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# Baby was a black sheep: Digit ratio (2D:4D), maternal bonding and primary and secondary psychopathy

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ABSTRACT

Psychopathy is generally considered to be a male adaptation. While studies have elucidated a relationship to freely circulating testosterone, less is known about the role of prenatal testosterone (PT) in the development of primary and secondary psychopathy and how this pertains to sex differences. In this study ($N = 148$), digit ratio (2D:4D) was used to investigate the relationship between prenatal testosterone and primary and secondary psychopathy. In addition, quality of recalled maternal bonding was measured to see if postnatal experience could affect the influence of PT on psychopathic behaviours. Low LH2D:4D predicted primary and secondary psychopathy in women. In men, low maternal care predicted primary psychopathy and high maternal protection predicted secondary psychopathy. Low maternal care also predicted primary psychopathy in women. Lower levels of maternal care and higher levels of maternal control contributed to primary psychopathy above and beyond PT. Lower levels of maternal care were also an influential factor for secondary psychopathy above and beyond PT, although higher levels of mother control were not.

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## 1. Introduction

Although there is extensive research on the development of primary and secondary psychopathy, the contribution of prenatal hormones currently remains relatively under-investigated. Psychopathy is hypothesized as a male-typical personality style (Jonason, Li, Webster, & Schmitt, 2009) and is related to circulating testosterone (Stålenheim, Eriksson, von Knorring, & Wide, 1998; van Honk & Schutter, 2006), therefore prenatal testosterone (PT) could be a factor in its development. Maternal stress may elevate prenatal testosterone levels, which, from an evolutionary perspective, could indicate the process of fetal programming - the mechanism by which prenatal development is adjusted according to in utero hormonal changes caused by maternal experience (Del Giudice, 2012). Postnatal experience, such as relationship quality between mother and child, may either reinforce or negate the effect of fetal programming. Therefore, we investigated the contribution of PT and quality of mother–child relationships in the development of primary (i.e., callous and exploitive predisposition) and secondary (i.e., risky and impulsive behaviours) psychopathic traits and behaviours in men and women using the 2D:4D digit ratio (as a biomarker for PT) and recalled maternal bonding.

Psychopathy, PT and parenting practices can be contextualised within a Life History theoretical framework. People vary in a fitness optimising strategy continuum from slow (i.e., high parenting and low mating effort) to fast (i.e., low parenting and high mating effort), which is regulated in response to cues signalling information about socio-ecological conditions (Kaplan & Gangestad, 2005). Primary and secondary psychopathy are putative fast life-history strategies. Psychopathic individuals use deception and antisocial behaviours to exploit others for resources and mating opportunities (Mealey, 1995) and exhibit short-term mating behaviours such as mate poaching (Kardum, Hudek-Knezevic, Schmitt, & Grundler, 2015) and sexual coerciveness (Muñoz, Khan, & Cordwell, 2011). Being psychopathic could be successful in harsh environments, as a "live fast, die young" (have more children) strategy.

From a developmental perspective, to adopt a mating strategy that will optimise fitness, a child should be sensitive to cues that signal information about the environment before puberty. Inadequate parental care may be one such proximate trigger. Children are more likely to have experienced sub-optimal parenting in harsh socio-ecological conditions (Pinderhughes, Nix, Foster, & Jones, 2001). Parenting also plays a crucial role in the development of fast life history strategies (Lukaszewski, 2015), and psychopathic traits and behaviours (Beaver et al., 2014). Sub-optimal maternal bonding is associated with primary and secondary psychopathic traits and behaviours (Blanchard & Lyons, 2016; Gao, Raine, Chan, Venables, & Mednick, 2010). However, what remains un-investigated is whether information about the environment

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can reach an unborn child, prompting development of psychopathic traits and behaviours. The mechanism in this case is "fetal programming", specifically, the alteration of in-utero hormone levels that change the fetal neurobiological development (Del Giudice, 2012). Therefore, the connection between high levels prenatal maternal stress and higher levels of PT implicates PT as a proximate trigger in the development of psychopathic traits and behaviours.

The precise mechanism between prenatal stress and elevated levels of PT is not clear, although increased cortisol caused by the activation of the hypothalamic–pituitary-adrenal (HPA) axis in response to stress is implicated (Barrett & Swan, 2015; Gitau, Adams, Fisk, & Glover, 2005; Sarkar, Bergman, O'Connor, & Glover, 2008). One hypothesis suggests that biological changes caused by maternal stress eases transference of maternal cortisol into the placenta, which then augments adrenal, ovarian/testicular function of the fetus (Barrett, Redmon, Wang, Sparks, & Swan, 2014). Although evidence demonstrates that the link between maternal stress and PT pertain only to female fetuses (Ward & Weisz, 1984). There are comparable behavioural outcomes for children subjected to stress prenatally and those exposed to higher levels of PT. Maternal anxiety is associated with externalising behaviours and emotional problems in children (O'Connor, Heron, Golding, & Glover, 2003; Van Den Bergh & Marcoen, 2004), while PT is associated with a range of psychopathic-type behaviours. In men these include physical aggression (Bailey & Hurd, 2005), sensation seeking and boredom (Fink, Neave, Laughton, & Manning, 2006). In women, PT is related to low empathy and aggression (Benderlioglu & Nelson, 2004; Kempe & Heffernan, 2011). Only one study previously has investigated PT and psychopathy (Blanchard & Lyons, 2010), and contrary to expectations, found higher levels of prenatal estrogen were associated with overall psychopathy in females and callous affect in males. Nevertheless, the general lack of research on psychopathy in this area highlights the need for further investigation.

