. Children in the social phobia group, however, *also* displayed an equivalent elevation in bedtime cortisol, which was not observed for comparison children or for children of mothers with GAD. Children in the social phobia group who were classified as 'inhibited' at 14 months displayed significantly higher afternoon cortisol levels overall.

Summary: A persistent stress response to school in the morning and afternoon is typical for all children, but children of mothers with social phobia also display atypical elevations in evening cortisol levels when at school – signalling longer-term disruption of the circadian rhythm in HPA axis activity. This is the first study to report HPA axis disruption in children at increased risk of

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developing social phobia. Future research should determine whether this represents a pathway for symptom development, taking early temperament into account.

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1. Introduction

Social phobia is a disabling, chronic, anxiety disorder, characterised by excessive and persistent fear of scrutiny and negative evaluation, and marked restrictions in social functioning. Social phobia is one of the most common psychiatric disorders, with lifetime and 12 month prevalence rates of around 13% and 6%, respectively (Furmark, 2002). Onset is typically during adolescence (e.g. Schneier et al., 1992; Verhulst et al., 1997; Grant et al., 2005), although precursors are commonly seen earlier, and these can have a profound deleterious impact on child socio-emotional development (Beidel et al., 1999).

As with other affective disorders, social phobia aggregates in families, and clinical studies demonstrate specificity of transmission (i.e. first degree relatives of individuals with social phobia are at increased risk of social phobia, but not other anxiety disorders) (e.g. Maier et al., 1993; Fyer et al., 1995; Stein et al., 1998; Merikangas et al., 2003). Such specificity has been supported in both top-down and bottom-up studies. For example, Mancini et al. (1996) found that 23% of children of mothers with social phobia had the disorder; a four-fold increase over child base-rate. Similarly, elevated rates of life-time social phobia, but no other affective disorder, were reported in mothers of shy children, compared to mothers of children with another form of disturbance (e.g. fearfulness) or no disturbance at all (Cooper and Eke, 1999).

A genetic contribution to symptom development has been identified, but is modest, with an estimated heritability of around 10% (Nelson et al., 2000). This implies that additional mechanisms are important in the transmission of symptoms, and it appears that both child temperament and parenting style also play important aetiological roles (see review by Murray et al., 2009).

Recent research in the context of depression suggests that the involvement of physiological systems, particularly those implicated in the response to stress, such as the hypothalamic pituitary adrenal (HPA) axis, should also be considered in the transmission of affective disorders. The HPA axis is an essential part of the organism's homeostatic system, and one of the most reactive of all neuroendocrine systems. The mechanism through which the axis operates is characterised by the activation of a cascade of biochemical events, which begins with the release of corticotropin-releasing hormone (CRH) from the hypothalamus, and culminates with the release of glucocorticoids (primarily cortisol) from the adrenal gland (Antoni, 1986; Plotsky, 1991). HPA axis activity is one of the many bodily functions to display a distinct daily or diurnal rhythm. Cortisol levels are at their highest at awakening and then decline over the day, reaching near zero in the evening and remaining low until the later hours of sleep, when levels begin to increase (Clow et al., 2004). Imposed on this rhythm is a response to awakening, consisting of a 50–160% increase in the first 30–40 min after eyes open, the 'cortisol awakening response'

(CAR) (Fries et al., 2009); and this, in turn, fluctuates in anticipation of the stressors or challenges of the day.

While the release of glucocorticoids is critical for enabling adaptation to perturbations in homeostasis caused by both real and anticipated increases in demand, i.e., stress (Selye, 1973), chronically, or frequently elevated cortisol levels (e.g. raised baseline levels or poorly regulated stress responses) are associated with adverse effects on health, including the development of mental health problems, particularly affective disorders (Goodyer et al., 2001, 2003, 2009, 2010; Harris et al., 2000). A proportion of depressed adults are reliably reported to display hyperactivity of the HPA axis, as demonstrated by elevated baseline cortisol levels (particularly in the evening), hypertrophy of the adrenal gland, raised cerebral-spinal levels of CRH, and reduced negative feedback in response to administration of synthetic glucocorticoids, such as dexamethasone (Arato et al., 1989; Heuser et al., 1994; Rubin et al., 1995; Carroll et al., 2007). Furthermore, a recent meta-analysis confirms that increased HPA axis activity is also a feature of depression in both children and adolescents (Lopez-Duran et al., 2009). And this finding extends to include children at risk for depression as a result of their mothers having a history of the disorder (Halligan et al., 2004; Dougherty et al., 2009).

With regard to anxiety, elevated basal cortisol levels have been consistently reported in individuals with panic disorder (e.g. Wedekind et al., 2000), and, to a lesser extent, in individuals with OCD (Gustafsson et al., 2008) and GAD (e.g. Mantella et al., 2008). Furthermore, research suggests that comorbid anxiety may explain some of the HPA axis hyperactivity effects commonly observed in depression (Young et al., 2004). Research in the context of social phobia is more limited and findings have not always been consistent. However, some studies have reported elevated cortisol levels in response to stress in individuals with social phobia compared to controls (Furlan et al., 2001; Condren et al., 2002; Roelofs et al., 2009). For example, Roelofs et al. (2009) found that individuals with social phobia displayed significantly elevated stress-induced cortisol levels (i.e. post-stressor samples) following the Trier Social Stress Test than those with PTSD and a healthy comparison group. Moreover, stress-induced cortisol levels in the social phobia group were found to be positively correlated with an objective measure of social avoidance behaviour to angry faces manifested during an affect evaluation task, providing the first evidence of a link between HPA axis hyperactivity and levels of social avoidance behaviour in the context of a social phobia diagnosis.

There is more data on HPA axis functioning in relation to child temperamental traits thought to be associated with the development of social phobia, i.e., behavioural inhibition, or BI (Kagan, 1994). Inhibited/socially wary infants have been shown to display the highest cortisol levels under both stressed (e.g. Kagan et al., 1987) and non-stressed (e.g. Schmidt et al., 1997) conditions. Studies involving children undergoing normative stressors, such as entering novel peer-group settings (e.g. starting

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