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Research report

Major depression and life satisfaction: A population-based twin study

Ragnhild B. Nes [a,*], Nikolai O. Czajkowski [a,b], Espen Røysamb [a,b], Ragnhild E. Ørstavik [a], Kristian Tambs [a], Ted Reichborn-Kjennerud [a,c]

[a] Division of Mental Health, The Norwegian Institute of Public Health, Oslo, Norway
[b] Department of Psychology, University of Oslo, Norway
[c] Department of Psychiatry, University of Oslo, Norway

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ABSTRACT

Background: The extent to which positive and negative indicators of mental health share etiological influences has been studied to a limited degree only. This study examines the genetic and environmental influences on association between liability to lifetime DSM-IV Major Depressive Disorder (MDD) and dispositional life satisfaction (LS).
Methods: Two-wave questionnaire data on LS (assessed 6 years apart) and lifetime MDD obtained by structured clinical interviews in a population-based sample of adult twins were analysed using structural equation modelling in Mx.
Results: The prevalence of lifetime MDD was estimated to be 11.1% and 15.8% in males and females, respectively. Individuals fulfilling the criteria for MDD reported significantly lower levels of LS. The covariation in MDD and dispositional LS was found to be accounted for by genetic and unique environmental influences only. The phenotypic correlation was estimated to be 0.36, of which genetic influences accounted for 74% and environmental factors the remaining 26%. The correlation between genetic factors for MDD and LS was estimated to be −0.55 and the correlation between unique environmental factors to be −0.22. Heritability was estimated to 0.34 and 0.72 for MDD and LS, respectively.
Limitations: The sample consists of twins only and there are limitations associated with the twin design.
Conclusions: Whereas genetic influences on vulnerability to lifetime MDD are considerably shared with liability to (low) LS, environmental influences are more distinct. Thus, environmental factors associated with risk of MDD do not strongly impact on dispositional LS, and conversely, environmental factors influencing dispositional LS do not strongly buffer against MDD.

1. Introduction

Internalizing disorders have consistently been shown to be influenced by genetic as well as environmental factors. A number of biometric studies also suggest that co-occurrence between different internalizing disorders, such as anxiety and depression, is likely to be due to common genetic factors that predispose individuals to clusters of internalizing psychiatric disorders (Kendler et al., 2003; Krueger and Markon, 2006; Krueger, 1999) with distinct environmental stressors leading to the various forms of psychopathology to manifest (Kendler and Karkowski-Shuman, 1997).

Different aspects of positive mental health have also been shown to share a genetic core (Bartels and Boomsma, 2009; Caprara et al., 2009; Keyes et al., 2010). Partly overlapping sets of genes has been shown to influence emotional, social, and psychological well-being (Keyes et al., 2010) as well as self-esteem, life satisfaction (LS), and optimism (Caprara et al., 2009). Using an extended twin-sibling design, Bartels and Boomsma (2009) also recently showed that four common well-being indicators (quality of life in general, quality of life at present, LS, subjective happiness) load on similar sets of genes, suggesting that these different well-being measures are not different at the genetic level. The collective findings have lead some researchers to propose a general and genetically influenced, "positive orientation" (Caprara et al., 2009, p. 278), or "co-vitality factor" (Weiss et al., 2002, p. 1147), similar to the "comorbidity factors" accounting for association between different internalizing disorders.

A related question concerns the commonality and specificity underlying association between well-being and internalizing psychopathology. Outside of the genetic arena, numerous studies

* Corresponding author. Tel.: +47 21078249; fax: +47 21078260.
E-mail addresses:
Ragnhild.bang.nes@fhi.no (R.B. Nes), Nikolai.czajkowski@fhi.no (N.O. Czajkowski), Espen.roysamb@fhi.no (E. Røysamb), Ragnhild.orstavik@fhi.no (R.E. Ørstavik), Kristian.tambs@fhi.no (K. Tambs), Ted.reichborn-kjennerud@fhi.no (T. Reichborn-Kjennerud).

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have dealt with the association between distinct positive and negative emotions and the issue of whether positive and negative emotions are relatively independent (the dual continua, bivariate view) or operate inversely (the one-dimensional, bipolar view). Most studies report moderate correlations and support a partly independent model (Bradburn, 1969; Knutson et al., 1998) indicating that research on indicators of positive mental health is adding a distinctive dimension to psychiatric research.

To date, few have used formal diagnostic data to examine the relationship between common mental disorders and positive emotional states. One study using diagnostic data showed that 85% of subjects with double/chronic depression, 63% with a current major depressive disorder (MDD) and 56% of subjects with dysthymia had satisfaction scores in the severely impaired range, consistent with a monotonic gradient between the severity of depression and satisfaction impairments (Rapaport et al., 2005). A number of studies have also shown dissatisfaction to be strongly associated with poor health and health behaviour, poor social functioning, personality problems, current and future depression (Koivumaa-Honkanen et al., 2004a) and to predict mortality, suicide, and work disability due to psychiatric or somatic causes (Koivumaa-Honkanen et al., 2003; Koivumaa-Honkanen et al., 2004b). In addition, subjective life satisfaction has been shown to constitute an important component of functional outcome in both unipolar and bipolar mood disorders and to be impaired in patients with affective disorders even after recovery (Goldberg and Harrow, 2005). Few have explored genetic and environmental influences on the associations between mood disorders and well-being indicators, however, despite their important implications. Further insight into the etiological underpinnings of well-being and psychopathology may allow us to develop more targeted models of psychotherapy and care and articulate better strategies for health promotion and resource allocation.

