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Endocrine markers of puberty timing and antisocial behaviour in girls and boys

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Abstract

Background: Early puberty is associated with higher than average risk of antisocial behaviour, both in girls and boys. Most studies of such association, however, have focused on psychosocial mediating and moderating factors. Few refer to coterminous hormonal measures.

Aim: The aim of this review is to consider the role of hormonal markers as potential mediating or moderating factors between puberty timing and antisocial behaviour.

Method: A systematic literature search was conducted searching Medline, Embase, Web of Science, Scopus, Psycinfo, Cochrane and Google Scholar.

Results: Just eight studies were found to fit criteria, all cross-sectional. Measurements were too heterogeneous to allow meta-analysis. The most consistent associations found were between adrenal hormones—both androgens and cortisol—which were associated with early adrenarche and antisocial behaviours in girls and later adrenarche and antisocial behaviour in boys.

Conclusions: The findings from our review suggest that longitudinal studies to test bidirectional hormone–behaviour associations with early or late puberty would be worthwhile. In view of the interactive processes between hypothalamic–pituitary–adrenal and hypothalamic–pituitary–gonadal axes, integrated consideration of the hormonal end products is recommended.

1 | INTRODUCTION

Antisocial behaviour in children and adolescents is defined as behaviour by which basic norms, rights and rules are violated. Antisocial behaviour includes various inappropriate behaviours such as oppositional and aggressive behaviour, disruptive behaviour disorders and delinquency. Such behaviour disorders in children increase risk of several negative outcomes in adulthood including criminal offending, unemployment and psychiatric disorders such as mood disorders, anxiety disorders and substance abuse (Farrington, 1995; Maughan & Rutter, 2001). Disruptive behaviour disorders (DBDs), covering both oppositional defiant disorder (ODD) and conduct disorder (CD) can start in early childhood or adolescence, and often continue into adulthood, putting a heavy burden on individuals, families, justice and health care systems (Moffitt, 2006; Odgers et al., 2008). During puberty, numerous alterations occur in the brain and the body, including hormonal changes, physical development and reorganisation of different brain structures. Whereas puberty typically refers to the activation of the hypothalamic–pituitary–adrenal axis (HPA-axis) eventually resulting in gonadal maturation (Sisk & Foster, 2004), adolescence is rather conceived as the developmental stage between childhood and adulthood, largely covering the second decade of life, in which social and cognitive behaviours mature.

Two components of puberty, adrenarche and gonadarche, are important in the study of puberty timing (Grumbach & Styne, 2003). Adrenarche typically starts between ages 6 and 9 years and refers to the maturation of the HPA-axis. In this period, adrenal androgens like dehydroepiandrosterone (DHEA) and its sulphate (DHEA-S) begin to rise. The diagnosis of premature adrenarche (PA) is acceptable when clinical signs of androgen action, such as adult type body odour, appear before the age of 8 years in girls or 9 years in boys and are associated with adrenal androgen concentrations which are high for the chronological age (Voutilainen & Jääskeläinen, 2015). Gonadarche, generally starting between 9 and 11 years, refers to the maturation of the hypothalamic–pituitary–gonadal (HPG) axis. The hypothalamus stimulates hormones including gonadotropins (follicle stimulating hormone [FSH] and luteinising hormone [LH]), which in turn stimulate sex steroids, such as testosterone, oestradiol and progesterone, which influence gonadotropin production, and so on in a feedback loop. These hormones rise rapidly during the pubertal transition (Nottelmann et al., 1987). Early maturity may bring imbalance in behavioural adaptation since the brain's sensitivity to HPA/HPG axis hormones may be higher than among young people who mature at the usual time. Both during and after completion of puberty early maturers may secrete higher doses of these hormones (Mendle, Turkheimer, & Emery, 2007).

