

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.

Keywords: Primary psychopathy, Secondary psychopathy, Prenatal testosterone, 2D:4D digit ratio, Maternal bonding, Life History Theory, Fetal programming

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 hypothesised 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, 2003). 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 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, Redmond, 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; Mack, Hackney, & Pyle, 2011; Krischer & Sevecke, 2008). 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 measurements. Reliability was moderate to high between two observers. ICCs were .848 for R2D, .868 for R4D, .347 for RH2D4D, .892 for L2D, .913 for L4D and .468 for LH2D:4D (all $ps < .001$).

2.2.3. Parental Bonding Instrument (PBI)

Items ($n=25$) were used from the PBI (Parker, Tupling, & Brown, 1979) to measure recollections of parental bonding from which a score for maternal care (12 items) and maternal control (13 items) were gathered. Using a 4-point Likert scale ($1 =$ very like, $4 =$ very unlike), participants rate how statements such as “Spoke to me in a warm and friendly voice” are representative of their mother’s parenting style. Both had moderate to good internal reliability (Cronbach’s $\alpha = .92$ and $.67$ respectively).

3. Results

Men scored significantly higher than women for primary and secondary psychopathy, and significantly lower for RH2D:4D and LH2D:4D (Table 1). Women scored significantly higher for recalled maternal care.

Table 1

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

	Total	α	Men	α	Women
Primary psychopathy	2.51(.57)	.87	3.91(.40)	.68	2.19(.49)
Secondary psychopathy	2.18(.47)	.79	2.50(.35)	.58	1.92(.38)
Mother care	32.61 (10.20)	.92	32.61 (10.20)	.8	39.28 (9.13)
Mother protection	28.24 (6.81)	.67	28.24 (6.81)	.67	27.28 (5.42)
RH 2D:4D	.961 (.048)		.961 (.048)		.977 (.038)
LH 2D:4D	.955 (.054)		.955 (.054)		.983 (.037)

* $p < .05$

** $p < .01$

To explore whether primary and secondary psychopathy are related to 2D:4D and maternal care and control, zero-order (Table 2) and partial correlation coefficients (Table 3) were calculated, controlling for primary and secondary psychopathy respectively, to ensure that relationships were driven by the particular psychopathy variant rather than the shared variance. To compensate for multiple testing, the minimum alpha level was set at .001. Only women had a significant negative relationship between LH2D:4D and primary psychopathy. Comparisons of correlations between men and women revealed significant differences in the relationship between RH2D:4D, and maternal care and control.

Table 2

Zero order correlations between right and left hand 2D:4D, psychopathy variants and maternal bonding.

	Total	Men
Primary psychopathy	-.21**	-.03
Secondary psychopathy	-.21**	-.11
Maternal care	.02	-.17
Maternal protection	-.20*	-.18

Note. z is Fisher's z to compare dependent correlations.

* $p < .05$

** $p < .01$

Table 3

Partial correlations (controlling for other psychopathy variant) between variables for men and women.

	RH2D:4D				Total
	Total	Men	Women	z	
Primary/secondary psychopathy	-.09/-.09	.01/-.10	-.17/-.02	1.08/-.48	-.23**/-.16
Mother care	-.07/-.09	-.22/-.18	.08/.01	-1.68*/-1.14	.16/.10
Mother control	-.18*/-.16	-.17/-.18	-.20/-.17	-2.22*/-.06	-.13/-.09

Note. z is Fisher's z to compare dependent correlations. Primary psychopathy controlling for secondary psychopathy is above the diagonal, secondary psychopathy controlling for primary psychopathy is below the diagonal.

* $p < .05$

** $p < .01$

To determine the predictive power of each variable in primary and secondary psychopathy for men and women, we conducted a series of standard, simultaneous regressions (Table 4). In men, primary psychopathy was predicted by secondary psychopathy and maternal protection; in women, secondary psychopathy, LH2D:4D

and maternal care. In men, secondary psychopathy was predicted by primary psychopathy and maternal care; in women, primary psychopathy and LH2D:4D only.

