RESEARCH ARTICLE

Relationships Between Self-Reported and Observed Parenting Behaviour, Adolescent Disordered Eating Attitudes and Behaviours, and the *5-HTTLPR* Polymorphism: Data From the Australian Temperament Project

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Abstract

This study examined whether self-reported and observationally measured parental behaviours were associated with disordered eating, and investigated possible moderation by a serotonin-transporter polymorphism (*5-HTTLPR*). Study 1 included 650 adolescents from the Australian Temperament Project who completed the Eating Disorder Inventory-2 Drive for Thinness and Bulimia scales at 15/16 years and were genotyped for *5-HTTLPR*. Parents completed an Australian Temperament Project-devised measure of parental warmth and harsh punishment. Study 2 included a subgroup of 304 participants who also engaged in a video-recorded family interaction, with observed parental warmth and hostility coded by the Iowa Family Interaction Rating Scale. Greater self-reported parental warmth was associated with lower bulimia scores. Conversely, observationally measured parental warmth was associated with lower drive for thinness, but not bulimia. Self-reported parental harsh punishment was associated with bulimia only, with observed parental hostility associated with neither outcome. *5-HTTLPR* genotype did not moderate the relationship between parent behaviours and adolescent disordered eating.

Received 21 February 2017; Revised 18 May 2017; Accepted 19 May 2017

Keywords

disordered eating; gene environment interactions; *5-HTTLPR*; parenting behaviours; observational measurement

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Published online 10 July 2017 in Wiley Online Library (wileyonlinelibrary.com) DOI: 10.1002/erv.2530

The biopsychosocial approach to eating disorder (ED) aetiology proposes that risk factors for EDs range from those relating to the individual, such as genes and psychological traits, to those that form part of the environment, including relationships with parents and peers, significant life events, exposure to the 'thin ideal', and numerous other factors (Culbert, Racine, & Klump, 2015; Stice, 2002; Trace, Baker, Penas-Lledo, & Bulik, 2013). Risk of developing an ED is believed to be associated with an interplay between these factors. For example, individuals exposed to 'risky' environments may develop ED symptoms only if they also carry certain 'risky' psychological or genetic factors (Stice, Marti, & Durant, 2011). In particular, following a lack of findings of direct genetic effects (Root *et al.*, 2011), a growing area of ED research involves examining how individual genetic differences may moderate the impact of certain environmental variables on ED symptoms (Rozenblat *et al.*, 2017).

Most research to date has approached investigation of gene × environment (GxE) interactions through the diathesis-stress lens, investigating how genetic 'vulnerability' may increase susceptibility to stressful environments (e.g. Stoltenberg, Anderson, Nag, & Anagnopoulos, 2012). However, GxE investigations across other fields increasingly support an alternative model, under which certain genetic factors conceptualised as conferring 'risk' may be better conceptualised as 'plasticity' factors that are associated with better outcomes under positive or neutral environmental conditions (Belsky *et al.*, 2009). The current study will examine possible genetic plasticity in disordered eating attitudes and behaviours by examining adolescent exposure to both 'positive' and 'negative' parenting behaviours, using a multimethod multisource approach.

The concept of genetic plasticity has received some early support in the depression field (Belsky & Pluess, 2009; Uher &

McGuffin, 2008). Belsky and Pluess (2009) identified a number of studies in which participants with the short (s) allele of the serotonin transporter *5-HTTLPR* polymorphism, typically considered a 'risk' allele, exhibited lower depression under conditions of few or no stressful life events, compared with those homogenous for the long (l) allele (Brummett *et al.*, 2008; Eley *et al.*, 2004). However, recent meta-analyses cast some doubt over these earlier findings of plasticity (e.g. Culverhouse *et al.*, 2017).