As well as timing of puberty onset, pubertal stage and tempo must be considered. Pubertal stage, generally rated according to the Tanner Scale, refers to the development of external, physical primary and secondary sexual characteristics and is highly correlated with age, whereas puberty timing refers to the degree of pubertal maturation relative to that of same-sex and same-age peers and is often referred to as early, on-time or late pubertal timing. Tempo describes the rate at which individuals progress from Tanner Stage I to Stage V (full sexual maturity). Most research has emphasised puberty timing as particularly relevant for understanding adolescence onset of DBDs. Higher pubertal stage in itself is likely to be related to aggressive and rule breaking behaviours (Hemphill et al., 2010; Oldehinkel, Verhulst, & Ormel, 2011), while fast tempo has been associated with externalising behaviours in both boys and girls (Beltz, Corley, Bricker, Wadsworth, & Berenbaum, 2014; Marceau, Ram, Houts, Grimm, & Susman, 2011). Kretschmer, Oliver, and Maughan (2014) showed that both earlier timing and faster tempo predicted delinquency in both girls and boys.

The position is less clear with respect to any relationships between the hormonal end-products of HPA (cortisol and DHEA(-S)) and HPG axes (testosterone and oestradiol) and adolescent DBDs or antisocial behaviour. Alink et al. (2008), in a meta-analysis, found that externalising behaviours were not significantly associated with cortisol levels in adolescents, while in elementary school-aged children there was an association with lower basal cortisol (hypoactivity). A separate analysis for cortisol reactivity showed no association. In some individual studies, rapid increase in DHEA and DHEA-S during adrenarche has been positively related to CD and ODD in boys (Mundy et al., 2015; Van Goozen, Matthys, Cohen-Kettenis, & Buitelaar, 2000; Van Goozen, Matthys, Cohen-Kettenis, Thijssen, & Van Engeland, 1998) and girls (Belsky, Ruttle, Boyce, Armstrong, & Essex, 2015). In others, negative associations were found among boys (Nottelmann et al., 1987; Susman et al., 1987) or no association (Shirtcliff, Zahn-Waxler,

Klimes-Dougan, & Slattery, 2007). Among girls, two studies found a negative association (Nottelmann et al., 1987; Susman, Granger, Murowchick, Ponirakis, & Worral, 1996).

Testosterone levels rise up to 30-fold during puberty in boys. In adolescents testosterone levels and aggressive behaviour have been associated in childhood-onset persistent antisocial behaviour (Maras et al., 2003), clinic-referred adolescents (Scerbo & Kolko, 1994) and male offenders (e.g. Dabbs, Jurkovic, & Frady, 1991). In some community-based studies these associations have not been replicated from age 16 on (Tremblay et al., 1997; Van Bokhoven et al., 2006). For oestradiol, a review on the relationship of oestradiol with aggression, CD and delinquency yielded no significant correlations (Balzer, Duke, Hawke, & Steinbeck, 2015).

Most studies on the association of puberty timing and DBDs have focused on psychosocial mediating and moderating factors, such as ethnicity, peer and other environmental influences (Celio, Karnik, & Steiner, 2006; Graber, 2013; Mendle et al., 2007; Negriff & Susman, 2011). Previous reviews have focused *either* on the relationship between puberty timing or tempo and antisocial behaviour *or* on hormonal (adrenal or gonadal) associations with antisocial behaviour and DBDs, but not taken both together. Furthermore, in the light of the possibility that different results found for hormonal assessments in antisocial behaviour during adolescence and because puberty timing may influence antisocial behaviour differently depending on sex (see, Ullsperger & Nikolas, 2017), we were interested in sex differences with respect to these hormones. Our aim was to review the evidence for the following research questions:

1. Which associations have been found for adrenal and/or gonadal hormones in studies focusing on the relationship between puberty timing and antisocial behaviour?
2. Are the associations with these adrenal or gonadal hormones different for girls and boys?

