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ORIGINAL PAPER

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Psychopathological dimensions in first-episode psychoses

From the trunk to the branches and leaves

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Abstract *Background* Dimensional frameworks for structuring psychopathology have been formulated in recent years to overcome classification problems of categorical approaches. However, few studies have addressed the dilemma of hierarchy within symptoms or dimensions in psychosis. *Methods* This study was designed to examine the hierarchical structure of psychopathological dimensions in first episode psychosis. The sample consisted of 94 first-episode patients psychosis. An exhaustive psychopathological assessment was carried out using the AMDP-system. Consecutive principal component analyses of AMDP symptoms, determining 'a priori' the number of factors to be extracted, were carried out. *Results* Following the track of the resulting factor analyses, a 'vertical hierarchical' framework was achieved. Our schema organized dimensions in a series of echelons in which lower tiers are subsumed as subsets of those assigned to higher ranks. In addition, a final model comprising 10 dimensions provided an 'horizontal' and multidimensional structure comprising all relevant psychopathological dimensions in first-episode psychosis. *Conclusions* This study confirmed to a great extent the existence of a hierarchical organization within psychopathological dimensions in 'first-episode' psychosis. The present 'hierarchical and multidimensional' model of psychopathological dimensions allows for selection of the level of complexity of 'candidate phenotypes' to use in neurobiological research of psychosis.

Key words schizophrenia · psychosis · first-episode psychosis · psychopathological dimensions · hierarchical structure

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Introduction

The solution of the biological underpinnings of schizophrenia, and by extension of psychosis, has not been possible during the last century probably due to the neurobiological inconsistency of psychopathological models. Clinical 'prototypes' of psychoses were clearly described in the early years of the past century and they have been maintained with only slight modifications in modern nosotaxias, such as the DSM system (APA 1994). There are three important problems inherent to the psychopathological model of psychoses: heterogeneity, lack of stability of diagnosis and comorbidity.

Multidimensional models, such as the three-syndrome model (Liddle 1987), have been advocated to account for these problems (Stuart et al. 1999; Peralta and Cuesta 2001a). However, this model is not exempt of conceptual and methodological shortcomings and three dimensions seem to be a rather simple description of the rich psychopathological manifestations of schizophrenia. Recently, we proposed a new hierarchical and multidimensional model to overcome some of the limitations of three-dimension models (Cuesta and Peralta 2001).

The purpose of the present study was twofold: first, to advance our understanding of the basic dimensions and hierarchical structure of psychopathological symptoms in psychosis, and second, to further investigate the stability of our hierarchical and multidimensional model by examining the model in a 'first-episode' psychosis sample using a similar analytical strategy.

Methods

Subjects were 94 first-episode psychosis patients consecutively admitted to an acute unit in Vitoria (Spain) (Table 1). All patients gave

Table 1 Demographic and clinical characteristics of sample (n = 94)

	Mean	SD
Age at hospitalization	27.55	9.08
Age at onset	26.05	8.93
	N	%
Sex		
Men	68	72.3
Women	26	27.7
DSM-IV Diagnosis		
Schizophrenic disorder	33	35.1
Schizophreniform disorder	12	12.8
Brief psychotic disorder	19	20.2
Delusional disorder	7	7.4
Manic disorder with psychotic symptoms	21	22.3
Major depressive disorder with psychotic symptoms	2	2.1
Previous treatment status		
Never medicated	69	73.4
Previously medicated but not with antipsychotic drugs	15	15.9
Received previously antipsychotic drugs	10	10.6

written informed consent to enter into the study. Inclusion criteria were 1) to present an acute psychotic episode, which was defined by a score of 4 or greater in any of the following three items of PANSS scale: delusions, conceptual disorganization or hallucinatory behavior, and 2) age between 18 and 65 years. Patients were excluded if they had 'previous admissions' or antecedents of neurological illness, head trauma or substance dependence. Sixty-nine patients were drug-naive (73.4 %) and 84 (89.3 %) had not previously received antipsychotic drugs.

Clinical assessment

Diagnoses were based on the information gathered from patients directly and from family members through the Structured Clinical Interview for DSM-III-R (SCID) (Spitzer et al. 1992). Diagnoses were updated to meet DSM-IV criteria (APA 1994) after consensus between raters. In addition, to evaluate a wide range of psychopathological phenomena patients were assessed by PG through the Manual for the Assessment and Documentation of Psychopathology (AMDP) (Pietzcker et al. 1983). The AMDP system includes definitions of 100 symptoms extracted from classic psychopathology. These 100 symptoms have operationalized criteria and are scored on a 4-point scale ranging from 0 (absence) to 3 (severe) according with its presence and severity (Guy and Ban 1982).