5-HTTLPR has been the most heavily studied polymorphism in investigations of GxE interactions, with one or two copies of the s-allele resulting in reduced serotonin transcription (Heils *et al.*, 1996). The s-allele has been implicated in changes in appetite, mood, and the stress-response system (Gotlib, Joormann, Minor, & Hallmayer, 2008; Leibowitz & Alexander, 1998; Ruhé, Mason, & Schene, 2007) and thus is of direct relevance to ED aetiology. As such, most GxE investigations in eating pathology have focussed on *5-HTTLPR*, with a recent meta-analysis finding significant interactions between *5-HTTLPR* and traumatic life events, as well as sexual and physical abuse, in predicting EDs and bulimia nervosa (BN), respectively (Rozenblat *et al.*, 2017). However, no study in the ED field has examined GxE 'risk' from a plasticity perspective.

Parenting factors, such as parental behaviour and child–parent relationship quality, have been implicated in EDs (Pamies, Botella, & Treasure, 2013; Tetzlaff, Schmidt, Brauhardt, & Hilbert, 2016). As parenting practices range from more positive to more negative, they provide an excellent opportunity for investigating the plasticity hypothesis of GxE interactions in relation to eating pathology. This may involve analysing two dimensionally opposite parenting behaviours, such as warmth and hostility (Schaefer, 1959), which have some theoretical and empirical support for a role in disordered eating. For example, compared with controls, questionnaire-based studies investigating families of ED patients have found that families tend to be characterised by lower warmth and fewer positive family bonds (Calam, Waller, Slade, & Newton, 1990; Cunha, Relvas, & Soares, 2009; Vidovic, Juresa, Begovac, Mahnik, & Tocilj, 2004) but greater family conflict and parental control (Calam *et al.*, 1990; Canetti, Kanyas, Lerer, Latzer, & Bachar, 2008). Furthermore, parenting styles (Baumrind, 1971) conceptualised as low on warmth and high on control and hostility (i.e. authoritarian parenting) have been associated with greater disordered eating compared with parenting styles not low on warmth (Lobera, Ríos, & Casals, 2011; Zubatsky, Berge, & Neumark-Sztainer, 2015). Parental warmth is therefore a possible protective factor for disordered eating, while hostility or harsh punishment may constitute risk factors, although limited research specifically investigates the latter.

Given the importance of accurate measurement of environmental stimuli in GxE research (Moffitt, Caspi, & Rutter, 2005), a second approach may involve investigating parenting behaviours by using observational techniques. Unlike idiographic self-report measurement (Margolin *et al.*, 1998), observational measures assess families by using the same metric and may overcome issues such as limitations in self-awareness (Aspland & Gardner, 2003). Other than studies specifically examining parental expressed emotion (Vaughn & Leff, 1976), very few studies have thus far investigated the relationship between (non-mealtime) parental behaviours and eating pathology by using observational techniques, and most have not analysed parental warmth or hostility (these include Blair, Freeman, & Cull, 1995; Humphrey, 1989; Kog & Vandereycken, 1989; Lattimore, Wagner, & Gowers, 2000; Ratti, Humphrey, & Lyons, 1996; Stasch & Reich, 2000; Thomas, Hoste, & Le Grange, 2012). Across these studies, findings regarding parental warmth and hostility have been mixed (e.g. Humphrey, 1989, cf. Lattimore *et al.*, 2000). This inconsistency is unsurprising given that most existing observational studies are limited by use of poor, unestablished, or inappropriate instruments to code the observational data (e.g. Kog & Vandereycken, 1989; Lattimore *et al.*, 2000), have used small to medium sample sizes [largest $N = 74$ in Humphrey (1989)], and have analysed female-only samples. Furthermore, no studies have examined the relationship between observed parent behaviours and sub-clinical eating pathology. There remains a need to establish solid conclusions regarding how parental warmth and hostility are related to disordered eating by using reliable and consistent observational approaches, alongside self-report measures and use of large sample sizes. One further possibility is that genetics may, to an extent, account for some discrepancies in the literature.

To our knowledge, two studies (Karwautz *et al.*, 2011; van Strien, Snoek, van der Zwaluw, & Engels, 2010) have analysed parenting within a GxE framework in the ED field. One study ($N = 265$) found an interaction between problematic parenting styles, in particular parental control, and the s-allele of *5-HTTLPR* in predicting anorexia nervosa (AN) diagnosis in a female-only discordant AN sister-pair sample (Karwautz *et al.*, 2011). The second study ($N = 279$) found an interaction between parental psychological control and the A1 allele of the Taq1A polymorphism on *DRD2* in predicting greater emotional eating in a mixed-gender sample (van Strien *et al.*, 2010). However, neither study explicitly tested for genetic plasticity. Furthermore, both studies focussed largely on parental control and did not shed light on the role of other parenting styles, including positive parent behaviours.