2 | METHOD

A systematic literature search for publications on puberty timing/tempo, adrenal and gonadal hormones and antisocial behaviour, with narrative analysis, was undertaken using an adaptation of the Sample, Phenomenon of Interest, Design, Evaluation, Research (SPIDER) model (Cooke, Smith, & Booth, 2012). Relevant studies on puberty timing and antisocial behaviour were identified by using Medline (Ovid SP), Embase, Web of Science, Scopus, Psycinfo, Cochrane and Google Scholar. Search terms included ("puberty," "menarche," "adrenarche") AND ("antisocial behaviour," "criminal behaviour," "delinquency," "violence," "aggression," "conduct disorder," "externalising"). For the full list and search strategies adjusted for each database, see Appendix S1. The SPIDER model structured the search terms and eligibility of the criteria (see Table 1). The search covered the period January 1, 1985 to March 28, 2018, as the research on biological correlates of antisocial behaviour started in the mid-1980s. Exclusion and inclusion criteria for paper selection are listed in Table 1. In brief we included papers about children/adolescents aged 5–18, which measured relevant hormones and puberty timing or tempo but not staging and scaled measures of antisocial or disruptive behaviours. We excluded studies on known genetic or organic causes of precocious puberty, as these are rare and are often complicated by somatic comorbidity for which treatment like, for example, brain surgery or administration of gonadotropin releasing hormone agonists is needed.

Title and abstract were screened by two independent researchers. An overall agreement level of 95% was achieved. In a final meeting the remaining 5% were discussed and 100% consensus was achieved.

3 | RESULTS

The searches yielded 2,811 unique titles (see Figure 1). In total, 124 articles were selected for full text reading. Within these results we excluded existing reviews on the association of puberty timing or tempo and antisocial behaviour and included studies in which HPA/HPG axis hormones were studied as potential mediators or

TABLE 1 SPIDER inclusion–exclusion criteria

	Inclusion	Exclusion
Sample	Children–adolescents >5 years with externalising disorders	Substance use disorders or ADHD without antisocial behaviour
		Children: 0–5 years
		Adults: >18 years
Phenomenon of interest	Studies on puberty timing in boys/girls/mixed gender combined with hormonal measure (HPA and/or HPG axis)	Puberty staging or pubertal tempo
		Studies on puberty timing without hormonal measures or other hormonal measures
		Central or peripheral precocious puberty with known genetic or organic cause
Design	Peer-reviewed journals with all empirical approaches (observational, retrospective, prospective, cross-sectional, cohort studies)	Non-peer reviewed journal, book chapter, conference proceedings, PhD thesis
		Review articles, meta-analysis, opinion, new direction
Evaluation	Hormonal (HPA and/or HPG axis) measures associated with puberty timing or tempo	Other hormonal measures or neuropeptides, for example, vasopressin or oxytocin
Research type	Peer-reviewed journal articles published between 1985 and March 2018 with full text available in English	Peer-reviewed journals published before January 1985 or after March 2018
		Full text in other language than English

Abbreviation: ADHD, attention deficit hyperactivity disorder; HPA, hypothalamic-pituitary-adrenal; HPG, hypothalamic-pituitary-gonadal; SPIDER, "Sample, Phenomenon of Interest, Design, Evaluation, Research" Model.

independent variables, although checked their reference lists, leaving eight studies for inclusion which incorporated hormone assessments in the association between puberty timing and onset of antisocial behaviour (see Table 2).

In all included studies antisocial behaviour of some kind had to be treated as the dependent variable and puberty timing as an independent variable. Puberty timing was measured in different ways between studies. The most common methods were variants of Tanner staging (genital and pubic hair stage for boys; breast and pubic hair stage for girls) by physical examination, or by children's or adolescents' reports and/or parent report by confirming stage according to standard Tanner pictures, or by the Pubertal Development Scale (Petersen, Crockett, Richards, & Boxer, 1988), a five-item self-report questionnaire that assesses body hair, growth speed and secondary sex changes in both girls and boys. In studies of girls only, self-reported age of menarche was accepted as evidence of timing. These methods are not interchangeable in terms of validity. Clinician-reported Tanner stages are more accurate than other methods, while self-reported age of menarche has been shown less valid in longitudinal studies (Dorn, Dahl, Woodward, & Biro, 2006). Early puberty timing, based on cut-off scores for Tanner staging, was compared to on-time or late onset adolescents within most studies; in two studies (Graber, Brooks-Gunn, & Warren, 2006; Sontag, Graber, Brooks-Gunn, & Warren, 2008) the sample was stratified according to national norm groups adjusted for age, gender and ethnicity.