Table 4

Summary of standard regression analyses for variables predicting primary and secondary psychopathy in men and women

Variable	Primary psychopathy			
	B	SEB	β	B
Secondary/Primary psychopathy	.28/.74	.14/.11	.25*/.58**	.23/.54
RH2D:4D	12.08/39.19	32.72/48.92	.05/.10	-23.45/-68.
LH2D:4D	-24.23/-108.09	29.03/49.35	-.10/-.26*	-14.54/86.2
Maternal care	-.26/-.42	.16/.14	-.21/-.25**	-.31/.09
Maternal protection	.48/.30	.22/.24	.26*/.10	.20/-.17
R^2			.25/.53	
F			4.08**/16.74**	

Note: Men are above the diagonal, women below the diagonal.

* $p < .05$

** $p < .01$

To look at the contribution of maternal bonding above and beyond PT on primary and secondary psychopathy for the overall sample, we ran four hierarchical regressions (Table 5). In the first step 2D:4D (RH and LH alternately) was regressed on to primary and secondary psychopathy (alternately). In the second step, mother care and mother protection were added to the model. In all models, 2D:4D significantly predicted both primary and secondary psychopathy. At the second step, apart from secondary psychopathy in the RH and LH2D:4D models, mother care and mother protection added significantly to all other models. Specifically, lower levels of mother care and higher levels of mother protection significantly predicted levels of primary psychopathy over and above the influence of PT. Lower levels of mother

care also significantly added to secondary psychopathy above and beyond PT in the final model, however, mother control did not. In all of the final models, PT remained a significant predictor.

Table 5

Hierarchical regression of 2D:4D and mother care and protection on primary and secondary psychopathy

	PP/SP RH2D:4D					
	B	SE	β	ΔR^2	B	SE
Step 1						
2D:4D	-2.89/-2.32	1.07/.88	-.22**/-.21**	.04**/.05**	-4.41/-2.89	. .
Step 2						
2D:4D	-2.28/-2.03	.96/.82	-.17*/-.19*		-2.86/-1.90	. .
Mother care	-.03/-.02	0/0	-.45**/-.38**		-.02/-.02	. .
Mother protection	.02/.01	.01/.01	.18*/.09	.24**/.15**	.02/.01	. .

* $p < .05$

** $p < .01$

Note: Scores for primary psychopathy are above the diagonal, scores for secondary psychopathy are below the diagonal. PP in the LH2D:4D model: $R^2 = .05$, $F(1, 146) = 7.25$, $p < .01$; Step 1: $\Delta R^2 = .05$, $F(1, 146) = 7.25$, $p < .01$; Step 2: $\Delta R^2 = .24$, $F(2, 144) = 23.76$, $p < .001$. PP in the LH2D:4D model: $R^2 = .22$, $F(1, 146) = 22.54$, $p < .001$; Step 1: $\Delta R^2 = .22$, $F(1, 146) = 22.54$, $p < .001$; Step 2: $\Delta R^2 = .17$, $F(2, 144) = 17.96$, $p < .001$. SP in the RH2D:4D model: $R^2 = .20$, $F(3, 144) = 11.94$, $p < .001$; Step 1: $\Delta R^2 = .20$, $F(3, 144) = 11.94$, $p < .001$; Step 2: $\Delta R^2 = .15$, $F(2, 144) = 13.77$, $p < .001$. SP in the LH2D:4D model: $R^2 = .20$, $F(3, 144) = 11.94$, $p < .001$; Step 1: $\Delta R^2 = .11$, $F(2, 144) = 10.17$, $p < .001$.

4. Discussion

We investigated whether PT and quality of maternal-child bonding are related to primary and secondary psychopathic traits and behaviours in men and women.

Only in women were higher levels of PT were related to primary and secondary psychopathic traits, although they also reported uncaring mothers. Quality of mother-

child bonding was implicated in the development of primary and secondary psychopathic traits in men, for which PT was not relevant. For the overall sample, PT was, independently, an important contributing factor to primary and secondary psychopathy. However, mother bonding was also influential. Primary psychopathic individuals who had been exposed to more PT recalled mothers as cold or controlling. While secondary psychopathic individuals exposed to more PT also reported uncaring mothers, they had not experienced controlling mothers.