The present investigation aimed to assess the relationship between self-reported and observed parental behaviours and adolescent disordered eating and to investigate whether *5-HTTLPR* moderated this relationship, using the largest sample to date. This is an important improvement on previous ED research, given the critical nature of sample size in genetic research (Duncan & Keller, 2011). The first study included self-reported measures of parenting behaviour to examine the direct effects of parental warmth and use of harsh punishment on disordered eating attitudes and behaviours, as well as possible moderation by *5-HTTLPR*. The second study aimed to replicate these analyses with the use of observationally measured parenting behaviours, to provide insight into how observed parenting behaviours are related to adolescent disordered eating by using a sample size multiple times larger than previously assessed, as well as aiming to provide further support for any GxEs identified in Study 1. These studies represent an advancement both in research relating to the role of parenting factors in EDs, which has included predominantly self-report measures, as well as GxE interactions in the ED field, which has focussed on presence or absence of negative

outcomes only. This is the first investigation of observed parenting within a GxE framework in the ED field, and inclusion of a mixed-gender community sample allows for results to inform ED prevention initiatives.

Study 1

Method

Participants

Australian Temperament Project participants were initially recruited in infancy (4–8 months) in Victoria in 1983 via maternal and child health centres in urban and rural locations. The first survey included 2443 infants (48.0% female), with 16 surveys completed to date. The present study involved a subset of 650 participants (50.2% female), who had completed the 11th survey at age 15–16 years and self-identified as Caucasian. The participants provided information on disordered eating attitudes and behaviours, as well as a saliva sample for genotyping. One parent of each participant completed a parent-reported measure of parenting behaviours. To increase sample size for the present study, in 2015, 196 participants who had completed the survey but had not provided saliva for DNA extraction were invited to provide a saliva sample. Of 196 participants, 107 participants were successfully contacted and 83 individuals agreed to provide a sample. The parents and adolescents provided written informed consent for each survey wave and for the collection of saliva samples. The data collection was approved by the Australian Institute of Family Studies Ethics Review and carried out in accordance with the latest version of the Declaration of Helsinki. The final sample included a greater proportion of participants from the highest socioeconomic status (SES) quartile compared with the original sample in 1983 (see Table 1 for participant characteristics).

Table 1 Sociodemographic details of participants included in Study 1 and Study 2

	Full sample			Women		Men	
	N	*N*	%	*N*	%	*N*	%
Study 1							
Sex	650						
Male		312	49.8				
Female		338	50.2				
SES quartile	632						
Highest		231	36.6	116	36.6	115	36.5
Medium–high		197	31.2	103	32.5	94	29.8
Medium–low		129	20.4	59	18.6	70	22.2
Lowest		75	11.9	39	12.3	36	11.4
Parent marital status	645						
Married/defacto		538	83.4	264	81.5	274	85.4
Separated/divorced		73	11.3	42	13.0	31	9.7
Single/widowed		22	3.4	10	3.1	12	3.7
Remarried		12	1.8	8	2.5	4	1.2
Study 2							
Sex	304						
Male		143	47.0				
Female		161	53.9				
SES quartile	296						
Highest		103	34.8	53	33.3	50	36.5
Medium–high		100	33.8	55	34.6	45	32.8
Medium–low		61	20.6	31	19.5	30	21.9
Lowest		32	10.8	20	12.6	12	8.8
Parent marital status	299						
Married/defacto		242	81.0	126	80.3	116	81.7
Separated/divorced		37	12.4	17	10.8	20	14.1
Single/widowed		12	4.0	8	5.1	4	2.8
Remarried		8	2.7	2	3.8	2	1.4

