Psychological Medicine, 2004, **34**, 1177–1186. © 2004 Cambridge University Press
DOI: 10.1017/S0033291703001892 Printed in the United Kingdom

Dimensions of depression, mania and psychosis in the general population

L. KRABBENDAM*, I. MYIN-GERMEYS, R. DE GRAAF, W. VOLLEBERGH, W. A. NOLEN, J. IEDEMA AND J. VAN OS

Department of Psychiatry and Neuropsychology, South Limburg Mental Health Research and Teaching Network, EURON, Maastricht University, Maastricht, The Netherlands; Division of Psychological Medicine, Institute of Psychiatry, De Crespigny Park, Denmark Hill, London, UK; Netherlands Institute of Mental Health and Addiction, Trimbos Institute, Utrecht, The Netherlands; Rudolf Magnus Institute, Department of Psychiatry, University Medical Centre Utrecht and Altrecht Institute for Mental Health Care, Utrecht, The Netherlands; Social and Cultural Planning Office, The Hague, The Netherlands

ABSTRACT

Background. In order to investigate whether correlated but separable symptom dimensions that have been identified in clinical samples also have a distribution in the general population, the underlying structure of symptoms of depression, mania and psychosis was studied in a general population sample of 7072 individuals.

Method. Data were obtained from the three measurements of the Netherlands Mental Health Survey and Incidence Study (NEMESIS). Symptoms of depression, mania and the positive symptoms of psychosis were assessed using the Composite International Diagnostic Interview. Confirmatory factor-analysis was used to test statistically the fit of hypothesized models of one, two, three or seven dimensions.

Results. The seven-dimensional model comprising core depression, sleep problems, suicidal thoughts, mania, paranoid delusions, first-rank delusions and hallucinations fitted the data best, whereas the unidimensional model obtained the poorest fit. This pattern of results could be replicated at both follow-up measurements. The results were similar for the subsamples with and without a lifetime DSM-III-R diagnosis. The seven dimensions were moderately to strongly correlated, with correlations ranging from 0·18 to 0·73 (mean 0·45).

Conclusions. In the general population, seven correlated but separable dimensions of experiences exist that resemble dimensions of psychopathology seen in clinical samples with severe mental illness. The substantial correlations between these dimensions in clinical and non-clinical samples may suggest that there is aetiological overlap between the different dimensions regardless of level of severity and diagnosable disorder.

INTRODUCTION

The positive symptoms of psychosis and the mood symptoms of mania and depression frequently co-occur in the same patient (Brockington *et al*. 1979, 1980; Taylor, 1992; Siris, 2000). In clinical samples of patients with psychosis, they constitute separable but co-morbid symptom dimensions (Kitamura *et al*. 1995; McGorry *et al*. 1998; Van Os *et al*. 1999; Cuesta & Peralta, 2001; McIntosh *et al*. 2001) that are likely to share some genetic and non-genetic aetiological influences (Bailer *et al*. 2002; Cardno *et al*. 2002; Murray *et al*. 2003). However, aetiological continuity may not be the only source of overlap between the positive and the mood symptom dimension. Co-occurrence of two symptom dimensions in patient samples

* Address for correspondence: Dr L. Krabbendam, Department of Psychiatry and Neuropsychology, Maastricht University, P.O. Box 616 (PAR45), 6200 MD Maastricht, The Netherlands.
(Email: l.krabbendam@sp.unimaas.nl)

may also arise if both dimensions independently of each other increase the likelihood of need for care and help-seeking. This results in a spurious correlation between these dimensions in clinical settings, also known as treatment seeking bias or Berkson's bias. If Berkson's bias accounts for the correlations between the positive and mood dimensions in clinical samples, these correlations should be much lower in the general population.

It is possible to measure in the general population the same symptoms and experiences that are encountered in clinical samples. The implicit assumption of this approach is that experiencing certain symptoms is not inevitably associated with the presence of disorder. The latter may be dependent on symptom factors, such as intrusiveness and frequency of symptoms, and on personal and cultural factors, such as coping, illness behaviour and societal tolerance (Johns & Van Os, 2001). Thus, even though the prevalence of the clinical disorder may be low, the prevalence of the symptoms can be much higher. Based on this approach, several studies have suggested that the symptoms of psychosis are prevalent in the general population and exist as part of a continuous, albeit very skewed, distribution (Eaton *et al*. 1991; Barrett & Etheridge, 1992; Peters *et al*. 1999; Van Os *et al*. 2001; Stefanis *et al*. 2002). Similarly, the depression phenotype has been shown to exist as a continuous distribution of symptoms in the population (Anderson *et al*. 1993; Whittington & Huppert, 1996; Kendler & Gardner, 1998) and the same may be true for mania (Akiskal *et al*. 2000; Angst & Marneros, 2001).

In a previous study in the general population, experiences of depression and positive psychotic symptoms appeared as separable but highly correlated domains (Stefanis *et al*. 2002). These data, however, were based on self-report and did not include manic symptoms. The present study, therefore, was interview-based and designed to investigate whether three distinct, but correlated, dimensions of psychosis, depression and mania can be identified in the general population. In addition, we investigated finer dimensional distinctions within the main symptomatic domains. It has been shown that the positive dimension of psychosis is not a unidimensional construct, but can be better captured by a three- or even five-dimensional model, that distinguishes between hallucinations, non-Schneiderian delusions, Schneiderian delusions, formal thought disorder and bizarre behaviour (Minas *et al*. 1994; Peralta & Cuesta, 1998; Cardno *et al*. 2001). Likewise, studies in patients with depression have shown that the symptoms of depression can be divided into a cognitive and a somatic/affective dimension (Steer *et al*. 1999; Whisman *et al*. 2000), or into dimensions of core depression, psychic anxiety, psychomotor agitation and, less consistently, anorexia and insomnia (Fleck *et al*. 1995; Pancheri *et al*. 2002), depending on the characteristics of the sample and the instrument used. Few studies have investigated the structure of mania, but there is some evidence for at least five independent dimensions, representing dysphoric mood, increased hedonic function (including euphoric mood), psychosis, psychomotor pressure or hyperactivity, and irritable aggression (Cassidy *et al*. 1998). Two other possible manic syndromes are depressive inhibition and sleep disturbances (Sato *et al*. 2002). In the present study, exploratory factor analysis was performed in order to form an idea of the number and nature of the factors underlying the data. Subsequently, we used confirmatory factor analysis (CFA) to test statistically the fit of increasingly complex models.