We grouped studies that specifically dealt with timing of adrenarche and gonadarche separately because these are independent events controlled by separate mechanisms (Ibanez, Dimartino-Nardi, Potau, & Saenger, 2000).

3.1 | Timing of adrenarche and behavioural difficulties among girls

Girls with DHEA(S) PA, meaning high levels of DHEA and Tanner Stage 2 pubic hairs in 6–8-year-olds, had higher adrenal androgen concentrations than on-time adrenarche girls and they more often met criteria for ODD and DBD according to

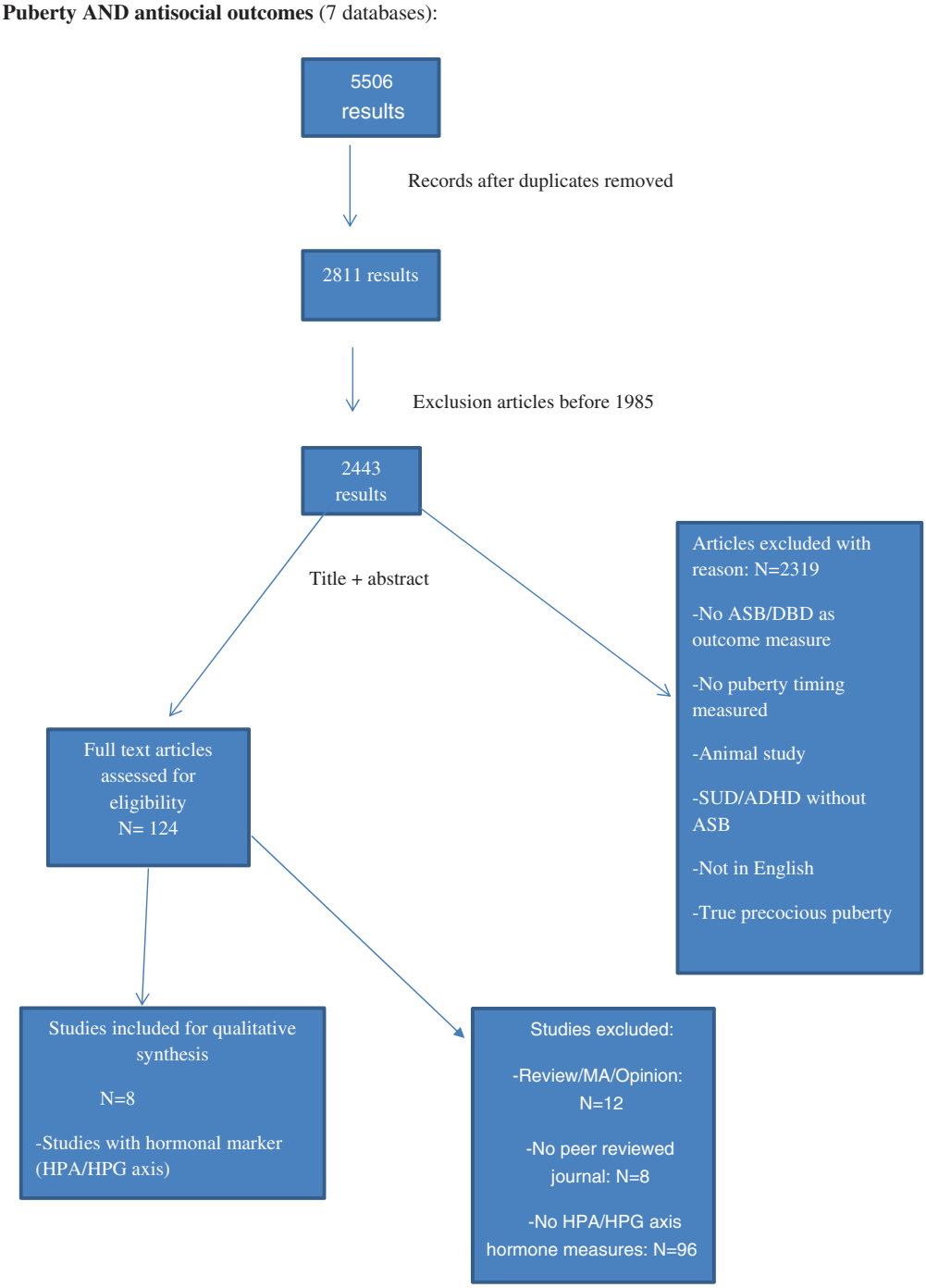

FIGURE 1 PRISMA flow diagram search. ADHD, attention deficit hyperactivity disorder; ASB, antisocial behaviour; DBD, disruptive behaviour disorders; HPA, hypothalamic-pituitary-adrenal; HPG, hypothalamic-pituitary-gonadal; MA, meta-analysis; SUD, substance use disorders [Colour figure can be viewed at wileyonlinelibrary.com]

TABLE 2 Studies on puberty timing, HPA–HPG end-products and antisocial behaviour

Kolom1	Kolom2	Kolom3	Kolom4	Kolom5	Kolom52	Kolom6	Kolom7	Kolom8	Kolom82	Kolom9	Kolom10	Kolom11
Author (year)	N	Gender	Setting	Design	Number of measurements	Hormones	Measurement	Age	Puberty measure	Outcome measure	Instruments	Results
Dorn et al. 1999	9 + 21 controls	8 girls	Pediatric endocrine department	Cross-sectional	3 times (T0,T20, T40)	DHEA	Serum	7.8 (± 1.3) years for PA	Tanner staging pubic hair: PA group had stage 2 or 3 versus stage 1 for on-time group	Externalising Behaviour	CBC-L	Higher score in parent reported CBC-L scores
		1 boy			(every 20 minutes)			8.0 (±) 1.2) years for on-time adrenarche	Comparison between groups			on externalising (t = 2.83; p = 0.01) and total behaviour problems (t = 2.86; p = 0.01)
					3 times (T0, T20, T40)	DHEA-S	Serum					in PA children versus on-time adrenarche
					3 times (T0, T20, T40)	Estradiol (E2)	Serum					Aggressive (t = 1.83) and delinquent scale (t = 1.71) non-significant