Measures

Disordered eating attitudes and behaviours. The Eating Disorder Inventory-2 (EDI-2; Garner, 1991) Drive for Thinness and Bulimia subscales assessed adolescent disordered eating. The Drive for Thinness subscale consists of seven items measuring the participants' desire to lose weight or fear of weight gain (e.g. 'I am preoccupied with the desire to be thinner') with Cronbach's $\alpha = .92$ in the current sample. The Bulimia subscale consists of eight items measuring bulimic behaviours, including binging and purging (e.g. 'I stuff myself with food'), with a somewhat lower internal consistency of $\alpha = .74$ in the present sample. Responses ranged from *Never* to *Always* and were scored on the original 1–6 scale, as recommended for nonclinical samples (Schoemaker, van Strien, & van der Staak, 1994), and then averaged with higher mean scores reflecting greater disordered eating attitudes and behaviours. Wording of the EDI-2 was modified somewhat for an Australian audience, as detailed in Krug *et al.* (2016).

Parent behaviours. The ATP-devised Parenting Practices Scale (Prior, Sanson, Smart, & Oberklaid, 2000) was used to assess parental behaviours, with parents responding to five questions investigating parental warmth (e.g. 'In general, how easy is it to spend time with your teenager?'), with $\alpha = .77$, and 10 questions regarding parental punishment (e.g. 'I use threats of punishment to control him/her'), also $\alpha = .77$. Responses were recorded on a 5-point scale from *Always/Almost Always* to *Never* and recoded so higher scores reflected greater warmth or greater punishment.

5-HTTLPR genotype. DNA was isolated by using Qiagen QIAamp kits from buccal epithelial cells via cotton swabs and genotyping performed as described previously in Jorm *et al.* (2000). The additional 83 saliva samples obtained for Study 2 were collected via Oragene saliva tubes, with DNA extracted and genotyped at the Australian Genomics Research Facility, Adelaide, Australia. In both cases, *5-HTTLPR* genotype was coded into s-present (s/s or s/l genotype) or s-absent (l/l genotype) groups.

Sociodemographics. Adolescent age, height in centimetres, and weight in kilograms were self-reported, allowing for calculation of participant body mass index (BMI; kg/m^2). SES was calculated on the basis of parent-reported maternal and paternal education and employment status.

Data analyses

Using IBM SPSS version 21, four multiple linear regression models separately assessed the direct effects of parental warmth and parental harsh discipline on participant EDI-2 Drive for Thinness and Bulimia scores, controlling for BMI and gender. Additional models tested for moderation by the *5-HTTLPR* polymorphism by including the GxE interaction terms. These models also controlled for the potential confounding effects of gender and BMI as per Keller (2014), by including all the covariate x gene and covariate x parenting style interaction terms in the regression models. Prior to analyses, missing data (23.5%) for the BMI variable were imputed by using multiple imputation, with no systematic patterns of missingness observed.

Power analysis

Power analysis was conducted by using Quanto (http://biostats.usc.edu/Quanto.html), with the following specifications: continuous outcome, independent individual design, and a dominant model with s-allele frequency of 43% (based on allele frequencies in the succeeding texts). To detect a large-sized to medium-sized GxE interaction ($R^2 = 0.02$) at an alpha level of .05, a sample of 389 was required, compared with the present sample of 650. However, this sample was not powered to detect small effects (i.e. $R^2 < 0.01$).

Results

Descriptive statistics for participants in Study 1 are featured in Table 2. Participant *5-HTTLPR* genotype distribution (l/l = 197, s/l = 341, and s/s = 112) met the Hardy–Weinberg equilibrium, $\chi^2 = 2.96$, $df = 1$, $p > .05$. Tables featuring the results of all regression models are in the supporting information.

Across all regression models examining the main effects, there was a direct effect of female gender and BMI on Drive for Thinness scores, with the former also predicting Bulimia scores (all $p < .001$). There was a significant relationship between Bulimia, but not Drive for Thinness, and self-reported parental warmth ($\beta = .11$, $p = .005$) and harsh punishment ($\beta = .11$, $p = .003$). There was also an interaction between gender and harsh punishment in predicting drive for thinness ($\beta = .30$, $p = .038$), with scores for men lower under conditions of higher harsh punishment, while they remained largely unchanged for women. There were no main effects of *5-HTTLPR* or any significant interaction effects between *5-HTTLPR* and parent behaviours.