To the extent that aetiological continuity is the source of overlap between the mood and the positive dimensions, the patterns of associations with demographic risk factors will be similar across the distinct dimensions of psychopathology, although there may be variation in effect size across dimensions (Van Os *et al*. 1998). Therefore, we investigated the effects of previously identified risk factors on the dimensions of psychopathology derived from the best-fitting model. These risk factors were age, sex, single marital status, neuroticism, family history of depression and family history of delusions and hallucinations.

METHOD

Sample

This study forms part of the Netherlands Mental Health Survey and Incidence Study (NEMESIS), a longitudinal study of the prevalence, incidence, course and consequences of psychiatric disorders in the Dutch general population. The

https://doi.org/10.1017/S0033291703001892 Published online by Cambridge University Press

study proposal was approved by the ethics committee of the Netherlands Institute of Mental Health and Addiction. Subjects were contacted at three points over a period of 3 years: at baseline, 1 year thereafter (T_1 – assessing the period between baseline and T_1) and again 2 years after T_1 (T_2 – assessing the period between T_1 and T_2) (Bijl *et al.* 1998*a*, *b*). A multi-stage, stratified, random sampling procedure was used to identify the sample. First, 90 municipalities were sampled randomly. Secondly, addresses from private households were randomly selected. Thirdly, the individual with the most recent birthday and aged 18–64 years was asked to participate. Individuals living in institutions, including individuals residing in psychiatric hospitals were not included in the sampling frame. All subjects received an introductory letter from the Minister of Health. A total of 7076 subjects were enlisted at baseline. The response rate was 69·7%. No difference in psychiatric morbidity based on the General Health Questionnaire, 12 items (GHQ-12; Goldberg & Williams, 1988) was found between responders and non-responders (Bijl *et al.* 1998*a*, *b*). At T_1, 5618 subjects participated for the second time; at T_2, 4848 subjects participated. Subjects with missing data for the purpose of the current investigation were excluded from the analyses. This led to the exclusion of four subjects from the baseline analysis, five from the analysis of the T_1 data and four from the analysis of the T_2 data.

Instruments

The sample was interviewed at home with the Composite International Diagnostic Interview, version 1.1 (CIDI; Smeets & Dingemans, 1993) at all three measurements. The CIDI is a fully structured interview that yields DSM-IIIR and ICD-10 diagnoses. It is designed for use by trained interviewers who are not clinicians and has satisfactory inter-rater reliability (Cottler *et al.* 1991; Wittchen *et al.* 1991) and test–retest reliability (Wittchen, 1994). Ninety interviewers experienced in systematic data collection administered the interview, having received a 3-day training course in recruiting and interviewing, followed by a 4-day course at the World Health Organization-CIDI training centre in Amsterdam, The Netherlands.

Symptoms of depression were examined using ratings from the 24 items of the CIDI core depression section (E). Items related to dysthymia were excluded, because the way these questions were asked induced correlations between the items (i.e. a screening question followed by four items that were asked when the screening item was endorsed). Symptoms of mania were assessed using ratings from the 11 items of the CIDI core mania section (F). All these items can be rated either 'yes' (1) or 'no' (0).

In order to assess psychotic experiences, ratings from the CIDI core psychosis section on delusions (13 items) and hallucinations (4 items) were used (items G1–G13, G15, G16, G20, G21). These concern classic psychotic symptoms involving, for example, thought interference and passivity phenomena (i.e. first-rank delusions), persecution and auditory hallucinations. All these items can be rated in six ways: '1', no symptom; '2', symptom present but not clinically relevant (not bothered by it and not seeking help for it); '3', symptom result of ingestion of drugs; '4', symptom result of somatic disease; '5', true psychiatric symptom; '6', symptom may not really be a symptom because there appears to be some plausible explanation for it. Because psychotic symptoms are difficult to diagnose using structured lay-interview, clinical re-interviews were conducted over the telephone by a senior registrar in psychiatry for all individuals who had at least one rating of 5 or 6, using questions from the Structured Clinical Interview for DSM-III-R (SCID), an instrument with proven reliability and validity in diagnosing schizophrenia (Spitzer *et al.* 1992). CIDI ratings of 5 and 6 were corrected on the basis of these clinical interviews. This reduces the possibility that endorsement rates for these ratings reflect misinterpretation of CIDI psychosis probes on the part of respondents or misinterpretation of their answers on the part of the lay interviewers. For the current investigation, scores of 2, 5 and 6 were considered relevant for inclusion in the analyses. The justification for this was derived from a previous study, where it was shown that the different ratings on the CIDI psychosis items were strongly associated with each other and independently showed a similar pattern of associations with known risk factors for psychosis (Van Os *et al.* 2000, 2001).

Level of neuroticism was assessed at baseline with the 14-item Groningen Neuroticism Scale (Ormel, 1980). Family history was examined at

T_1, by asking the subject whether any of his first-degree biological relatives had experienced a depression and/or delusions or hallucinations during the last 12 months.

Analyses

In order to examine patterns of clustering of the experiences of depression, mania and psychosis, Principal Component Analysis (PCA) with varimax rotation was performed in the STATA program, version 7 (Stata, 2001). The goal of PCA is to extract maximum variance from the data set with a few orthogonal components. We then compared the resulting model with more parsimonious models using Confirmatory Factor Analysis (CFA). CFA was carried out using LISREL, version 8.54 (Jöreskog & Sörbom, 1993). In CFA, predictions can be examined by relating the hypothesized symptom dimensions to empirical data in a factor analytic model. According to this model, the unobserved symptom dimensions are constructs, or latent variables, that can be studied indirectly through individual symptoms that can be considered as their indicators (Fergusson, 1997).