Statistical analysis

Data analysis was conducted in three stages. First, an inspection of the 100 items of the AMDP inventory was performed to select symptoms by their base rate frequencies in order to avoid bias due to symptoms of very low prevalence. Thus, 70 out of the 100 AMDP symptoms were entered in the analysis since they scored 1 or higher in at least 15 % of patients.

In the next step, consecutive principal component analyses with the number of factors to be extracted fixed 'a priori' were carried out. Analyses of rotated factor matrices from 1 to 10 factors were inspected. Oblique rotation of the factors was used since clinical dimensions are not independent but related phenomena (Hair et al. 1992). Following the track of symptoms from high-order to low-order dimensions on consecutive factor analyses, it was possible to obtain a graphic representation of its hierarchical structure and to comprehend the origin of psychopathological dimensions. Internal consistency of the final dimensions was evaluated through Cronbach alpha test (Cronbach 1951). Only items with significant loadings in each factor were computed in alpha coefficients.

Results

Principal component analyses

Fig. 1 shows the graphic representation of the set of factor analysis from 1 to 10 factors, in which items were fused from lower to higher order levels to reach the

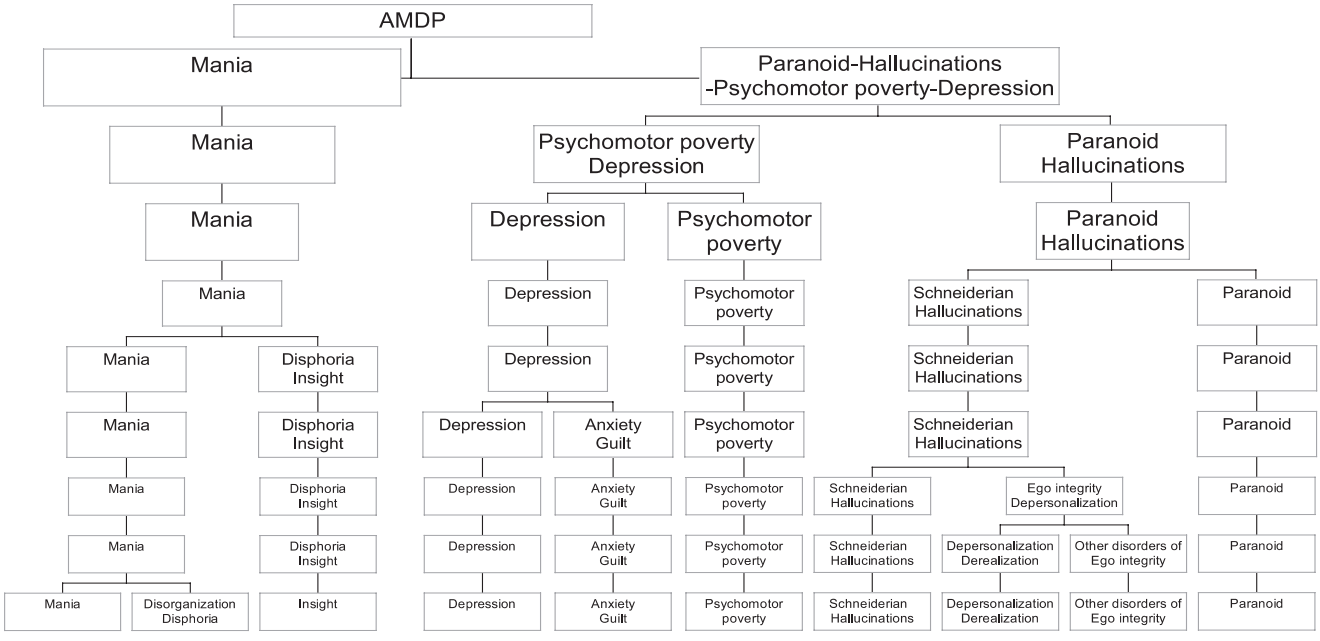

Fig. 1 AMDP Hierarchical and multidimensional of first-episode psychosis (symptom prevalence ≥ 15 %)

trunk of general psychopathology factor (n = 1 dimension).

