Intrauterine testosterone exposure and risk for disordered eating

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Summary

Previous research has suggested that prenatal testosterone exposure masculinises disordered eating by comparing opposite- and same-gender twins. The objective of the current study is to replicate this finding using a sample of 439 identical and 213 fraternal females, 461 identical and 344 fraternal males, and 361 males and 371 females from opposite-gender twin pairs. Disordered eating was compared across twin types using the Eating Disorder Inventory–2. Inconsistent with previous findings, a main effect of co-twin gender was not found. Our results raise questions about the validity of prior evidence of the impact of prenatal testosterone exposure on patterns of disordered eating.

Recently, Culbert *et al*[1] suggested that prenatal exposure to gonadal hormones may contribute to the substantial gender difference in the prevalence of eating disorders. The Michigan State University Twin Registry (MSUTR) included 582 twins (113 opposite-gender twins) among whom levels of disordered eating in same- and opposite-gender twin pairs were compared to examine for a 'free martin effect' (i.e. *in utero* exposure to testosterone masculinises behaviour in females).[2]

They assessed the free martin effect indirectly by examining opposite-gender twin pairs where the female twin shares a prenatal environment with her male co-twin and, therefore, should be exposed to testosterone *in utero*. Consistent with this effect, Culbert *et al* found that levels of disordered eating have a significant linear trend, with same-gender female twins exhibiting the highest levels followed by females from opposite-gender pairs, males from opposite-gender pairs, and finally same-gender male twin pairs exhibiting the lowest

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levels. In this report we attempt to replicate and extend these findings using the same statistical method as Culbert *et al*.

Method

The present sample, the Swedish Twin study of CHild and Adolescent Development (TCHAD), began with all twin pairs born in Sweden between May 1985 and December 1986.[3] Twins were recruited through the medical birth registry and identified twins/parents of twins were posted study questionnaires.[4] Eighty-two per cent of participants responded to the questionnaires when disordered eating was examined.[3]

The sample includes 461 and 439 monozygotic, 344 and 312 dizygotic individual females and males respectively from same-gender pairs, and 361 males and 371 females from opposite-gender pairs who are 15–17 years old. Zygosity was determined based on computer algorithms of questionnaire responses.[3] Questions were validated by a discriminant analysis of 106 same-gender pairs where zygosity had been determined by typing 16 polymorphic DNA markers.[3]

The majority of the participants' parents were born in Sweden (85.8%). The highest level of education obtained by either parent was a university degree (47%). Overall, 15% of parents reported employment as 'unskilled labourer', 27.6% as 'skilled labourer', 28.6% reported a medium-level white-collar career, and 29% reported a professional/high-level white-collar career. Rates for professional careers are similar to those reported by Culbert *et al* (33%).[1] However, our sample consists of fewer medium-level careers (28% *v*. 43%) and more unskilled labourers (15% *v*. 3%).

Disordered eating was examined using three subscales from the Eating Disorder Inventory–2[5] (EDI–2): drive for thinness (i.e. excessive concern with dieting, weight preoccupation and extreme pursuit of thinness); bulimia (i.e. tendency towards episodes of binge eating followed with an impulse to purge); and body dissatisfaction (i.e. belief that specific body parts are too large). A total score was created using all available questions. Questionnaires were approved by the ethics committee of the Karolinska Institutet, Stockholm, Sweden.

Hierarchical linear models were used to examine mean differences in disordered eating across same- and opposite-gender twin pairs; such models can account for the non-independence of twin data. Two predictors were used: twin's gender (male *v*. female) and co-twin's gender (male *v*. female). These two variables were utilised and coded following Culbert *et al*: males=1 and females=−1. $P < 0.05$ was considered significant.

If more than 10% of the questions for any specific subscale were missing, the score was coded as missing. Subscales were log transformed prior to analyses to correct for a positive skew.

Results

Hierarchical linear model results indicate a significant main effect for twin gender on the three subscales, with females reporting significantly higher levels for drive of thinness

(t(2050)=−14.25, $P<0.001$), body dissatisfaction (t(2185)=<16.37, $P<0.001$) and total score (t(1994)=−15.80, $P<0.001$). However, a main effect was not found for bulimia (t(2059)= −1.00, P=0.43). This indicates no gender difference for aspects related to the bulimia subscale in our sample. Contrary to Culbert *et al*'s findings, no significant main effects were found for co-twin gender with any subscale. Results remained consistent utilising only dizygotic twin pairs. Because no difference was exhibited between opposite- and same-gender twins, this suggests that intrauterine testosterone exposure does not have an impact on the risk for disordered eating. Mean scores for EDI–2 subscales are shown in Fig. 1 as a function of gender and zygosity.