The most parsimonious, unidimensional model was based on the assumption that a single undifferentiated psychopathology factor underlies seemingly different symptom dimensions in the population. This is particularly relevant since recent studies of childhood psychopathology have found that the existence of up to eight dimensions of child problem behaviour derived from exploratory factor analysis of the Child Behaviour Checklist (Achenbach *et al.* 1989) were not supported by empirical data using CFA. Instead, it was found that a large proportion of the covariation among symptoms could be explained by one undifferentiated factor (Greenbaum & Dedrick, 1998; Hartman *et al.* 1999). In the two-dimensional model, patterns of comorbidity were assumed to be in accordance with the higher-order categories in the DSM-IV classification (APA, 1994), namely a mood dimension that included the depression and mania symptoms, and a psychosis dimension. Finally, we tested the fit of a three-dimensional model of depression, mania and psychosis. In order to examine any possible sex-differences, the best-fitting model was tested for males and females separately. The models were first tested in the baseline data, which contained lifetime ratings of psychopathology, and subsequently in the T_1 and T_2 data, in order to examine whether the results could be replicated using ratings of the 12-months and 24-months prevalence.

Structure in subsamples with and without a clinical diagnosis

In order to examine whether any findings could be the results of correlated symptom domains in the subgroup of individuals in the sample who had a lifetime DSM-IIIR diagnosis of depressive disorder ($n=1164$), bipolar disorder ($n=132$), or affective or non-affective psychotic disorder ($n=107$), the model-fitting analyses of the baseline data were repeated separately for the subsamples with and without any of these clinical diagnoses.

Analysis to account for low base rate of symptoms

Symptoms of mania and psychosis have a low base rate in the general population. In the case of rare symptoms, the estimated correlations between symptoms can become unacceptably imprecise, resulting in poor model fit even if the symptom is used as indicator for its natural dimension. In order to examine whether the results were unstable because of the low base rate of certain symptoms, all analyses were repeated in a more restricted sample, defined by the presence of at least three symptoms of depression and/or mania and/or psychosis. Application of this selection criterion increased the prevalence of symptoms compared to the primary analyses that included all individuals.

CFA

In CFA, a symptom covariance matrix is constructed based on the estimated values of three parameters, the factor loadings (or the extent to which the observed symptoms are related to the latent dimensions), the covariance between the factors (or the extent to which the latent dimensions are related to each other), and the unique factors of the observed symptoms (or the extent to which the observed symptoms contain variance that is unrelated to the latent dimensions). If the estimated covariance matrix is close to the observed covariance matrix, the model is said to fit the data well. The model parameters were

https://doi.org/10.1017/S0033291703001892 Published online by Cambridge University Press

estimated with the maximum likelihood procedure. The fit of the models was evaluated using the χ^2 statistic. This index reflects the discrepancy between the model-estimated and sample-derived correlations. The smaller the χ^2 statistic, the more the estimated and observed covariance matrices are consistent with each other. The normed fit index (NFI) was calculated in order to compare the χ^2 of the estimated models to the χ^2 of a model in which no interrelationships are assumed among any of the variables (Bentler & Bonnet, 1980). A model with NFI in the mid-0·90s or higher is viewed likely to represent a reasonably good approximation of the data.

While indices derived from the χ^2 statistic are useful to select the model that is most consistent with the data among a set of competing models, they do not necessarily indicate how consistent this model actually is with the observed data. This is especially the case in large samples, because large sample sizes lead to large χ^2 values, which may result in false rejection of the model. Therefore, we used other indices of fit, namely the standardized root mean square residual (SRMR), and the root mean square error of approximation (RMSEA). The SRMR is based on the size of the residuals and is least biased by sample size. A model with SRMR value of 0·05 or less is considered to fit the data reasonably well. The RMSEA allows for the description of discrepancy between the hypothesized model and the observed data, corrected for the size of the model. This measure takes into account both accuracy and parsimony of the model. An RMSEA value of 0·05 or less has been proposed as indicative of reasonable fit between model and data (Browne & Cudeck, 1993).

Associations between dimensions of psychopathology and known risk factors

In order to investigate the effect of known risk factors for psychotic disorders on the dimensions of psychopathology, regression analyses were performed using the latent factors as dependent variables. In the first analysis, age (five categories), sex, single status and neuroticism were entered simultaneously as independent variables in the analysis. In the second analysis, family history of depression and family history of delusions and hallucinations were used as independent variables.

Table 1. *Fit indices for 1-, 2-, 3- and 7-dimensional (D) models of symptoms of psychosis, depression and mania in the general population at baseline and follow-up ($p<0·001$)*

Factor models	χ^2*	df	NFI	SRMR	RMSEA
T_0 ($n=7072$)					
1D	33 983	1224	0·91	0·067	0·081
2D	23 497	1223	0·93	0·052	0·058
3D	19 098	1221	0·95	0·041	0·050
7D	12 798	1203	0·96	0·034	0·039
T_1 ($n=5613$)					
1D	31 971	1224	0·87	0·071	0·084
2D	23 145	1223	0·90	0·055	0·062
3D	20 901	1221	0·91	0·050	0·059
7D	15 592	1203	0·94	0·042	0·049
T_2 ($n=4844$)					
1D	34 913	1224	0·83	0·081	0·093
2D	24 926	1223	0·88	0·059	0·069
3D	22 882	1221	0·89	0·053	0·065
7D	17 177	1203	0·92	0·047	0·054

NFI, normed fit index; SRMR, standardized root mean square residual; RMSEA, root mean square error of approximation.
* Rounded to nearest integer.