Psychopathy is considered a male fast life history strategy (Jonason et al., 2009), and psychopathic type behaviours are associated with higher levels of PT (e.g., Bailey & Hurd, 2004; Fink et al., 2006), as well as freely circulating testosterone (Yildirim & Derksen, 2012). So it is interesting to find that only women appear subject to fetal programming for psychopathic behaviour. Perhaps fetal programming is more important in women, or that female fetuses are more responsive to fluctuations in in-utero hormone levels. Indeed, the relationship between personality traits and PT are more often evidenced in women rather than men (Fink, Manning, & Neave, 2004) and the developmental outcomes of prenatal maternal stress are more detrimental in females than males (Barrett & Swan, 2015). Evidence suggests that maternal stress increases prenatal testosterone in female fetuses only (Barrett et al., 2014; Sarkar et al., 2008). There is also little to no relationship between the development of primary psychopathic behaviours and adverse postnatal environmental factors in girls (Hicks et al., 2012). Estrogen may serve as a postnatal protective factor against the development of neurobiological imperfections (Wise, Dubal, Wilson, Rau, & Böttner, 2001) that are associated with primary psychopathy in men. It should be noted that as male fetuses are often exposed to higher levels of PT, the absence of a significant finding in men may be due to a “ceiling effect” where

the lengths of the fingers cannot go beyond a masculinisation threshold (Hampson, Ellis & Tenk, 2008). Nevertheless, relationships between PT and types of offending behaviour in men are evidenced (Hoskin & Ellis, 2015).

It is also interesting that the influence of suboptimal levels of maternal bonding in primary and secondary psychopathic traits differed in men and women. Primary and secondary psychopathy are suggested to have different etiologies, namely, primary as genetic and secondary as environmental (Karpman, 1941; Mealey, 1995, although see Hicks et al., 2012). Low maternal care might serve as a proximate trigger for the development of psychopathic behaviours in both men and women (Gao et al., 2010). However, women high in primary psychopathic traits may inherit those traits from a mother who have a similar cold and un-empathetic personality style to them (Loney, Huntenburg, Counts-Allan, & Schmeelk, 2007). Men could develop psychopathic traits as a postnatal response to their mother's behaviour. Research also shows that the sex of the fetus alters gene expression caused by maternal stress (Grundwald & Brunton, 2015). There could be a yet undiscovered genetic relationship between PT and the manifestation of primary psychopathic behaviours in women, since the 2D:4D ratio is highly heritable (Voracek & Dressler, 2009).

The finding that primary psychopathic individuals had experienced cold and controlling mothering is to be expected. If fluctuations in PT are caused by maternal stress, then unless the status of the rearing environment had improved between pregnancy and post-birth, there should be a continuation of factors that encourage a "tough-minded" personality that is adaptive for a hostile environment. Actually, psychopathic behaviour in children lessens if their parents receive parent training and emotional support, and worsens in the absence of such interventions (McDonald, Dodson, Rosenfield, & Jouriles, 2011). If levels of PT were attributed more to

genetic influences, then it is possible that the same genes could also contribute to a mother who is less empathetic and more controlling of her children. Furthermore, controlling mothers producing primary psychopathic children may be attributed to passive gene x environment correlation. Indeed, it is interesting that secondary psychopathic individuals also reported uncaring mothers, but had not been subject to controlling behaviour. It is possible then that these mothers do not exhibit primary psychopathic behaviour but are less caring due to environmental circumstances. Evidently, PT is an important factor that should be considered in developmental models of psychopathy, yet maternal caring appeared more important and may indeed be a mechanism by which PT leads to psychopathic behaviours. However, examining genetic and environmental causation remains complicated and speculative until we know more about the precise mechanisms involved.

There are limitations to our study. Using 2D:4D as a biomarker in the context of studying individual differences has been challenged (Berenbaum, Korman Bryk, Nowak, Quigly, & Moffat, 2009). However, its popularity as a measure in personality research indicates that it is sufficient for an exploratory study such as this one. Retrospective and self-report measures engender potential problems of accurate recall and self-serving bias. It is important to note that differences in the size of the digit ratio can vary more between countries than between sexes (Manning, Churchill, & Peters, 2007), thus in the future, it is essential to use participants from different countries and ethnic backgrounds.

To our knowledge, this is only the second study that has highlighted a relationship between primary and secondary psychopathy and the in-utero hormonal environment, but is unique in having also examined the role of maternal bonding. We revealed prenatal and postnatal influences for primary psychopathic behaviours in

women, while in men, secondary psychopathic behaviours derive from postnatal experiences. Our findings add to the current literature, by highlighting how fledging psychopathy may be nurtured before birth, and that this biological preparedness is more important for women.

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