					3 times (T0, T20, T40	Cortisol	Serum + saliva					DHEA, DHEA-S, E2 and cortisol scores increased in PA group
Dorn et al. 2008	40 +36 controls	girls	Endocrinology department, USA	Cross-sectional	3 times (T0, T20, T40)	DHEA	Serum	7.7 ± 0.9 for PA	Tanner staging pubic hair: PA group had stage 2 or 3 versus stage 1 for on-time group	Aggressive Behaviour	CBC-L/TRF	Girls with PA had a higher rate for ODD in the past month (17,5% vs 0%; p = 0.02) and higher scores for externalising behaviour
					3 times (T0, T20, T40)	Androstenedione		7.4 ± 0.8 for on-time	PA defined as Tanner 1 breast or Tanner 2 pubic hair for girls age 6-8 years	ODD	DISC	PA girls had concentrations of DHEAS (p = 0.0003), androstenedione (p = 0.0001),Testosterone (p = 0.002) than on-time girls

TABLE 2 (Continued)

Kolom1	Kolom2	Kolom3	Kolom4	Kolom5	Kolom52	Kolom6	Kolom7	Kolom8	Kolom82	Kolom9	Kolom10	Kolom11
Author (year)	N	Gender	Setting	Design	Number of measurements	Hormones	Measurement	Age	Puberty measure	Outcome measure	Instruments	Results
					3 times (T0, T20, T40)	Testosterone						For cortisol and estradiol no differences were found between groups
					3 times (T0, T20, T40)	Cortisol						
					3 times (T0, T20, T40)	Estradiol						
Graber et al. 2006	100	girls	Private School, USA	Cross-sectional	1 x	Estradiol	Serum	12.13 (mean)	Physical examination	Aggressive Behaviour	YSR	Puberty timing and estradiol levels not associated with aggression
						DHEA-S			Tanner staging			Puberty timing and DHEA-S negatively associated with aggression ($r = -0.21$; $p < 0.05$)
									Classification as early/ on-time/late			
									according to stages by age from the National Health Examination Survey			
Sontag et al. 2008	111	girls	School, USA	Cross-sectional	5 samples every 15-20 minutes	Cortisol (reactivity)	Saliva	11.84 ± 0.77	Self-reported age of menarche	Aggressive Behaviour	YSR	Girls with early menarche had more cortisol reactivity to a stress test

