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Research report

Genetic and environmental contributions to depressive personality disorder in a population-based sample of Norwegian Twins

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Abstract

Background: Depressive personality disorder (DPD) was introduced in DSM-IV as a new category requiring further study. The aim of this study was to estimate genetic and environmental contributions to DPD in a population-based twin sample, and include data on criteria performance, prevalence and diagnostic overlap.
Methods: Axis I and Axis II diagnoses were obtained by structured interviews in a population-based sample of 2794 young adult twins. Statistical analyses included correlation and factor analysis based on polychoric correlation coefficients, and diagnostic overlap applying adjusted odds ratios. Contributions from additive genetic and common and unique environmental influences to the liability to DPD were computed using structural equation modelling, applying a multiple threshold variable.
Results: Liability to DPD could best be explained by additive genetic and unique environmental factors, with heritability estimates of 49% (95% CI 0.41–0.57) in females and 25% (95% CI 0.12–0.40) in males. The best-fitting model indicated that some of the genes contributing to DPD differ between men and women. Chronbach's alpha was 0.87. 2.0% of participants fulfilled the criteria for DPD, and overlap was most pronounced for dysthymic disorder and avoidant personality disorder.
Limitations: Low prevalence rates and subsequent inclusion of subthreshold criteria could have influenced parameter estimates, especially in males.
Conclusions: DPD was almost twice as heritable in females as in males, comparable to previous studies on major depression. The proposed criteria showed good measurement properties, and DPD was not completely subsumed within any other disorder.

Keywords: Depressive personality disorder; Personality; Heritability; Twin study

1. Introduction

The concept of depressive personality was first introduced in psychiatric literature at the beginning of the previous century. Various descriptions have been proposed, but most emphasize traits of gloominess, worry,

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pessimism and self-criticism (Phillips et al., 1995). Depressive personality disorder (DPD) was, however, first introduced in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) (American Psychiatric Association, 1994) as a new category requiring further study. The proposed diagnosis requires the fulfilment of five of seven criteria.

Despite previous suggestions that DPD is a common disorder (Klein, 1999), there is little data to support this claim. Only one previous study has reported the prevalence of DSM-IV DPD in a small, non-clinical sample: Ryder et al. (2001) found that 17 of 368 (4.7%) young adults fulfilled the criteria for DPD. To date, we are unaware of any larger, population-based studies on DPD prevalence, and such data are in demand (Ryder et al., 2005).

The personality disorder (PD) work group for DSM-IV addressed several issues regarding DPD (Phillips et al., 1995), including the reliability of diagnostic tools, criteria performance, and the overlap between DPD and other PDs and mood disorders. Several studies have addressed the latter question; with some concluding that the overlap is too great to justify a diagnostic entity for DPD (Bagby et al., 2003; Ryder et al., 2002), and others the opposite (Klein and Shih, 1998; Markowitz et al., 2005; McDermut et al., 2003; Phillips et al., 1998). The conflicting results demonstrate that this problem is far from solved and justify further studies.

An important diagnostic validation criterion is that of familial aggregation (Robins and Guze, 1970). However, the number of twin studies on PDs and dimensionally defined PD traits is limited: Results from a Norwegian study based on clinical samples showed heritabilities for DSM-III-R PDs in the range of 0.28–0.78, but did not include DPD (Torgersen et al., 2000). In a study on dimensionally defined PD traits, Jang et al. (1996) found heritabilities ranging from 0.38 to 0.48 for facet scales relevant to DPD such as anhedonia, pessimism and guilt proneness. Numerous studies have shown that DSM PDs can be predicted by the five-factor model for personality (NEO-PIR) (Costa and McCrae, 1992), for review see (Saulsman and Page, 2004; Widiger and Costa, 2002). DPD correlates highly (0.50–0.75) with both the higher-order trait of Neuroticism and the lower order trait of Depression (Dyce and O'Connor, 1998), which again has been shown to be heritable (Jang et al., 1998).

We are unaware of any prior family, twin or adoption study of DPD. The main aim of this study was to explore the relative contribution of genetic and environmental factors to the liability of DSM-IV DPD in a large, population-based sample of Norwegian twins, including possible sex differences. We also estimated the prevalence of DPD and its co-occurrence with Axis I mood disorders and other Axis II disorders to address questions about the need for an independent diagnosis of DPD.

2. Methods

2.1. Sample

Subjects included in this study were recruited from The Norwegian Institute of Public Health Twin Panel (NIPHTP). NIPHTP consists of twins identified through information in the national Medical Birth Registry, established January 1, 1967, which receives mandatory notification of all live and stillbirths of at least 16 weeks gestation. The current panel includes information on 15,370 like and unlike sexed twins born from 1967–1979. During that time period the percentage of pairs for which both twins survived to age 3 ranged from 82% to 89%. Two questionnaire studies have been conducted; in 1992 (twins born 1967–1974) and in 1998 (twins born 1967–1979). Altogether, 12,700 twins received the second questionnaire, and 8045 responded after one reminder (response rate 63%). The sample included 3334 pairs and 1377 single responders. The NIPHTP is described in detail elsewhere (Harris et al., 2002).

Data for the present report derive from an interview study of Axis I and Axis II Psychiatric Disorders. Participants were recruited among 3153 complete pairs who in the second questionnaire agreed to participate in the interview study, and 68 pairs who were drawn directly from NIPHTP. Of these 3221 eligible pairs, 0.8% was unwilling or unable to participate, and in 16.2% of pairs only one twin agreed to the interview. 38.2% did not respond after two contacts requesting participation (the maximum number of contacts allowed in the licence obtained from The Regional Committee for Medical Research Ethics). Altogether 2794 twins (44% of those eligible) were interviewed for the assessment of PDs. Our final sample consisted of 1022 males and 1772 females; 221 monozygotic male (MZM) pairs, 116 dizygotic male (DZM) pairs, 448 monozygotic female (MZF) pairs, 261 dizygotic female (DZF) pairs, 340 dizygotic opposite sex (DZO) pairs and 22 single responders. The mean age of attendants was 28.2 years (range 19–36).

Approval was received from The Norwegian Data Inspectorate and The Regional Committee for Medical Research Ethics, and written informed consent was obtained from all participants after complete description of the study.

Zygosity was initially determined by questionnaire items previously shown to categorize correctly 97.5% of