Discussion

The aim of this report was to replicate the findings of Culbert *et al*.[1] Using a different self-report measure and similar analytical methods, our results did not corroborate. There are four possible reasons for this. First, the current study could have been under-powered. However, a power analysis conducted with same-gender females and opposite-gender females as comparison groups, with an effect size provided by Culbert (K. Culbert, personal communication, 1 May 2008), revealed a power of 0.99.[6] Thus, our negative results could not plausibly arise from low power.

Second, the two samples differed. Our participants were adolescent Swedish twins while Culbert *et al*'s was an ethnically diverse sample of young adults (mean 20 years) living in the midwestern USA. Our sample may also be more representative of its respective general population. The TCHAD sample was obtained by contacting twins through a medical birth registry, whereas most MSUTR twins are recruited through advertisement and live within a 2 h radius of MSUTR headquarters.[1] However, both samples are volunteer-based. The two populations could also have a differential prevalence of eating disorders. However, studies indicate that the prevalence of eating disorders in Sweden and other Scandinavian countries is similar, if slightly less prevalent than in the USA.[7,8] Similarly, prenatal hormone exposure is a biological effect that occurs *in utero* and one might expect its effects to remain constant across age levels and populations.

Third, different measures of disordered eating were used. Culbert *et al* used a total score derived from the Minnesota Eating Behaviors Survey (MEBS).[9] There is one main difference between this survey and our EDI–2 subscales. The MEBS divides binge eating and compensatory behaviours into two separate subscales allowing for more information to be obtained about each variable, whereas the EDI–2 combines these into the bulimia subscale. For example, the MEBS includes questions about several different types of purging behaviours and the EDI–2 only enquires about self-induced vomiting.

Finally, the EDI–2 may not be an adequate measure of disordered eating for males[10] or for a Swedish population. For example, the bulimia subscale may represent more normative aspects of behaviours in males.[11] Many of the questions on this subscale deal with binge-eating behaviours and 15- to 17-year-old boys may commonly consume large amounts of food.[11] Sources of a drive for thinness and body dissatisfaction are also likely to vary between genders and the EDI–2 focuses on core areas of the female body with which

women are more typically dissatisfied (e.g. stomach and thighs). The EDI–2 was also normalised and created with a clinical sample of females with eating disorders from the USA, so its constructs may not extrapolate to a Swedish population. However, studies indicate that the EDI–2 may be an acceptable measure of disordered eating in both a male[12,13] and a Swedish population.[12,14] For example, in a study utilising the identical adolescent sample used in our study, Cronbach's alpha coefficients were estimated at 0.81, 0.70 and 0.88 for the drive for thinness, bulimia and body dissatisfaction subscales respectively for males, indicating high internal reliability.[11]

Taken together, the results of our study are inconsistent with previous research. Because of the similarities between our report and the previous report,[1] the evidence for the hypothesis that prenatal hormone exposure has an impact on the development of eating disorders and disordered eating is lacking.

Acknowledgments

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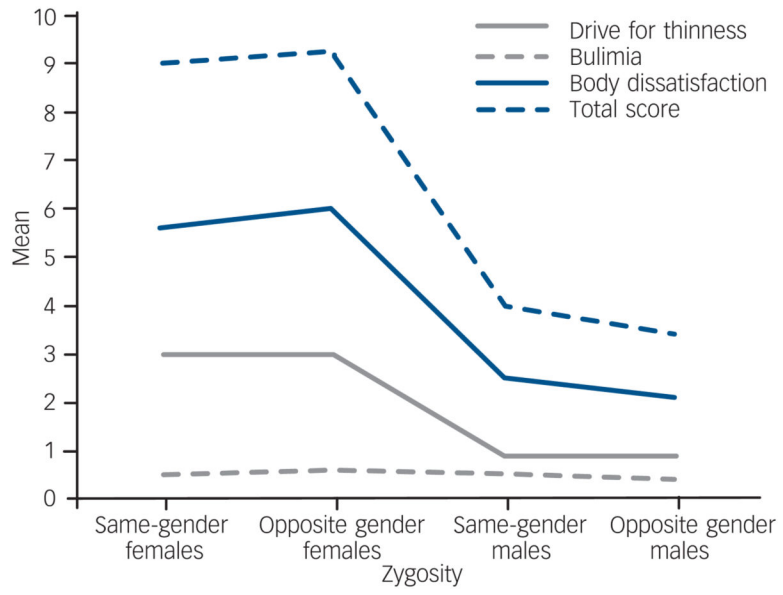
Fig. 1.
Mean scores of Eating Disorder Inventory–2 subscales by gender and zygosity.