(Continues)

TABLE 2 (Continued)

Kolom1	Kolom2	Kolom3	Kolom4	Kolom5	Kolom52	Kolom6	Kolom7	Kolom8	Kolom82	Kolom9	Kolom10	Kolom11
Author (year)	N	Gender	Setting	Design	Number of measurements	Hormones	Measurement	Age	Puberty measure	Outcome measure	Instruments	Results
									early menarche defined as one half SD below the mean White or American age norms.			and scored higher on aggression than girls in the on-time/late menarche group.
									Comparison group with on-time and late maturers			
Sontag-Padilla et al. 2012	40 + 36 controls	girls	Endocrinology department, USA	Cross-sectional	3 samples (0,20,40 min)	Cortisol reactivity	Serum	7.65 ± 0.90 PA girls	PA defined as Tanner 1 breast, Tanner 2 pubic hair, age 6-8	Externalising Behaviour	CBC-L	Girls with PA who demonstrated increased cortisol reactivity
						DHEA		7.53 ± 0.77 control group				had higher externalising symptoms compared to the on-time group
						Androstenedione						
Susman et al. 2007	111	55 girls	Community, USA	Cross-sectional	3 samples (0,20,40 min) in the morning	Cortisol	Saliva	Girls 10.49 ± 1.51	Physical exam and interview on Tanner stages using pictures	Aggression/ Rule Breaking	CBC-L	Timing of puberty did not interact with A.M to P.M. cortisol ratio
		56 boys			additional samples at noon,			Boys 11.44 ± 1.63		Conduct Disorder	DISC-IV	There was a direct effect of early puberty on Conduct Disorder symptoms in boys and relational aggression in girls
					4 PM and at bedtime							

TABLE 2 (Continued)

Kolom1	Kolom2	Kolom3	Kolom4	Kolom5	Kolom52	Kolom6	Kolom7	Kolom8	Kolom82	Kolom9	Kolom10	Kolom11
Author (year)	N	Gender	Setting	Design	Number of measurements	Hormones	Measurement	Age	Puberty measure	Outcome measure	Instruments	Results
Susman et al. 2010	135	69 boys	Community, USA	Cross-sectional	5 samples within 15 minutes	Alpha-Amylase	Saliva	10.06 ± 1.644 girls	Physical exam and interview on Tanner stages using pictures	Aggression/ Rule Breaking	CBC-L	Higher cortisol reactivity was related to antisocial behaviour and rule breaking in late timing boys
		66 girls			5 samples within 40 minutes	Cortisol		10.94 ±1.607 boys		Conduct Disorder	DISC-IV	Lower alpha-amylase reactivity and early puberty were related to rule breaking and CD symptoms
Whittle et al. 2015	100	54 girls	Community, Australia	Cross-sectional	Samples on two consecutive days	Alpha-Amylase	Saliva	9.44 ±0.31 girls	Parent report of puberty development	Externalising Behaviour	CBC-L	Early adrenarche defined by high DHEA levels was associated with externalising symptoms in girls
		46 boys			after waking	DHEA		9.56 ± 0.38 boys	using the Sexual Maturity Status Line report			

Abbreviation: ADQ, adolescent delinquency questionnaire; CBC-L, child behaviour checklist; CD, conduct disorder; DHEA, dehydroepiandrosterone; DHEA-S, dehydroepiandrosterone-sulphate; DISC-IV, diagnostic interview schedule for children; HPA, hypothalamic-pituitary-adrenal; HPG, hypothalamic-pituitary-gonadal; ODD, oppositional defiant disorder; PA, premature adrenarche; PDS, pubertal development scale; SD, standard deviation; TRF, teacher rating form; YSR, youth self-report.

Dorn et al. (2008). In contrast, Sontag-Padilla et al. (2012) found no evidence for an association between increased DHEA-S and androstenedione and externalising symptoms in PA girls and only a trend interaction between DHEA-S and lower levels of executive functioning with more externalising problems (Sontag-Padilla et al., 2012). Whittle et al. (2015) found higher DHEA levels to be associated with externalising symptoms in girls, but not boys, and found decreased affect-related brain activity in the mid-cingulate cortex and some other cortical and subcortical regions.