Table 2 Descriptive statistics for key variables investigated in Study 1

		Mean (*SD*)	
Variable	Full sample	Women	Men
Age (years)	15.72 (0.16)	15.74 (0.14)	15.72 (0.17)
Parental warmth	4.22 (0.63)	4.25 (0.65)	4.19 (0.61)
Parental punishment	2.00 (0.56)	1.94 (0.55)	2.06 (0.57)
EDI-2 bulimia	1.78 (0.65)	1.94 (0.73)	1.62 (0.53)
EDI-2 drive for thinness	2.23 (1.15)	2.81 (1.22)	1.64 (0.70)
BMI	21.27 (3.28)	21.20 (3.10)	21.34 (3.44)

Study 2

Method

Participants

Study 2 consisted of a subsample of 304 participants (53.0% female) from Study 1 who were selected to participate in an observational family interaction task based on responses to the 11th survey, as part of an earlier investigation into factors contributing to risk and resilience for adolescent adjustment. Approximately three-quarters of those invited consented to participate and were characterised into a problem (16.7%), high-risk (21.7%), or low-risk (55.2%) group. The participants in the problem group either exhibited elevated levels of depressed mood, as indicated by endorsing five or more Diagnostic and Statistical Manual of Mental Disorders, Third Edition (APA, 1980) symptoms of depression, reported frequent substance use, or exhibited antisocial behaviour, determined by endorsing four or more delinquent acts (e.g. stealing, fighting, and driving a car without permission). Based on a number of factors that differentiated the problem group from the main ATP sample (such as earlier behaviour problems, school adjustment difficulties, and peer relationships), a group of problem-free yet 'high-risk' participants was identified. A gender-balanced low-risk group was drawn randomly from the remaining sample. The participants completed the EDI-2 and provided saliva samples as described in Study 1. The final sample for Study 2 included a lower proportion of participants from the lowest SES quartile compared with the original sample. Demographic information is featured in Table 1.

Measures

Disordered eating attitudes and behaviours, BMI, and age were measured as in Study 1.

Observational measure of parent behaviours. Trained interviewers performed home visits during which the participants and one of their parents (usually the mother) engaged in a video-recorded 15-min discussion based on a set of cards containing 14 questions about family life (e.g. teenager's accomplishments and disappointments and parental rules and fairness). Parenting behaviours were coded by a team who had undergone extensive training and were blind to adolescent group membership, using the Iowa Family Interaction Rating Scale (Melby *et al.*, 1998), a macro-level observational coding system that was designed to measure behaviours and emotions in family discussions with adolescents. This is considered a valid tool for measuring behaviours in a variety of dyadic interactions and has been validated against self and other reports from family members (Melby & Conger, 2001). Two scales were selected for the present study, parental warmth and parental hostility.

Warmth measured parental expressions of care, concern, and support directed at their child, including expressions of approval, affectionate physical contact, and building upon or reciprocating warmth displayed by their child. It was rated on a 9-point scale from *no warmth* to *frequent warmth*. Average intraclass correlation to assess inter-rater reliability for this scale was 0.80, based on cross examination of XX% of the videos.

Hostility measured the extent to which parents engaged in hostile behaviours directed to their child, including rejection, active ignoring, showing contempt, or expressing complaints or critical remarks. It was rated on a 9-point scale from *no hostility* to *frequent hostility*. The average intraclass correlation for this scale was 0.75, which compares favourably with previous studies using the Iowa Family Interaction Rating Scale and is deemed acceptable (Ge, Best, Conger, & Simons, 1996).

