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Cortisol and alpha amylase reactivity and timing of puberty: Vulnerabilities for antisocial behaviour in young adolescents

Elizabeth J. Susman [a,1], Samantha Dockray [b,*], Douglas A. Granger [a], Keeva T. Blades [a], William Randazzo [c], Jodi A. Heaton [a], Lorah D. Dorn [d]

[a] *Department of Biobehavioral Health, The Pennsylvania State University, 314 Health and Human Development East, University Park, PA 16802, USA*
[b] *Epidemiology & Public Health, University College London, 1-10 Torrington Place, London WC1E 6BT, United Kingdom*
[c] *387 Townhouse, The Pennsylvania State University College of Medicine/Milton S Hershey Medical Center, Hershey, PA 17033, USA*
[d] *Division of Adolescent Medicine, University of Cincinnati College of Medicine, 3333 Burnet Avenue Cincinnati, OH 45229-3039, USA*

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Summary The theoretical framework proposed that cortisol and saliva alpha amylase (sAA) reactivitiy are vulnerabilities for antisocial behaviour. These indices of hypothalamic-pituitary-adrenal (HPA) and sympathetic-adrenal-medulary (SAM) components of the stress system, respectively, were considered vulnerabilities that also interact with the putative stressful transition of timing of puberty to predispose adolescents toward antisocial behaviour. The sample consisted of 8- to-13-year-old boys and girls (N = 135) and a parent. For boys, timing of puberty moderated the association between cortisol and sAA reactivity and antisocial behaviour. Higher cortisol reactivity in *later* timing boys was related to a composite index of antisocial behaviour and rule-breaking behaviour problems. In contrast, lower sAA reactivity and *earlier* timing of puberty in boys was related to *rule breaking* and *conduct disorder symptoms*. The interaction between timing of puberty and HPA or SAM regulation and timing of puberty in boys suggests that reproductive, neuroendocrine mechanisms may be involved in the extensively documented adverse consequences of off-time pubertal development.

1. Introduction

Aggressive and antisocial behaviours may increase in some adolescents for a host of reasons including neuroendocrine and physical maturational changes, increasingly complex social roles, peer influences, and asynchronies between brain development and emotional and behavioural regulation. Recent studies suggest that these problems begin in later

* Corresponding author. Tel.: +44 0 20 7679 1805; fax: +44 0 20 7813 0242.
E-mail addresses: esusman@psu.edu (E.J. Susman), s.dockray@ucl.ac.uk (S. Dockray).
[1] Tel.: +1 814 863 2281; fax: +1 814 863 7525.

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childhood and the early adolescent years (Andrews et al., 2003) and become more problematic in mid to late adolescence. The problem is that neuroendocrine developmental transitions are rarely examined as influences on behaviour problems even though major neuroendocrine changes are the mechanisms responsible for pubertal development. Early adolescence is considered an especially stressful and vulnerable period for the expression of antisocial behaviour problems because of the rapid, neuroendocrine, puberty-related changes that are differentially timed for males and females. A promising mechanism linking early vulnerabilities and antisocial behaviour is the putative stress of differential timing of puberty. The purpose of this report was to test hypotheses regarding psychobiological, stress system vulnerabilities and the interaction between these vulnerabilities and timing of puberty and antisocial behaviour includes parent reports of externalizing behaviour problems and conduct disorder and oppositional defiant disorder symptoms. The theoretical perspective upon which the study is based integrates multi-level processes: reactivity of the hypothalamic-pituitary-adrenal (HPA) and sympathetic-adrenal-medullary (SAM) response to stress, timing of puberty as indexed by pubertal physical maturation, and antisocial behaviour.

1.1. Psychobiological vulnerabilities and timing of puberty

The specific heuristic model guiding this report proposes, first, that certain psychological risks (*vulnerabilities*) predispose adolescents to behaviour problems during puberty. This diathesis-stress model assumes that most individuals have some level of predisposing risk factors, or diatheses, for psychosocial problems (Richters and Weintraub, 1990; Abela and D'Alessandro, 2002). The tendency to develop psychosocial problems varies depending on the interaction between the degree to which risk factors are present and the degree of stress experienced by the individual (Richters and Weintraub, 1990; Monroe and Simons, 1991). We expanded this perspective by including HPA and SAM vulnerabilities as risks given the documented relation between these systems and antisocial behaviour (McBurnett et al., 1991; 2000; Raine, 2005; Shirtcliff et al., 2005; Gordis et al., 2006; Granger et al., 2006, 2007). Gordis et al. (2006) showed the importance of including both HPA and SAM stress regulation in understanding aggressive behaviour.

