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BRIEF COMMUNICATION

A preliminary population-based twin study of self-reported eating disorder

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ABSTRACT

Background. Twin studies have concluded that there is a substantial genetic contribution to the aetiology of eating disorders. The aim of the present study was to estimate the genetic contribution to the aetiology of self-reported eating disorders in a sample of representative twins.

Method. A population cohort of 34 142 young Danish twins was screened for eating disorders by a mailed questionnaire.

Results. Concordance rates differed significantly across monozygotic and dizygotic twin pairs for broadly defined self-reported anorexia nervosa and bulimia nervosa. Heritability estimates of 0·48, 0·52 and 0·61 respectively were estimated for narrow and broad definitions of self-reported anorexia nervosa and for self-reported bulimia nervosa.

Conclusions. There is a genetic contribution to the aetiology of self-reported eating disorders in the general population. The relationship between self-reported and clinical eating disorder remains to be examined.

INTRODUCTION

Mixed clinic and self-referral based twin studies of anorexia nervosa (AN) have found significantly higher concordance rates in monozygotic (MZ) than in dizygotic (DZ) twin pairs with a heritability estimate as high as 0·98 (±0·12) (Holland *et al*. 1984, 1988). A population-based study of AN (Wade *et al*. 2000) reporting on interview data about 2163 female twins estimated heritability of AN at 0·58 (95% CI 0·33–0·84).

Regarding bulimia nervosa (BN) a significant difference in MZ/DZ concordance rates has been reported in one mixed clinic and self-referral study on 21 female twin pairs (Fichter & Noegel, 1990). One population-based twin study (Kendler *et al*. 1991) estimated heritability of BN at 55%.

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The epidemiology of eating disorders (ED) has been studied in two twin populations. In a face-to-face interview study of 2163 female twins, lifetime prevalence of AN (Walters & Kendler, 1995) and BN (Kendler *et al*. 1991) was estimated at 1·6% and 2·8% respectively, according to narrow diagnostic criteria (3·7% and 5·7% respectively according to broad diagnostic criteria). In an Australian telephone interview study of 3869 female twins (Wade *et al*. 1996), lifetime prevalence of AN and BN was estimated at 0·4% and 1·8% respectively.

It has been demonstrated that questionnaires are likely to over-estimate the prevalence of disturbed eating behaviour, especially the more complex traits such as binge eating (Fairburn & Beglin, 1994; Black & Wilson, 1996). Even though the strength of the association between self-reported and clinically diagnosed ED is not sufficiently known, we believe that self-reporting of ED does reflect some degree of perceived

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eating behaviour disturbance. The aim of the present study was to examine epidemiological and genetic findings from a self-report questionnaire study, performed as the first step of a two-step screening procedure for ED in a large, unselected twin population.

METHOD

The sample

The Danish Twin Register (Kyvik *et al*. 1996) is located at University of Southern Denmark, Odense University, Faculty of Medical Sciences. The present study is based upon the young cohort (birth years 1953–1982) of the Danish Twin Register, ascertained in 1991 (Kyvik *et al*. 1995), and consisting of 20888 twin pairs (38506 individuals). Zygosity was established by means of a self-report similarity questionnaire with an accuracy of $> 90\%$ of all cases (Hauge, 1981). Of the original cohort 34142 individuals (88·7%) were available for further studies.

Procedure

The questionnaire

In 1994 all retrievable twins who had earlier agreed to participate in further studies ($N =$ 34076) were sent a questionnaire of 60 items. The ED items were embedded in the questionnaire, surrounded by items relating to various disorders and symptoms. Two items pertained to the life time occurrence of AN: (*i*) AN according to personal judgement; and (*ii*) AN in the opinion of relatives or friends. A third item pertained to the lifetime occurrence of (*iii*) symptoms of BN. For specific wording of the questions see Appendix. The items are referred to as (*i*) AN-Self, (*ii*) AN-Others and (*iii*) BN in the following. The twins were aged 11 to 41 years when they received the questionnaire. One reminder was mailed to all non-responders.