RESULTS

PCA

The PCA yielded 11 factors with an eigenvalue >1. As several factors were not interpretable, we explored factor solutions with a smaller number of factors, until a seven-factor solution was obtained which was fully interpretable. The seven-factor solution consisted of three factors related to depression, that were denoted core depression, sleep problems and suicidal thoughts, one mania factor and three psychosis factors, that were denoted paranoid and other delusions, first-rank delusions and hallucinations. Based on theoretical grounds (see above), this model was then compared to a three-dimensional model, a two-dimensional model and a unidimensional model.

Latent dimensions at baseline

The results revealed that the seven-dimensional model provided a better fit to the data than the one-, two- and three-dimensional models (Table 1). The unidimensional model provided the poorest fit. All fit-indices showed a similar pattern, the seven-dimensional model yielding the highest NFI (0·96) and the lowest SRMR (0·034) and RMSEA (0·039), which suggests that this model is reasonably close to the data. The three-dimensional model (NFI = 0·95,

https://doi.org/10.1017/S0033291703001892 Published online by Cambridge University Press

Table 2. *Correlations between the seven latent dimensions of psychopathology at baseline*

	Core depression	Sleep problems	Suicidal thoughts	Mania	Paranoid delusions	First-rank delusions	Hallucinations
Core depression	1						
Sleep problems	0·66	1					
Suicidal thoughts	0·66	0·39	1				
Mania	0·61	0·43	0·51	1			
Paranoid delusions	0·35	0·20	0·35	0·55	1		
First-rank delusions	0·29	0·18	0·32	0·46	0·71	1	
Hallucinations	0·33	0·23	0·38	0·50	0·54	0·73	1

SRMR = 0·041, RMSEA = 0·050) was also reasonable, but fitted the data less well than the seven-dimensional model. As expected, all seven latent dimensions covaried with each other, with correlations in the range from 0·18 to 0·73 (see Table 2). Stratified analyses by sex indicated that the fit of the seven-dimensional model was satisfactory for both males ($\chi^2 = 7803$, df = 1203, $p < 0{\cdot}001$; NFI = 0·95, SRMR = 0·038, RMSEA = 0·043) and females ($\chi^2 = 8388$, df = 1203, $p < 0{\cdot}001$; NFI = 0·96, SRMR = 0·038, RMSEA = 0·042).

Structural replication at T_1 and T_2

In order to examine the structure of current psychopathology, fit indices were computed for each of the tested models for the samples at T_1 and T_2 separately. Again, the model with seven dimensions provided the best fit (see Table 1). The fit indices displayed a similar pattern as was found for the baseline assessment, with the seven-dimensional model giving the highest NFI, and the lowest SRMR and RMSEA, followed by the three-dimensional model, then the two-dimensional model, and finally the unidimensional model.

Structure in subsamples with and without a clinical diagnosis

After exclusion of all individuals with a lifetime prevalence of a diagnosis of depressive disorder, bipolar disorder, or non-affective psychotic disorder (resulting sample size 5780), the seven-dimensional model again fitted the data well ($\chi^2 = 9917$, df = 1203, $p < 0{\cdot}001$; NFI = 0·89, SRMR = 0·035, RMSEA = 0·037). When the sample was restricted to the individuals with a diagnosis of depressive disorder, bipolar disorder or non-affective psychotic disorder (resulting sample size 1293), the model with seven dimensions again provided a reasonable fit, although slightly worse than in the subsample without a clinical diagnosis ($\chi^2 = 3793$, df = 1203, $p < 0{\cdot}001$; NFI = 0·88, SRMR = 0·047, RMSEA = 0·043).

Analyses to account for low base rate of symptoms

The analysis was repeated in a more restricted sample, defined by the presence of at least three symptoms of depression and/or mania and/or psychosis (resulting sample size 3565). Again, the seven-dimensional model fitted the data well ($\chi^2 = 10\,467$, df = 1203, $p < 0{\cdot}001$; NFI = 0·88, SRMR = 0·047, RMSEA = 0·048).

Associations with risk factors

Except for sleep problems, the risk of having experiences of psychopathology decreased with increasing age for all dimensions (see Table 3). Female sex was significantly associated with the three depression dimensions and hallucinations, while male sex was associated with mania and non-significantly with paranoid and first-rank delusions. Single marital status increased the risk for the psychosis dimensions and non-significantly for mania and suicidal thoughts, but had the opposite effect for core depression and sleep problems, although not significantly so. Level of neuroticism was significantly and positively associated with all seven dimensions, with the strongest effect being found for the mood dimensions. There was a significant effect of family history on all seven dimensions, which was particularly strong for the effect of having a family member with depression.

DISCUSSION

Using positive psychotic and mood dimensions of psychopathology in clinical samples as a template, this study examined whether correlated

https://doi.org/10.1017/S0033291703001892 Published online by Cambridge University Press

Table 3. *Associations between risk factors and dimensions of psychopathology in the general population, expressed as standardized regression coefficients*

	Age	Sex	Single	Neuroticism	FH depression	FH psychosis
Core depression	−0·047**	0·103***	−0·024	0·520***	0·187***	0·049***
Sleep problems	0·133***	0·100***	−0·031	0·433***	0·144***	0·034*
Suicidal thoughts	−0·043	0·072***	0·036	0·463***	0·164***	0·039*
Mania	−0·133***	−0·100***	0·037	0·447***	0·157***	0·047**
Paranoid delusions	−0·046	−0·048	0·094**	0·335***	0·126***	0·039
First-rank delusions	−0·071*	−0·026	0·085*	0·334***	0·158***	0·067**
Hallucinations	−0·062*	0·053*	0·098***	0·370***	0·110***	0·065**

FH, Family history.
* $p<0·05$; ** $p<0·01$; *** $p<0·001$.

but separable dimensions of depression, mania, and psychosis had a distribution in the general population. In a population sample of 7072 individuals, there was evidence for the existence of seven correlated but separable dimensions of experiences that resembled dimensions of psychopathology seen in clinical samples: core depression, sleep problems, suicidal thoughts, mania, paranoid delusions, first-rank delusions and hallucinations. Compared to the three-, two-, and unidimensional models, the seven-dimensional model fitted the data best across clinical, non-clinical and follow-up measurements. A consistent finding was that the unidimensional model provided the poorest fit, indicating that covariation among the experiences of depression, mania and psychosis in the general population cannot be seen as the expression of a single undifferentiated factor. At the same time, the high correlations between dimensions suggest that they are highly 'co-morbid' and vary in part as a result of the same underlying cause.