Second, the heuristic model includes the concept of *transitional stress* and describes the stress imposed by ambiguity related to individualization and destandardization of roles that accompany periods of change like puberty. Transitional stress with regard to puberty is directly linked to neuroendocrine and physical-maturational changes and predicts that an increase in adrenal and gonadal hormones and the physical changes of maturation lead to an increase in antisocial behaviours (Graber et al., 1997, 2004; Angold et al., 1999; Stoff and Susman, 2005; Ge et al., 2006). Transitional stress is especially acute if there is an asynchrony between the timing of the physical changes relative to same-age peers. Specifically, an earlier or later pubertal transition biases vulnerable individuals toward behaviour problems (Stattin and Magnusson, 1990; Caspi and Moffitt, 1991) in some but not all adolescents.

1.2. Biological vulnerabilities

A recent theoretical perspective (Bauer et al., 2002) suggests that arousal and recovery from reactivity, as reflected in reactivity to stressors entailing novelty and uncertainty, is representative of how individuals generally regulate their arousal. Difficulty in arousal regulation, including both high and/or sustained responsivity, is considered a vulnerability for behaviour problems (Kagan et al., 1994). Bauer et al. (2002) go on to suggest that differences in arousal may explain differences in susceptibility to the adverse effects of stressors. Variations in arousal, as indexed herein by cortisol and salivary alpha amylase (sAA) reactivity, reflect coordinated, yet distinct axes of the stress response system. Central components of the stress system are the corticotrophin-releasing-hormone (CRH) neurons of the endocrine HPA axis system and the locus coeruleus, norepinephrine system (LC/NE), and the sympathetic-adrenal-medullary (SAM) system (Chrousos and Gold, 1992). Individual differences in both HPA and SAM reactivity are expected to be related to antisocial behaviour.

1.2.1. Cortisol

From the prenatal period onward, regulatory patterns of cortisol are related to psychological functioning. For instance, low basal, maternal prenatal cortisol levels predicted difficult infant temperament in 3-year-old children (Susman et al., 2001). At the older end of the life span, higher basal cortisol levels were related to cognitive declines (Seeman et al., 1997; Kudielka et al., 2004a,b) and exaggerated cortisol reactivity (Préville et al., 2008). In addition, non-invasively collected salivary cortisol and its links to emotions and behaviours have been extensively validated in laboratory settings (Kirschbaum et al., 1992; Schwartz et al., 1998; Dickerson and Kemeny, 2004). With regard to antisocial behaviour, lower basal salivary cortisol levels are characteristic of individuals exhibiting disruptive behaviour problems (McBurnett et al., 1991; Susman et al., 1997; McBurnett et al., 2000; Oosterlaan et al., 2005), including oppositional defiant behaviour (van Goozen et al., 1998), conduct disorder (Vanyukov et al., 1993; Pajer et al., 2001;), habitual violence (Virkkunen, 1985), and abuse of others (Bergman and Brismar, 1994). Furthermore, low basal cortisol is both a concurrent correlate and risk factor for future alcohol use (Moss et al., 1995) and externalizing behaviour problems (Shirtcliff et al., 2005). With regard to cortisol reactivity, hyper-reactivity characterized the HPA axis response to stressors in some studies (Susman et al., 1997), whereas hyporeactivity was characteristic of antisocial boys in other studies (Fairchild et al., 2008). The precise mechanisms whereby inconsistencies occur are unknown (Dickerson and Kemeny, 2004). Nonetheless, age, dysfunction in the serotonin system, developmental differences between children and adults, composition of the sample and outcome measures are explanations of the inconsistencies (van Goozen et al., 2007), Given the similarity of our methods with those of previous studies that used a cognitive and social evaluative stressor, we expected that higher cortisol reactivity would be associated with antisocial behaviour.

1.2.2. Salivary alpha amylase

A new marker of stress reactivity, sAA is considered a surrogate marker of SAM activity (Granger et al., 2007; Stroud