Girls with PA and higher serum cortisol levels measured after a stress test had more externalising behaviours than those with stable patterns (Sontag-Padilla et al., 2012). Dorn, Hitt, and Rotenstein (1999) found higher cortisol levels in a small sample of on-time girls, but in a later study (Dorn et al., 2008) they found no differences in cortisol between premature and on-time adrenarche girls.

Dorn et al. (2008) measured testosterone and oestradiol as well and found that testosterone levels were significantly higher for the PA girls who were more likely to have been given a diagnosis of ODD than the on-time group.

3.2 | Timing of gonadarche and menarche

In one study, girls with early menarche who experienced higher levels of peer stress and exhibited stronger cortisol reactivity to a stress test were at greater risk of aggression, although this relationship was moderated by emotional/cognitive numbing on the stress test (Sontag et al., 2008). Susman et al. (2010), by contrast, found no association between puberty and cortisol levels and behaviour among girls. In an earlier study, Susman et al. (2007), arguing that the morning: evening cortisol ratio is more reliable than a single morning cortisol sample, examined the hypothesis that early puberty timing and a low morning to evening cortisol ratio would be related to antisocial behaviour. Among 55 girls they found that puberty timing did not interact with the morning-to-evening cortisol ratio but did describe a general effect for timing of puberty on antisocial behaviour.

Graber et al. (2006) found a weak negative association between DHEA levels and aggression in 100 girls, but in this study no significant association was found between puberty timing and aggression, neither were oestradiol or DHEA tested as a mediator or moderator in this association. For oestradiol no association was found with aggression. The main conclusion of this study was that early puberty timing predicted higher emotional arousal and later depression, but not other behavioural disturbances.

3.3 | Adrenarche and gonadarche among boys

With respect to adrenarche, Whittle et al. (2015) found no association between higher DHEA levels and externalising behaviour. Susman et al. (2010) found higher cortisol reactivity in later timing boys was related to a composite index of antisocial behaviour. In the study by Susman et al. (2007), like in girls, a general effect of early puberty timing was found on antisocial behaviour, irrespective of the morning-to-afternoon cortisol ratio.

No studies were found on boys and hormones of gonadarche, like testosterone or oestradiol.

4 | DISCUSSION AND FUTURE DIRECTIONS

In this review we sought to examine associations between adrenal and gonadal hormones and puberty timing as independent variables and antisocial behaviour as the dependent variable and found some support for an association between pre-six-year-old detection of adrenal androgens, such as DHEA, indicative of early adrenarche, and externalising symptoms in girls. The only study to include boys found no such association, but numbers were small (Whittle et al., 2015).

4.1 | DHEA and premature adrenarche in girls

A possible explanation for the findings among girls is that early adrenarche and DHEA rise may be a reflection of early life stress. Early life stress as a risk factor for later externalising disorders has been shown to have a long-lasting impact into adolescence on HPA-axis functioning (Essex et al., 2011; Roisman et al., 2009). Early adversity like absence of father or "negative parenting" before age 5 are thought to impact on puberty timing and tempo in girls (Ellis, 2004; Ellis, Shirtcliff, Boyce, Deardorff, & Essex, 2011; Zhang, Zhang, & Sun, 2019). From a developmental perspective the results found for girls may fit with the accentuation theory (Ge & Natsuaki, 2009; Skoog & Stattin, 2014), which implies transitional stress at puberty onset tends to accentuate previous emotional and behavioural difficulties.

4.2 | Cortisol reactivity in girls and boys

As for cortisol reactivity girls with PA (Sontag-Padilla et al., 2012), girls with premature gonadarche (Sontag et al., 2008) and late timing boys (Susman et al., 2010) showed increased cortisol reactivity relating to higher risk of antisocial behaviours. When examining only the relationships between cortisol levels and externalising behaviours, Alink et al. (2008) found no overall relationships between them, so it seems likely that further study of this taking account of pubertal timing would be warranted.