The ED group

Probands were defined as all twins who endorsed one or more of the ED questions. The probands will be referred to as the ED group. Two subgroups were defined according to the AN items: AN-Narrow contained all twins who had endorsed the AN-Self item irrespective of their answers to the other ED items. AN-Broad contained all twins who had endorsed the AN-Self and/or the AN-Others item, irrespective of their answer to the BN item. AN-Narrow is thus a subset of AN-Broad. A third subgroup, BN-Only, was defined as the twins who had only endorsed the BN item. Only pairs in which both members had responded to the questionnaire, and in which at least one member was a proband, were included in the concordance estimates.

Genetic analyses

Pairwise concordance rates were calculated as the number of concordant pairs divided by the total number of affected pairs. The probandwise concordance rates are calculated by entering the number of concordant pairs doubly when independently ascertained and singly when the ascertainment of the second twin follows the ascertainment of the first twin.

Correlations and heritability in liability to develop ED were estimated by standard methodology (Neale & Cardon, 1992) under the assumption of the multifactorial threshold model (Falconer, 1965). The assumption of the model is that the observed categorical phenotype reflects an underlying normally distributed liability to disease, consisting of several small contributions from environmental and genetic factors. The manifestation of disease occurs when an individual exceeds the threshold of affection on the liability distribution. The variance in liability can be decomposed in four variance components owed to additive genetic effects (A), non-additive genetic effects (D), environmental effects shared by individuals (C), and environmental effects not shared by individuals (E). The variance components and confidence intervals were estimated by means of structural equation modelling with the software MX (Neale, 1997). ACE, ADE, AE, CE and E models were fitted to the data and the best-fitting model chosen based on a chi-square Goodness-of-Fit test and the Akaike Information Criterion. Heritability, i.e. the proportion of the total phenotypic variance due to genetic variance, was derived from the best-fitting model.

Ethical considerations

The study was approved by all regional Scientific-Ethical Committees of Denmark as part of a multi-centre study, and by the Danish Data Protection Agency.

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RESULTS

Epidemiological findings

After one reminder 29424 questionnaires (86·2%) were returned. Non-responders comprised more males and members of opposite-sexed pairs. The ED group comprised 1270 individuals (7·2% of all female responders, 1·2% of all male responders) AN-Narrow comprised 2·7%, AN-Broad 4·9% and BN-Only 2·9% of adult ($\geqslant$ 20 years) women (0·5%, 0·9% and 0·3% respectively of adult men). Male and opposite-sexed pairs, and twins $<$ 20 years of age were omitted from further analyses.

Genetic findings

There were 190 complete female MZ pairs and 248 complete female DZ pairs available for further analyses. The pair-wise and proband-wise concordance rates are shown in Table 1. Significant differences in pairwise concordance rates were found for AN-Broad and BN-Only. Confidence intervals for probandwise concordance rates overlapped slightly between MZ and DZ pairs only for AN-Narrow.

The results of the structural equation modelling are shown in Table 2. An AE model provided the best fit of the data for all the ED subgroups, while ACE, ADE, CE and E models provided a poorer fit (data not shown).

DISCUSSION

The sample and generalizability of findings

Our ongoing clinical interviewing of the ED group will help clarify some of the outstanding issues, including to what degree self-reported ED corresponds to an operationalized clinical diagnosis of the disorder. Nevertheless, our twin sample is representative of the general population of Danish twins, and the attrition rate was within acceptable limits, meaning that the results should be of interest when discussing the contribution of genetic factors of the pathogenesis of ED.