The baseline assessment relied on lifetime diagnoses, so that no distinction could be made between subjects with current experiences of depression, mania or psychosis and those with these experiences temporally separated over the lifespan. However, the replication of the results at both follow-up measurements indicated that the seven-dimensional model also reflects patterns of covariation among current symptomatology.

The substantial correlations between the latent dimensions in the general population sample reflect the substantial degree of overlap seen in clinical samples (Peralta & Cuesta, 1999; Van Os *et al.* 1999). Co-morbidity may arise from a number of sources (Klein & Riso, 1993). Berkson's bias may affect the results, when subjects with more than one disorder are more likely to be part of a clinical sample. Our findings in the general population suggest that the overlap cannot be solely explained by the effect of this sampling bias. Another source of apparent co-morbidity is population stratification, which occurs when two disorders have non-overlapping sets of risk factors, but these risk factors both tend to be more common in certain strata of the population (Klein & Riso, 1993). Alternatively, having symptoms of disorder A may itself generate symptoms of disorder B, so that these symptoms arise because of disorder A and are unrelated to liability for disorder B (e.g. the heterogeneity model suggested by Klein and Riso). Finally, the co-morbidity of symptom domains may be the result of sharing of causal risk factors. Familial co-aggregation suggests that schizophrenia shares common genetic risk factors with both bipolar and unipolar disorder (Kendler & Gardner, 1997; Potash *et al.* 2001; Bailer *et al.* 2002; Cardno *et al.* 2002) and there is similar evidence for a genetic relationship between bipolar and unipolar disorder (Blacker *et al.* 1996; Karkowski & Kendler, 1997; McGuffin *et al.* 2003). Risk factors associated with life events, ethnic group, prenatal famine and urban birth tend to overlap between psychotic and mood disorders (Bebbington *et al.* 1993; Van Os *et al.* 1996; Marcelis *et al.* 1998; Brown *et al.* 2000) as do cognitive impairments and high levels of neuroticism prior to the onset of disorder (Jones *et al.* 1994; Van Os & Jones, 2001; Myin-Germeys *et al.* 2003; Goodwin *et al.* 2003). The current findings may suggest that the same shared risk factors also cause the overlap

https://doi.org/10.1017/S0033291703001892 Published online by Cambridge University Press

on lower levels of the continuum. For some of the risk factors, differences were qualitative, suggesting that these are part of the unique liabilities. Thus, female sex increased the risk for all dimensions of depression, while decreasing the risk for the dimension of mania. For other indicators of risk, however, differences in effect size were quantitative. Level of neuroticism, family history of depression and family history of delusions and hallucinations increased the risk for all seven dimensions of psychopathology. This would be indicative of continuity between correlated dimensions of psychopathology at the clinical and subclinical level. Support for this hypothesis comes from studies in samples with subclinical psychotic psychopathology that show similar neuropsychological abnormalities and associations with risk factors as the ones that have been reported in the clinical disorder (Lenzenweger & Korfine, 1994; Chen *et al*. 1997; Verdoux *et al*. 1998; Van Os *et al*. 2000; Voglmaier *et al*. 2000). Thus, the results may suggest that aetiological factors impact on the general population, resulting in continuous and correlated experience of affective and non-affective dimensions across the population, the extremes of which are disorders that come to the attention of services.

Methodological issues

The results should be considered in the light of several methodological limitations. First, the choice of instruments and the level of analysis influence the identification of psychopathological dimensions. Studies at the symptom level yield more complex factor structures than do studies that use global ratings (Peralta & Cuesta, 2001). Although we assessed psychopathology at the symptom level, the selection of items may in some respects be considered limited. This was particularly true for the measurement of the dimension of mania, which did not allow for the replication of the structure of mania that has been found in a previous study (Cassidy *et al*. 1998). Likewise, our investigation was limited to the positive symptoms of psychosis and did not include measures of negative symptoms, disorganization and lack of insight, all of which have been shown to constitute major dimensions of psychopathology (Peralta & Cuesta, 2001).

Second, the low base rate of symptoms of mania and psychosis poses a problem for analyses like the current investigation, because the estimated correlations between rare symptoms can become imprecise. However, after exclusion of individuals with less than three symptoms of depression, mania or psychosis, the fit of the seven-dimensional model was still satisfactory. In addition, the seven-dimensional solution appeared to be robust and displayed the best fit through a number of different measurement occasions (i.e. in the unrestricted samples at baseline and both follow-up measurements, in the restricted sample defined by the presence of at least three symptoms of depression, mania or psychosis, in the sample of individuals with a clinical diagnosis, and in the sample of individuals without a clinical diagnosis).

Third, the CIDI assessment of symptoms does not include the degree of distress or disability associated with symptoms. A recent comparison between the CIDI and the Schedules for Clinical Assessment in Neuropsychiatry (SCAN; Wing *et al*. 1990) showed that higher prevalence rates obtained by the CIDI reflected the stricter requirement in the SCAN that each symptom must be distressing, difficult to control and excessive under the circumstances in order for it to be rated (Brugha *et al*. 2001). Thus, many endorsed CIDI items were considered by the SCAN interviewer but judged as subthreshold. However, these items should not be seen as 'false-positives', as there is now substantial evidence that even psychotic experiences may occur in the population without accompanying distress (Kendler & Gardner, 1998; Henderson *et al*. 2001; Van Os *et al*. 2001). For example, we have previously reported evidence that community level of non-distressing psychosis-like experiences is linked to the prevalence of psychotic disorder and similarly associated with known risk factors for psychotic disorder (Van Os *et al*. 2000, 2001).