The difference between boys and girls in this respect is in itself worthy of further exploration. While early puberty seems a candidate for a problematic mediator among girls it is late puberty in boys (Dorn et al., 2003; Graber, Seeley, Brooks-Gunn, & Lewinsohn, 2004). Consideration of stress as a further mediator seems crucial, with early abuse being a common stress precursor among studied girls but the late gonadarche a stress in itself among the boys. Later maturing boys may be treated more according their stature than their biological age, possibly resulting in exclusion from same-age peer activities (Lindfors et al., 2007).

4.3 | Testosterone and premature adrenarche in girls

Testosterone was positively associated with ODD (Dorn et al., 2008) in girls with PA versus on-time adrenarche. Similarly, they had higher DHEA-S and androstenedione concentrations, which are precursors for the production of testosterone. In girls testosterone has been linked to CD and DBD (Granger et al., 2003; Pajer et al., 2006), but in community samples no significant association was found (Dorn et al., 2009; Maras et al., 2003). These findings may fit with the diathesis stress model, which proposes that some children are vulnerable to exogenous stressors and endogenous stressors like the changes in endocrine milieu at puberty (Susman, Peckins, Bowes, & Dorn, 2017; Zuckerman, 1999). While no study in this review calculated testosterone/cortisol ratios it is worth mentioning the testosterone/cortisol ratio has been positively associated with overt aggression in studies in adolescents in either clinical or detention settings (Dabbs et al., 1991; Popma et al., 2007; Tackett, Herzhoff, Harden, Page-Gould, & Josephs, 2014). Hypothetically, cortisol and testosterone play a more significant role in early maturers: testosterone affects behaviour in an opposite way compared to cortisol. It stimulates behavioural activation, induces aggression and reduces fear and social withdrawal (Hermans, Putman, & Van Honk, 2006). Cortisol increases fear, punishment sensitivity and behavioural inhibition (Schulkin, 2003; Van Honk & Schutter, 2006). The balance between high testosterone levels and low cortisol levels shifts an individual to low punishment-high reward sensitivity (Carver & Harmon-Jones, 2009) and risky decision making as measured with the Iowa Gambling Task (Van Honk, Schutter, Hermans, & Putman, 2003). It is important to note HPA and HPG axes are highly interactive and have antagonistic properties (Viau, 2002). For example, DHEA is derived from both the adrenal cortex and testes and is an important precursor for testosterone in males and oestradiol in females.

4.4 | Strengths and limitations

This is a first review of the inter-relationships between hormonal measures as indicative of the timing of puberty and how these relate to antisocial behaviours. Our search was systematic but limited to literature databases and follow up of references in included articles, however in a topic like this, it is unlikely that there will be grey literature unless possibly in the form of unpublished theses.

Main limitations, therefore, are in the small number of studies found and that the diversity of measures meant that meta-analysis was impossible. Further, all studies we included were cross-sectional, so neither directional nor causative conclusions can be drawn with any safety. It may be of interest that all studies found on hormonal markers were with community-based populations, so it may be that we were not able to tap into true disorder level antisocial behaviour disorders and this may partly account for the mixed findings. Finally, although stress was invoked as relevant, it was inconsistently measured.

5 | CONCLUSIONS

In conclusion we found some evidence for adrenal hormone, notably DHEA, indicative of PA in girls, being associated with antisocial behaviour. This adds to preliminary knowledge on the role of PA as a risk factor for antisocial behaviour. Contrary to expectation, deviant puberty timing was related to increased cortisol reactivity in both sexes, but early in girls and late among boys. The most important hormones relevant at puberty onset are adrenal in origin—both androgens (DHEA) and cortisol. Although sample sizes were too small to allow firm conclusions, this indicates a potentially useful way forward in research, although in view of the feedback loops between hormonal axes, we recommend measures of all relevant hormones. Further directions for research would include prospective evaluation of long-term patterns of antisocial behaviours during and after puberty development and hormonal measures at standard time points and the mediating or moderating effect of stressors and their timing.

CONFLICT OF INTEREST

None.

DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no new data were created or analysed in this study.

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