Data analyses

Data were analysed by using IBM SPSS version 21, as in Study 1, via four linear regression models examining the direct effects of observed parental warmth and observed parental hostility on EDI-2 Drive for Thinness and Bulimia scales. Additional models examined possible moderation by *5-HTTLPR*, again controlling for gender and BMI. In the analyses investigating parental warmth and *5-HTTLPR*, covariate x gene and covariate x environment contrast terms were included, as recommended by Keller (2014). However, this was not possible in the investigation of parental hostility and *5-HTTLPR* due to issues of excessive collinearity (further discussed in the Results section). Finally, to ensure that any differences between Studies 1 and 2 reflected measurement effects as opposed to sample effects, the direct effects of self-reported parental warmth and self-reported use of harsh punishment were also tested in the Study 2 subsample. GxE interactions were not included in this analysis to avoid multiple testing issues. Missing data for BMI (28.29%) were imputed via multiple imputation, with no systematic patterns of missingness observed.

Power analysis

Power analysis was conducted by using Quanto (http://biostats.usc.edu/Quanto.html), as for Study 1, with s-allele frequency of 43% (based on allele frequencies for Study 2 participants). The current sample was powered to detect a large-sized to medium-sized GxE interaction, $R^2 = 0.03$, which requires 258 participants at an alpha level of .05 but was not powered to detect effects smaller than this.

Results

Descriptive statistics for the participants in Study 2 are featured in Table 3. *5-HTTLPR* genotype distribution (l/l = 90, s/l = 154, and s/s = 60) met the Hardy–Weinberg equilibrium, $\chi^2 = 0.16$, $df = 1$, $p > .05$. The results from all regression models are located in the supporting information.

Table 3 Descriptive statistics for key variables investigated in Study 2

		Mean (*SD*)	
Variable	Mean (*SD*)	Women	Men
Age (years)	15.73 (0.15)	15.73 (0.13)	15.72 (0.10)
Parental warmth	4.38 (1.93)	4.57 (1.92)	4.16 (1.92)
Parental hostility	1.92 (1.53)	2.06 (1.65)	1.76 (1.37)
EDI-2 bulimia	1.89 (0.73)	2.08 (0.80)	1.68 (0.58)
EDI-2 drive for thinness	2.34 (1.25)	2.99 (1.28)	1.61 (0.67)
BMI	21.55 (3.59)	21.65 (3.40)	21.45 (3.77)

Female gender predicted greater Drive for Thinness and Bulimia scores (all $p < .001$), while BMI was associated with the former only. There were also significant direct effects of greater observed parental warmth on reduced Drive for Thinness ($\beta = -.10$, $p = .029$), but not Bulimia, and no direct effects of observed parental hostility. No significant main effects of *5-HTTLPR* or interaction between *5-HTTLPR* and observed parenting behaviours were observed.

The models investigating moderation by *5-HTTLPR* in the relationship between observed parental hostility and disordered eating do not include gene × covariate and environment × covariate contrast terms due to issues with collinearity. Although Keller (2014) argues collinearity functions to control for alternate explanations of any GxE interaction, the GxE interaction showed minimal change regardless of whether the additional contrast terms were included in the models. Therefore, the simpler model less affected by collinearity is presented.

The follow-up analysis investigating self-reported parental warmth and use of harsh punishment in the present subsample revealed main effects of the warmth and punishment on bulimia scores ($\beta = -.14$, $p = .013$ and $\beta = .14$, $p = .011$ respectively) but not drive for thinness scores. These results mirrored those of the larger sample in Study 1.

Discussion

We adopted a multimethod assessment approach in two studies to investigate the relationship between parental behaviours (warmth, harsh punishment, and hostility) and disordered eating attitudes and behaviours. We also investigated whether the relationship between parenting factors and eating pathology was moderated by the serotonin transporter *5-HTTLPR* polymorphism. The results indicated that greater parent-reported warmth was associated with lower bulimia symptoms, and in concordance, parent-reported use of harsh punishment techniques was associated with greater bulimia symptoms. Conversely, greater observed parental warmth was associated with lower adolescent drive for thinness, while greater observed parental hostility did not predict either disordered eating outcome. No moderation by *5-HTTLPR* was identified in any of these relationships.

Parental warmth and harsh punishment

Findings of a relationship between parental warmth and use of harsh punishment techniques with disordered eating attitudes and behaviours support the results of past studies that have assessed these parental behaviours via questionnaires. For example, parental warmth and care have been identified as lower in families of patients with AN and BN compared with controls (Tetley, Moghaddam, Dawson, & Rennoldson, 2014), while parental harsh punishment has been associated with BN in a number of studies (Rorty, Yager, & Rossotto, 1995; Stuart, Laraia, Ballenger, & Lydiard, 1990), with these patterns of findings also reflected in studies of child development more broadly (e.g. Bender *et al.*, 2007; Yap, Pilkington, Ryan, & Jorm, 2014).