Fourth, good model fit does not necessarily mean that the model dimensions exist in nature. Structural equation modelling only suggested that the seven-dimensional model provided a better fit to the observed data than the other models. Structural equation models test *a priori* hypothesized relations among variables. If these *a priori* assumptions are incorrect, the resulting model will also be incorrect, no matter how close the model fits the data statistically.

REFERENCES

Achenbach, T. M., Conners, C. K., Quay, H. C., Verhulst, F. C. & Howell, C. T. (1989). Replication of empirically derived syndromes as a basis for taxonomy of child/adolescent psychopathology. *Journal of Abnormal Child Psychology* **17**, 299–323.

Akiskal, H. S., Bourgeois, M. L., Angst, J., Post, R., Moller, H. & Hirschfeld, R. (2000). Re-evaluating the prevalence of and diagnostic composition within the broad clinical spectrum of bipolar disorders. *Journal of Affective Disorders* **59** (Suppl. 1), S5–S30.

Anderson, J., Huppert, F. & Rose, G. (1993). Normality, deviance and minor psychiatric morbidity in the community. A population-based approach to General Health Questionnaire data in the Health and Lifestyle Survey. *Psychological Medicine* **23**, 475–485.

Angst, J. & Marneros, A. (2001). Bipolarity from ancient to modern times: conception, birth and rebirth. *Journal of Affective Disorders* **67**, 3–19.

APA (1994). *Diagnostic and Statistic Manual of Mental Disorders*. American Psychiatric Press: Washington, DC.

Bailer, U., Leisch, F., Meszaros, K., Lenzinger, E., Willinger, U., Strobl, R., Heiden, A., Gebhardt, C., Doge, E., Fuchs, K., Sieghart, W., Kasper, S., Hornik, K. & Aschauer, H. N. (2002). Genome scan for susceptibility loci for schizophrenia and bipolar disorder. *Biological Psychiatry* **52**, 40–52.

Barrett, T. R. & Etheridge, J. B. (1992). Verbal hallucinations in normals. I. People who hear voices. *Applied Cognitive Psychology* **6**, 379–387.

Bebbington, P., Wilkins, S., Jones, P. B., Foerster, A., Murray, R., Toone, B. & Lewis, S. (1993). Life-events and psychosis. Initial results from the Camberwell Collaborative Psychosis Study. *British Journal of Psychiatry* **162**, 72–79.

Bentler, P. M. & Bonett, D. G. (1980). Significance tests and goodness of fit in the analysis of covariance structures. *Psychological Bulletin* **88**, 588–606.

Bijl, R. V., Van Zessen, G., Ravelli, A., De Rijk, C. & Langendoen, Y. (1998*a*). The Netherlands Mental Health Survey and Incidence Study (NEMESIS): objectives and design. *Social Psychiatry and Psychiatric Epidemiology* **33**, 581–586.

Bijl, R. V., Ravelli, A. & Van Zessen, G. (1998*b*). Prevalence of psychiatric disorder in the general population: results of The Netherlands Mental Health Survey and Incidence Study (NEMESIS). *Social Psychiatry and Psychiatric Epidemiology* **33**, 587–595.

Blacker, D., Faraone, S. V., Rosen, A. E., Guroff, J. J., Adams, P., Weissman, M. M. & Gershon, E. S. (1996). Unipolar relatives in bipolar pedigrees: a search for elusive indicators of underlying bipolarity. *American Journal of Medical Genetics* **67**, 445–454.

Brockington, I. F., Kendell, R. E. & Wainwright, S. (1980). Depressed patients with schizophrenic or paranoid symptoms. *Psychological Medicine* **10**, 665–675.

Brockington, I. F., Kendell, R. E., Wainwright, S., Hillier, V. F. & Walker, J. (1979). The distinction between the affective psychoses and schizophrenia. *British Journal of Psychiatry* **135**, 243–248.

Brown, A. S., Van Os, J., Driessens, C., Hoek, H. W. & Susser, E. S. (2000). Further evidence of relation between prenatal famine and major affective disorder. *American Journal of Psychiatry* **157**, 190–195.

Browne, M. W. & Cudeck, R. (1993). Alternative ways of assessing model fit. In *Testing Structural Equation Models* (ed. K. A. Bollen and J. S. Long), pp. 28–57. Sage: Newbury Park, SA.

Brugha, T. S., Jenkins, R., Taub, N., Meltzer, H. & Bebbington, P. E. (2001). A general population comparison of the Composite International Diagnostic Interview (CIDI) and the Schedules for Clinical Assessment in Neuropsychiatry (SCAN). *Psychological Medicine* **31**, 1001–1013.

Cardno, A. G., Rijsdijk, F. V., Sham, P. C., Murray, R. M. & McGuffin, P. (2002). A twin study of genetic relationships between psychotic symptoms. *American Journal of Psychiatry* **159**, 539–545.

Cardno, A. G., Sham, P. C., Murray, R. M. & McGuffin, P. (2001). Twin study of symptom dimensions in psychoses. *British Journal of Psychiatry* **179**, 39–45.

Cassidy, F., Forest, K., Murry, E. & Carroll, B. J. (1998). A factor analysis of the signs and symptoms of mania. *Archives of General Psychiatry* **55**, 27–32.

Chen, W. J., Hsiao, C. K. & Lin, C. C. (1997). Schizotypy in community samples: the three-factor structure and correlation with sustained attention. *Journal of Abnormal Psychology* **106**, 649–654.

Cottler, L. B., Robins, L. N., Grant, B. F., Blaine, J., Towle, L. H., Wittchen, H. U. & Sartorius, N. (1991). The CIDI-core substance abuse and dependence questions: cross-cultural and nosological issues. The WHO/ADAMHA Field Trial. *British Journal of Psychiatry* **159**, 653–658.

Cuesta, M. J. & Peralta, V. (2001). Integrating psychopathological dimensions in functional psychoses: a hierarchical approach. *Schizophrenia Research* **52**, 215–229.