To our knowledge, only three previous studies have examined genetic and environmental influences on associations between well-being and internalizing problems in adults. Vinberg and colleagues examined quality of life in first-degree relatives of probands with affective disorder, finding quality of life to be impaired in twins with an affected co-twin, and suggesting a common familial vulnerability with affective disorders (Vinberg et al., 2007). Nes and colleagues (Nes et al., 2008) explored etiological factors explaining associations between LS and self-report symptoms of anxiety and depression, using a single-item LS measure and a short-form of the Hopkins Symptom Checklist (Tambs and Moum, 1993). More recently, Kendler et al. (2011) explored the generality and specificity of etiological factors for co-occurrence between internalizing psychopathology (IP) and (low) well-being (Kendler et al., 2011). The authors used a latent common IP factor based on three different diagnoses (MDD, generalized anxiety disorder, panic attacks) measured by the Composite International Diagnostic Interview Short Form, and a latent well-being construct comprising emotional, psychological, and social well-being (MWB). The three studies yield qualitatively similar findings. The two larger studies (Kendler et al., 2011; Nes et al., 2008) show a substantial, but far from complete overlap in genetic factors for current internalizing symptoms and well-being (40–60%), but less sharing of environmental influences. For example, the environmental overlap was estimated to be 5% using cross-sectional data and 29% when exploring the stable components of IP and MBW using longitudinal data (Kendler et al., 2011). This indicates that the environmental influences are largely specific. However, environmental influences with long-lasting impact on liability to IP have more impact on MBW than environmental influences with mainly transient effects on IP.

The findings from the biometric studies clearly extend our knowledge on the associations between well-being and psychopathology. There are, however, some limitations associated with the approaches used. First, the study by Nes and colleagues relied on self-report measures of psychological distress and not formal DSM-IV psychiatric disorders. The only study that have used formal diagnostic data (Kendler et al., 2011) therefore need replication. Second, Kendler et al. (2011) used an aggregate MWB measure containing both negative and positive affect items. Measures of IP and MBW are therefore likely to at least partially overlap, consequently generating problems of tautology and measurement redundancy. When indicators reflect broad and general phenomena (e.g., global well-being, common internalizing syndromes) this problem may be particularly present. Insights into the etiological mechanisms for association between more narrowly defined, clinically relevant indicators may also be more important to clinicians than knowledge on broader aggregate measures (i.e., comorbidity and co-vitality factors). Thirdly, when well-being and psychopathology are measured concurrently, state-dependent perception and memory are likely to constitute a common source of variance (i.e., well-being levels mainly mirror current levels of depression) (Atkinson et al., 1997).

The present study explores the genetic and environmental substrate for covariation between liability to lifetime DSM-IV MDD and a latent disposition to LS in a population-based sample of adult Norwegian twins. Both well-being and psychopathology are measured by specific, commonly used indicators rather than composite measures, and well-being is measured by a cognitive evaluation rather than emotional content. To reduce problems associated with mood-mediated state-dependent recall both LS and MDD are operationalized as stable liabilities, and most importantly—are not measured concurrently. In addition to reporting etiological influences on the covariation, we also present heritability estimates for MDD and stable LS. The latter has, to our knowledge, not previously been reported. The sample is fairly age homogenous (range 12 years), thus reducing potential problems due to different etiological risk and protective factors across age. We have also examined sex-specific effects, as a number of previous studies have reported sex-specific etiological influences on well-being and internalizing problems (Kendler et al., 2006; Nes et al., 2006; Nes et al., 2010).

2. Method

2.1. Sample

Subjects were recruited from the Norwegian Institute of Public Health Twin Panel which includes the entire cohort of twins ($N=15{,}370$) born in Norway between 1967 and 1979 (Harris et al., 2002; Harris et al., 2006; Tambs et al., 2009a). The twins were originally identified through the Medical Birth Registry of Norway (MBRN), which receives mandatory notification of all live births from 16 week gestation. Two questionnaire studies (Q_1 and Q_2) were conducted in 1992 (twins born 1967–1975) and in 1998 (twins born 1967–1979) and an interview study assessing Axis I and II disorders was conducted between 1999–2004. The present study is based on data from all three assessments (Q_1, Q_2, interview study).

Altogether, 5864 and 8045 twins participated in the Q_1 and Q_2 surveys, giving an individual response rate of 74% and 63%, respectively, and about 80% of the twins participating at Q_1 also participated at Q_2. Eligible participants for the interview study were defined as the 3153 complete twin pairs who participated in the Q_2 study and agreed to participate in the interview. Of eligible twins, a total of 2794 (44%) twins were interviewed. Non-participants included persons unwilling or unable to participate (0.8%), twin pairs in which only one twin agreed to participate (16.2%), and persons who did not respond after one reminder (38.2%).

Approval was received from The Norwegian Data Inspectorate and the Regional Ethical committee, and written informed consent