However, few studies have investigated these associations by using observed parenting behaviours, as we have here, and a novel pattern of results was identified compared with research based on questionnaires. Across both samples, the two self-reported measures of parenting behaviours were clearly associated with

adolescent bulimia symptoms, but neither was related to drive for thinness. Conversely, the observed parenting behaviours tended to be associated with drive for thinness but not bulimia. Other studies have also found limited correspondence between parent responses on questionnaires and their actual observed behaviours (e.g. Kallstrom-Fuqua, 2004). Such discrepancies are not unexpected for a number of reasons, including socially desirable or idiosyncratic responding to a questionnaire or issues of ecological validity during observational tasks. It is also possible that the two measures of parental warmth used in the present study tapped into slightly different constructs. The observational measurement may have had a stronger focus on caring and warm behaviour displayed by the parent, irrespective of the child's behaviour, while the questionnaire measure perhaps reflected more greatly the level of general positive interaction between parent and child, albeit from the parent's perspective. This may suggest that the precise relationship between parental behaviours and adolescent disordered eating is highly nuanced and varies according to the specific element of parenting or disordered eating measured.

Gene x environment interactions

The lack of significant GxE interactions identified in the present study contrasts with Karwautz *et al.* (2011) ($N = 256$), who found that problematic parenting styles interacted with the s-allele of *5-HTTLPR* to predict AN diagnosis in a discordant sister-pair sample. One possibility is that the influence of *5-HTTLPR* may only be evident when clinical-level disordered eating pathology, such as AN, is considered. Another consideration is that Karwautz *et al.* (2011) examined parental control, a different parental behaviour to those examined in the present study. The other study identifying a possible interaction between parenting and genes ($p = .06$) in the ED field also investigated parental control (van Strien *et al.*, 2010) ($N = 276$). One possibility, therefore, is that parental control but not other parenting behaviours may interact with genetic factors to predict eating pathology. However, it remains possible that discrepant findings between the current study and previous investigations are due to low power in both Karwautz *et al.* (2011) and van Strien *et al.* (2010), which increases the chances of false positive findings (Duncan, Pollastri, & Smoller, 2014). Sample sizes required for genetic association studies are very large due to the small effect sizes purportedly involved, and the present sample size ($N = 650$) was more than double that of the previous two studies identifying GxE interactions involving parenting in EDs. Similarly, numerous past studies that have identified plasticity related to the *5-HTTLPR* s-allele in other fields, and indeed have been cited as early evidence of this effect, have been vastly underpowered. A large portion of these studies included fewer than 150 participants (e.g. Kim, 2010; Pluess, Belsky, Way, & Taylor, 2010; Taylor *et al.*, 2006; Wilhelm *et al.*, 2006).

The lack of moderation by *5-HTTLPR* in the relationship between parental warmth and bulimic behaviours and drive for thinness supports the view that this polymorphism does not act as a plasticity allele in the presence of family environments typically considered 'protective'. This is also reflected in the fact that, to the authors' knowledge, no prior study investigating GxEs in the ED field has reported differential susceptibility in response to positive environments (distinct from absence of negative stressors). In other fields, parental warmth and *5-HTTLPR* have not been heavily studied within a GxE framework, with the few existing studies presenting a mix of results (e.g. Hankin *et al.*, 2011; Kochanska, Kim, Barry, & Philibert, 2011). Given the large-scale publication bias that purportedly affects studies of GxE interactions (Duncan & Keller, 2011), it cannot be ascertained how many other unpublished investigations have failed to find GxE effects involving parental warmth.