Eaton, W. W., Romanoski, A., Anthony, J. C. & Nestadt, G. (1991). Screening for psychosis in the general population with a self-report interview. *Journal of Nervous and Mental Disease* **179**, 689–693.

Fergusson, D. M. (1997). Annotation: structural equation models in developmental research. *Journal of Child Psychology and Psychiatry* **38**, 877–887.

Fleck, M. P., Poirier-Littre, M. F., Guelfi, J. D., Bourdel, M. C. & Loo, H. (1995). Factorial structure of the 17-item Hamilton Depression Rating Scale. *Acta Psychiatrica Scandinavica* **92**, 168–172.

Goldberg, D. & Williams, P. (1988). *User's Guide to the GHQ*. NFER-Nelson: Windsor, UK.

Goodwin, R. D., Fergusson, D. M. & Horwood, L. J. (2003). Neuroticism in adolescence and psychotic symptoms in adulthood. *Psychological Medicine* **33**, 1089–1097.

Greenbaum, P. & Dedrick, R. (1998). Hierarchical confirmatory factor analysis of the Child Behavior Checklist/4–18. *Psychological Assessment* **10**, 149–155.

Hartman, C. A., Hox, J., Auerbach, J., Erol, N., Fonseca, A. C., Mellenbergh, G. J., Novik, T. S., Oosterlaan, J., Roussos, A. C., Shalev, R. S., Zilber, N. & Sergeant, J. A. (1999). Syndrome dimensions of the child behavior checklist and the teacher report form: a critical empirical evaluation. *Journal of Child Psychology and Psychiatry* **40**, 1095–1116.

Henderson, S., Korten, A. & Medway, J. (2001). Non-disabled cases in a national survey. *Psychological Medicine* **31**, 769–777.

Johns, L. C. & Van Os, J. (2001). The continuity of psychotic experiences in the general population. *Clinical Psychology Review* **21**, 1125–1141.

Jones, P., Rodgers, B., Murray, R. & Marmot, M. (1994). Child development risk factors for adult schizophrenia in the British 1946 birth cohort. *Lancet* **344**, 1398–1402.

Jöreskog, K. G. & Sörbom, D. (1993). *Lisrel 8: Structural Equation Modeling with the Simplis Command Language*. Lawrence Erlbaum Associates: Hillsdale, NJ.

Karkowski, L. M. & Kendler, K. S. (1997). An examination of the genetic relationship between bipolar and unipolar illness in an epidemiological sample. *Psychiatric Genetics* **7**, 159–163.

Kendler, K. S. & Gardner, C. O. (1997). The risk for psychiatric disorders in relatives of schizophrenic and control probands: a comparison of three independent studies. *Psychological Medicine* **27**, 411–419.

Kendler, K. S. & Gardner Jr, C. O. (1998). Boundaries of major depression: an evaluation of DSM-IV criteria. *American Journal of Psychiatry* **155**, 172–177.

Kitamura, T., Okazaki, Y., Fujinawa, A., Yoshino, M. & Kasahara, Y. (1995). Symptoms of psychoses. A factor-analytic study. *British Journal of Psychiatry* **166**, 236–240.

Klein, D. N. & Riso, L. P. (1993). Psychiatric disorders: problems of boundaries and comorbidity. In *Basic Issues in Psychopathology* (ed. C. G. Costello), pp. 19–66. Guilford Press: New York.

Lenzenweger, M. F. & Korfine, L. (1994). Perceptual aberrations, schizotypy, and the Wisconsin Card Sorting Test. *Schizophrenia Bulletin* **20**, 345–357.

Marcelis, M., Navarro Mateu, F., Murray, R., Selten, J. P. & Van Os, J. (1998). Urbanization and psychosis: a study of 1942–1978

birth cohorts in The Netherlands. *Psychological Medicine* **28**, 871–879.

McGorry, P. D., Bell, R. C., Dudgeon, P. L. & Jackson, H. J. (1998). The dimensional structure of first episode psychosis: an exploratory factor analysis. *Psychological Medicine* **28**, 935–947.

McGuffin, P., Rijsdijk, F., Andrew, M., Sham, P., Katz, R. & Cardno, A. (2003). The heritability of bipolar affective disorder and the genetic relationship to unipolar depression. *Archives of General Psychiatry* **60**, 497–502.

McIntosh, A. M., Forrester, A., Lawrie, S. M., Byrne, M., Harper, A., Kestelman, J. N., Best, J. J., Johnstone, E. C. & Owens, D. G. (2001). A factor model of the functional psychoses and the relationship of factors to clinical variables and brain morphology. *Psychological Medicine* **31**, 159–171.

Minas, I. H., Klimidis, S., Stuart, G. W., Copolov, D. L. & Singh, B. S. (1994). Positive and negative symptoms in the psychoses: principal components analysis of items from the Scale for the Assessment of Positive Symptoms and the Scale for the Assessment of Negative Symptoms. *Comprehensive Psychiatry* **35**, 135–144.

Murray, R. M., Walker, J., Sham, P. & Van Os, J. (2003) Schizophrenia and bipolar illness: a single illness divided by the presence or absence of neurodevelopmental impairment? *submitted*.

Myin-Germeys, I., Peeters, F., Havermans, R., Nicolson, N., Delespaul, P., deVries, M. & Van Os, J. (2003). Emotional reactivity to daily life stress in psychosis and affective disorder: an Experience Sampling Study. *Acta Psychiatrica Scandinavica* **107**, 124–131.

Ormel, J. (1980) Moeite met leven of een moeilijk leven [Difficulties with living or a difficult life]. University of Groningen: Groningen, The Netherlands.

Pancheri, P., Picardi, A., Pasquini, M., Gaetano, P. & Biondi, M. (2002). Psychopathological dimensions of depression: a factor study of the 17-item Hamilton depression rating scale in unipolar depressed outpatients. *Journal of Affective Disorders* **68**, 41–47.

Peralta, V. & Cuesta, M. J. (1998). Factor structure and clinical validity of competing models of positive symptoms in schizophrenia. *Biological Psychiatry* **44**, 107–114.