We found overall rates of lifetime AN and BN consistent with those of studies reporting on broadly defined syndromes (Kendler *et al.* 1991; Wade *et al.* 1996). Our screening device is new, but there are clear similarities to the method

Table 1. *Concordance rates for the subgroups of the ED group* (*female*, *birth cohorts* 1953–74)

	Concordance rates (95% CI)			
	Pairwise		Probandwise	
ED-subgroup	MZ	DZ	MZ	DZ
AN-Narrow	0·10 (0·03–0·16)	0·04* (0·00–0·08)	0·18 (0·10–0·27)	0·07 (0·02–0·13)
AN-Broad	0·15 (0·08–0·21)	0·07† (0·03–0·10)	0·25 (0·18–0·33)	0·13 (0·08–0·17)
BN-Only	0·15 (0·06–0.23)	0·06‡ (0·01–0·10)	0·26 (0·16–0·35)	0·11 (0·04–0·17)

MZ–DZ difference test: *$\chi^2 = 2{\cdot}39$, df = 1, $P < 0{\cdot}2$; †$\chi^2 = 4{\cdot}75$, df = 1, $P < 0{\cdot}05$; ‡$\chi^2 = 3{\cdot}90$, df = 1, $P < 0{\cdot}05$.

Table 2. *Results of the structural equation modelling*, *best-fitting model*

ED-subgroup	Model	χ^2	df	*P*	AIC	a^2 (95% CI)	c^2	e^2
AN-Narrow	AE	0·475	4	0·976	−7·525	0·48 (0·274–0·647)	0	0·524
AN-Broad	AE	0·017	4	1	−7·983	0·52 (0·381–0·645)	0	0·355
BN-Only	AE	0·049	4	1	−7·951	0·61 (0·441–0·752)	0	0·244

a, Additive genetic effect (a^2 = heritability); c, common environmental effect; e, individual genetic effect; AIC, Akaike Information Criterion.

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used in the Australian twin study (Wade *et al.* 1996), in which a lifetime prevalence of 1·3% was found for self-reported AN among women aged under 45. This is lower than our rate of 2·7% in a group of women aged 19 to 41 years. However, in the Australian study a high rate of substantial underweight was reported (4·3%). Possibly some of the underweight women may have at some time in their life suffered from an unrecognized or hidden AN. The bulimia screening question covered bulimic behaviour, but there was no information relating to frequency. The life time prevalence of bulimic behaviour in our study was 4·2% (2·9% for bulimic behaviour only). This rate is higher than that reported by the Australian group (2·9% for self-induced vomiting, 0·9% for bulimia), but comparable to the findings of other studies (Kendler *et al.* 1991).

Genetic analyses

Our findings reinforce the notion of a genetic liability to ED. Our MZ concordance rates were substantially below those reported from clinical studies of both AN (Holland *et al.* 1984, 1988) and BN (Fichter & Noegel, 1990), but in keeping with those reported in the only previous population based study on BN (Kendler *et al.* 1991). The MZ/DZ differences in concordance rates were substantially lower than in former studies, but still significant differences were found for AN-Broad and BN-Only. Our heritability estimates for AN (0·48–0·52) were much lower than previously reported in clinical studies (Holland *et al.* 1988), but in keeping with the findings of the Virginia twin study (Wade *et al.* 2000). Our heritability estimate for BN (0·61) was within the range of former studies (Kendler *et al.* 1991). The differences in concordance rates and heritability between former clinic based studies and our study may reflect the risks of ascertainment biases connected to clinic-based studies which could lead to overestimation of genetic aetiology (Bulik *et al.* 2000).

In summary, the present study details results of self-reported ED using a shorter screening devise than previously employed. The relationship between these self-report data and clinical ED is currently being investigated. The epidemiological results of the study taken with the nature of the data suggest, that the findings represent disordered eating rather than clinical ED. The heritability estimates of both AN and BN are in keeping with those of other population-based twin studies, and thus adds to the growing evidence that genetic factors are influential in the development of disordered eating.

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APPENDIX 1

ED items – translation of exact wording

AN-Self: Have you ever had anorexia nervosa? (Y/N)

AN-Others: Has anybody among your friends or family ever suggested that you had anorexia nervosa? (Y/N)

BN: Have you ever had an irresistible craving for food, that made you binge, after which you deliberately threw up so as not to gain weight? (Y/N)

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