Strengths and limitations

The present paper contributes to research examining parenting factors in disordered eating by analysing a large-scale community sample, with a focus on replication across two samples using multimethod multisource assessment. In contrast, past studies have largely adopted questionnaire-based measures, often retrospective, with limited investigation using observational measurement of parenting behaviours. A few limitations of the present study must also be noted. The present study did not examine comorbid psychopathology, so was unable to assess whether this factor may possibly mediate the identified relationships between particular parenting behaviours and disordered eating outcomes. The present study also examined the biallelic model of *5-HTTLPR*. There is some evidence to suggest that examination of a triallelic model may better reflect *5-HTTLPR* activity (Wendland, Martin, Kruse, Lesch, & Murphy, 2006), with support from some studies in the ED field (Steiger *et al.*, 2009; Stoltenberg *et al.*, 2012 but not Richardson *et al.*, 2008), although more broadly findings have been tentative (e.g. Uher & McGuffin, 2008). In the present study, data were not available to examine the triallelic model, which may yet have resulted in identification of a GxE interaction in the present sample. Furthermore, the sample size, particularly in Study 2, was relatively underpowered for genetic association studies (although it was above the mean $N = 288$ of GxE studies in the ED field; Rozenblat *et al.*, 2017). Conversely, the sample in Study 1 constitutes the third largest investigation of its kind in the field (following Akkermann *et al.*, 2012, $N = 765$, and the combined-samples analyses in Rozenblat *et al.*, 2017, $N > 1000$). This is an important strength. Furthermore, assessing disordered eating attitudes and behaviours dimensionally increases power and sensitivity compared with case–control designs, in which the 'clinical' group may represent heterogeneous underlying patterns of eating pathology or genetic influences (Abbott, 2008). Assessing disordered eating symptoms in a population-based sample also increases generalisability of results and utility for informing prevention and early intervention initiatives, although it may limit the applicability of results to clinical populations. Finally, as with all studies of candidate genes, *5-HTTLPR* represents only one polymorphism in a system of genetic factors likely to be involved in ED pathology, and thus, such investigations constitute stepping stones towards increasing our understanding of the genetic underpinnings of EDs.

Clinical implications and future directions

The results support a clear relationship between parent behaviours and adolescent disordered eating, across both genders, independent of individual genetic predisposition. The findings can inform parenting programmes targeting ED prevention or psychoeducation for 'at risk' families. For example, one systematic review of parent-focussed ED prevention initiatives stated that

early positive findings required support from additional research regarding the family setting (Hart, Cornell, Damiano, & Paxton, 2015). Adoption of longitudinal designs would also help establish whether parental behaviours constitute protective/risk factors or are better conceptualised as correlates of eating pathology. Furthermore, investigations into genetic plasticity could also expand beyond *5-HTTLPR* to examine cumulative plasticity across numerous polymorphisms testing multiple positive environmental factors. Selection of positive eating-related outcomes, such as positive body image (Webb, Wood-Barcalow, & Tylka, 2015), as opposed to absence of eating-related pathology, may also be more appropriate for reflecting the relationship between genetic plasticity and environmental factors. However, it is also possible that regardless of precise measurement techniques, the role of *5-HTTLPR* in EDs may prove to be very minor at best, such that continued investigation into parenting and other environmental factors may constitute a more fruitful approach to untangling the complex aetiology of eating pathology.

Acknowledgements

The ATP study is located at The Royal Children's Hospital Melbourne and is a collaboration between Deakin University, The University of Melbourne, the Australian Institute of Family Studies, The University of New South Wales, The University of Otago (NZ), and the Royal Children's Hospital; further information is available at www.aifs.gov.au/atp. The views expressed in this paper are those of the authors and may not reflect those of their organisational affiliations nor of other collaborating individuals or organisations. We acknowledge all collaborators who have contributed to the Australian Temperament Project, especially Professors Ann Sanson, Margot Prior, Frank Oberklaid, and John Toumbourou and Ms Diana Smart. We would also like to sincerely thank the participating families for their time and invaluable contribution to the study. This paper forms part of Vanja Rozenblat's PhD with publication undertaken at The University of Melbourne.

This work was supported by an Early Career Researcher Grant (1350035), an Australian Research Council Senior Research Fellowship (DP 130101459), and the Australian Postgraduate Award.

Conflicts of interest

The authors declare no conflicts of interest.

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