Peralta, V. & Cuesta, M. J. (1999). Dimensional structure of psychotic symptoms: an item-level analysis of SAPS and SANS symptoms in psychotic disorders. *Schizophrenia Research* **38**, 13–26.

Peralta, V. & Cuesta, M. J. (2001). How many and which are the psychopathological dimensions in schizophrenia? Issues influencing their ascertainment. *Schizophrenia Research* **49**, 269–285.

Peters, E. R., Joseph, S. A. & Garety, P. A. (1999). Measurement of delusional ideation in the normal population: introducing the PDI (Peters *et al*. Delusions Inventory). *Schizophrenia Bulletin* **25**, 553–576.

Potash, J. B., Willour, V. L., Chiu, Y. F., Simpson, S. G., MacKinnon, D. F., Pearlson, G. D., DePaulo Jr, J. R. & McInnis, M. G. (2001). The familial aggregation of psychotic symptoms in bipolar disorder pedigrees. *American Journal of Psychiatry* **158**, 1258–1264.

Sato, T., Bottlender, R., Kleindienst, N. & Moller, H. J. (2002). Syndromes and phenomenological subtypes underlying acute mania: a factor analytic study of 576 manic patients. *American Journal of Psychiatry* **159**, 968–974.

Siris, S. G. (2000). Depression in schizophrenia: perspective in the era of 'atypical' antipsychotic agents. *American Journal of Psychiatry* **157**, 1379–1389.

Smeets, R. M. W. & Dingemans, P. M. A. J. (1993). *Composite International Diagnostic Interview* (*CIDI*) *Version 1.1*. World Health Organization: Amsterdam/Geneva.

Spitzer, R. L., Williams, J. B., Gibbon, M. & First, M. B. (1992). The Structured Clinical Interview for DSM-III-R (SCID). I. History, rationale, and description. *Archives of General Psychiatry* **49**, 624–629.

STATA (2001). STATA Statistical Software: Release 7.0. STATA Inc.: College Station, TX.

Steer, R. A., Ball, R., Ranieri, W. F. & Beck, A. T. (1999). Dimensions of the Beck Depression Inventory-II in clinically depressed outpatients. *Journal of Clinical Psychology* **55**, 117–128.

Stefanis, N. C., Hanssen, M., Smirnis, N. K., Avramopoulos, D. A., Evdokimidis, I. K., Stefanis, C. N., Verdoux, H. & Van Os, J. (2002). Evidence that three dimensions of psychosis have a distribution in the general population. *Psychological Medicine* **32**, 347–358.

Taylor, M. A. (1992). Are schizophrenia and affective disorder related? A selective literature review. *American Journal of Psychiatry* **149**, 22–32.

Van Os, J., Gilvarry, C., Bale, R., Van Horn, E., Tattan, T., White, I. & Murray, R. (1999). A comparison of the utility of dimensional and categorical representations of psychosis. UK700 Group. *Psychological Medicine* **29**, 595–606.

Van Os, J., Hanssen, M., Bijl, R. V. & Ravelli, A. (2000). Strauss (1969) revisited: a psychosis continuum in the general population? *Schizophrenia Research* **45**, 11–20.

Van Os, J., Hanssen, M., Bijl, R. V. & Vollebergh, W. (2001). Prevalence of psychotic disorder and community level of psychotic symptoms: an urban-rural comparison. *Archives of General Psychiatry* **58**, 663–668.

Van Os, J., Jones, P., Sham, P., Bebbington, P. & Murray, R. M. (1998). Risk factors for onset and persistence of psychosis. *Social Psychiatry and Psychiatric Epidemiology* **33**, 596–605.

Van Os, J. & Jones, P. B. (2001). Neuroticism as a risk factor for schizophrenia. *Psychological Medicine* **31**, 1129–1134.

Van Os, J., Takei, N., Castle, D. J., Wessely, S., Der, G., MacDonald, A. M. & Murray, R. M. (1996). The incidence of mania: time trends in relation to gender and ethnicity. *Social Psychiatry and Psychiatric Epidemiology* **31**, 129–136.

Verdoux, H., Van Os, J., Maurice-Tison, S., Gay, B., Salamon, R. & Bourgeois, M. (1998). Is early adulthood a critical developmental stage for psychosis proneness? A survey of delusional ideation in normal subjects. *Schizophrenia Research* **29**, 247–254.

Voglmaier, M. M., Seidman, L. J., Niznikiewicz, M. A., Dickey, C. C., Shenton, M. E. & McCarley, R. W. (2000). Verbal and nonverbal neuropsychological test performance in subjects with schizotypal personality disorder. *American Journal of Psychiatry* **157**, 787–793.

Whisman, M. A., Perez, J. E. & Ramel, W. (2000). Factor structure of the Beck Depression Inventory-Second Edition (BDI- II) in a student sample. *Journal of Clinical Psychology* **56**, 545–551.

Whittington, J. E. & Huppert, F. A. (1996). Changes in the prevalence of psychiatric disorder in a community are related to changes in the mean level of psychiatric symptoms. *Psychological Medicine* **26**, 1253–1260.

Wing, J. K., Babor, T., Brugha, T., Burke, J., Cooper, J. E., Giel, R., Jablenski, A., Regier, D. & Sartorius, N. (1990). SCAN. Schedules for Clinical Assessment in Neuropsychiatry. *Archives of General Psychiatry* **47**, 589–593.

Wittchen, H. U. (1994). Reliability and validity studies of the WHO – Composite International Diagnostic Interview (CIDI): a critical review. *Journal of Psychiatric Research* **28**, 57–84.

Wittchen, H. U., Robins, L. N., Cottler, L. B., Sartorius, N., Burke, J. D. & Regier, D. (1991). Cross-cultural feasibility, reliability and sources of variance of the Composite International Diagnostic Interview (CIDI). The Multicentre WHO/ADAMHA Field Trials. *British Journal of Psychiatry* **159**, 645–653.