Another question that remains relatively unexplored relates to sex differences. As men consistently score higher in psychopathy, psychopathy is generally considered as a male adaptation (Jonason et al., 2009). Less is known about female psychopathy (Rogstad & Rogers, 2008), so developmental trajectories to psychopathy could be different in women. Similar proximate triggers are implicated in both sexes such as adverse childhood experiences (Craig, Gray, & Snowden, 2013; Krischer & Sevecke, 2008; Mack, Hackney, & Pyle, 2011). However, when these triggers take effect maybe determined by when they have the most adaptive impact on reproductive schedule. Although a fast life history strategy concerns minimal parental investment, women are still expected to commit to a higher level of parental investment as the primary caregiver. Mate quality in terms of genes or resource acquisition are perhaps more important to women and might affect when psychopathic behaviours emerge as compared to men. The occurrence and role of fetal programming and postnatal influences may differ according to sex, although these ideas remain untested.

Postnatal maternal bonding quality may either compliment or limit the impact of the behavioural consequences of changes in hormonal levels caused by maternal stress. If the outside environment improves after birth and allows for longer-term parental investment, then higher levels of maternal care and lower levels of maternal control should signal to the child to augment their behaviour in relation to their future mating strategy. Indeed, a life history strategy must demonstrate developmental plasticity (West-Eberhard, 2003) in shifting to what is most adaptive for that environment. Taking risks, such as those associated with psychopathic behaviour, may not confer advantage when the environment is not suitable to that strategy.

We were interested in investigating the relative contribution of PT and the type of child–mother bonding in the development of primary and secondary psychopathic traits and behaviours in men and women. We expected that higher levels of PT and lower levels of maternal care and high maternal control to be related to primary and secondary psychopathy. We also wanted to investigate whether maternal factors would influence primary and secondary psychopathy over and above the effect of PT. The overall sample, and men and women separately were examined, owing to the inequity in parental investment between men and women, and how this might affect the development of primary and secondary psychopathy.

## 2. Method

### 2.1. Participants

148 participants, of which 67 were men (mean age: 23.48, $SD = 7.00$), and 81 were women (mean age: 21.62, $SD = 6.07$), were recruited from a North-West England university in exchange for course credits, and from the local community via snowball sampling.

### 2.2. Measures

#### 2.2.1. Self-Report Psychopathy Scale (SRP-III)

The SRP-III (Paulhus, Neumann, & Hare, 2009) is 64-item self-report questionnaire that measures psychopathy in non-clinical populations. Participants, using a 5-point Likert scale (1 = strongly disagree, 5 = strongly agree), assess the extent to which they agree or disagree with 64 statements such as "Most people are wimps". Items (n = 32) are summed and averaged to create a score for primary psychopathy (Callous Affect and Interpersonal Manipulation) and secondary psychopathy (Erratic Lifestyle and Criminal Tendencies). Both had good internal reliability (Cronbach's alpha = .81 and .87 respectively).

#### 2.2.2. Prenatal testosterone exposure

The 2D:4D digit ratio is considered as a proxy marker for PT exposure (Lutchmaya, Baron-Cohen, Raggatt, Knickmeyer, & Manning, 2004). The length of the second finger (2D) is divided by the length of the fourth finger (4D). Finger measurements were obtained from handscans using a Canon Canoscan LiDE120 scanner and measured using the ruler tool in Adobe Photoshop CS5. This is considered a superior method to using callipers or rulers (Kemper & Schwerdtfeger, 2009). Measurement was taken from the tip of the finger to the proximal crease of the palm by two independent raters. Digit ratio was calculated for the right (RH2D:4D) and left (LH2D:4D) hand. Intraclass correlation coefficients (ICCs) were calculated via a two-way mixed effects model with absolute agreement (Voracek, Manning, & Dressler, 2007) to ascertain interobserver repeatabilities of the finger

**Table 1**
Means, standard deviations and Cronbach's alpha for variables.

| | Total | α | Men | α | Women | α | t | d |
|---|---|---|---|---|---|---|---|---|
| Primary psychopathy | 2.51 (.57) | .87 | 3.91 (.40) | .68 | 2.19 (.49) | .87 | 9.85** | 1.61 |
| Secondary psychopathy | 2.18 (.47) | .79 | 2.50 (.35) | .58 | 1.92 (.38) | .79 | 9.60** | 1.59 |
| Mother care | 32.61 (10.20) | .92 | 32.61 (10.20) | .8 | 39.28 (9.13) | .92 | −4.15** | −.69 |
| Mother protection | 28.24 (6.81) | .67 | 28.24 (6.81) | .67 | 27.28 (5.42) | .67 | 0.92 | .16 |
| RH 2D:4D | .961 (.048) | | .961 (.048) | | .977 (.038) | | −2.20* | −.37 |
| LH 2D:4D | .955 (.054) | | .955 (.054) | | .983 (.037) | | −3.67** | −.6 |

* $p < .05$.
** $p < .01$.

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