The one-factor solution resulting from the first factor analysis reflected a global score of psychopathology since it consisted in a combination of all items of the AMDP inventory. *At the second level* of factor analysis, two factors were extracted with a first factor with high loadings (> 0.60) in manic symptoms (euphoria, pressure of speech, flight of ideas, delusions of grandeur, exaggerate self-confidence, hyperactivity, logorrhea, increased drive and increased social contact). And a second factor composed of the remaining 'non-manic' symptoms with loadings over 0.60 in apperception, concentration, inhibited and retarded thinking, blocking, incoherence, depersonalization, perplexity, blunted affect, lack of drive, inhibition of drive and mutism. Paranoid-hallucinatory symptoms loaded in this factor but with lower weights (between 0.40 and 0.50). Depressive symptoms loaded in both factors but slightly higher in the second factor. *At the third level*, the factor solution retained the mania dimension plus two factors originated from the non-manic factor of the previous level: a dimension comprising depressive symptoms and a mixed factor made up of 'psychomotor poverty' and 'paranoid-hallucination' symptoms. The pattern of salient loadings of *the four-factor solution* suggests the following labels for the factors: mania, psychomotor-poverty, paranoid-hallucinations and depression factors. *In the next step*, a dimension comprising 'schneiderian' symptoms merged from the paranoid-hallucinations factor of the previous level. The remaining four dimensions were also retained in the five-factor solution. *At the sixth level,* a dimension made up of insight and dysphoria symptoms arose from the mania dimension. *Next,* the depression dimension was divided into a pure depressive dimension and a dimension comprising 'anxiety' and 'guilt feelings', which we labelled 'anxiety-guilt' dimension. *In the following analysis*, the 'Schneiderian' dimension was split in two factors, one comprising auditory hallucinations and 'thought influence' experiences, for which we retain the label of Schneiderian factor, and another one composed of 'non-auditory' hallucinations and other influence phenomena. We labelled the latter as 'other disorders of ego integrity' factor, which was made up of 'other delusions' item (religious and infrequent delusions), 'other feelings of alien influence' (which comprised feelings of body and behavior influence), illusions, non-verbal hallucinations, derealization and depersonalization items. *At the ninth step*, a 'derealization-depersonalization' dimension arose from 'other disorders of ego integrity' dimension. *Finally*, a dimension composed of disorganization and dysphoria items from the manic and insight dimensions, respectively, merged as a separate dimension leaving also a 'pure' insight dimension. Disorganization items comprised thought disorders, such as (rumination, tangentiality, incoherence), and inappropriate affect (parathymia).

The final solution of 10 factors accounted for 66 % of the total variance. This final solution attained good simple structure, as indicated by the small number of items with complex loadings (13 out of 70 items loaded in two factors and only 2 out of 70 items loaded in three factors), and no 'hyperplane' items were found (items failing to have a salient loading in one factor) (Table 2).

Internal consistency of factors demonstrated to be good to excellent with Cronbach's alphas ranging from 0.66 to 0.90 (Table 2). This is important because one needs to have adequate measures of a given content domain in order to determine whether the domain corresponds to a distinct factor.

Factor models with more than 10 factors were non-feasible due to notable shortcomings, such as lack of interpretability of factor structures, as a result of either excessive simplification of dimensions or reduplication of existing dimensions.

Independence among psychopathological dimensions of the final 10-dimension model was achieved since there were no significant correlation coefficients, except for a weak association between mania and disorganization dimensions ($r = 0.31$, $p \leq 0.02$).

Discussion

The findings of the present study revealed that psychopathological symptoms of first-episode psychosis consisted of multiple dimensions, which are structured in a hierarchical manner. The 'psychosis construct' evolves from a common trunk indicating a global psychopathological index of severity at the first level (or n = 1 dimensional tree) through consecutive branches (from n = 2 to n = 10 dimensional arborizations) representative of psychopathological domains in psychosis to a hypothetical symptom level at an item level. This 'hierarchical organization' of dimensions allowed the integration of 'classic psychopathological models' of psychosis, such as archetypal Kraepelinian dichotomy between manic-depressive and schizophrenic psychoses, Bleulerian loss of associations (or its 'modern' label as 'disorganization dimension') and Schneiderian first-rank symptoms.

Moreover, contemporary psychopathological models, such as Crow's type I and type II schizophrenias (Crow 1980) could be ascertained in our hierarchical system within the non-manic branches (paranoid-hallucinatory-psychomotor-poverty-depression dimensions) at the second and third level of hierarchy. The three-syndromic models were also present with slight variations. While the negative and the disorganization dimensions showed face validity with respective dimensions in three-syndromic model, the 'reality distortion' dimension could be divided into three: paranoid, Schneiderian and 'other disorders of ego integrity' dimensions. Other authors have similarly reported two or more dimensions within the 'reality distortion' dimension to accomplish such a wide range of symptoms (Gur et al. 1994; Vázquez-Barquero et al. 1996; Peralta et al.

Table 2 Factor loadings and factor pattern correlation matrix of 10-dimension model of psychopathological symptoms

Symptoms	Prevalence %	Mania Factor	Psychomotor poverty	Paranoid Factor	Depression Factor	Other delusions Factor	Insight Factor	Anxiety/Guilt Factor	Schneiderian Factor	Depersonalization Derealization	Disorganization Factor
Self orientation	34									0.53	
Apperception	56.4		0.65								
Concentration	71.3		0.61								
Memorization	37.2		0.61								
Inhibited thinking	37.2		0.76								
Retarded thinking	41.5		0.79		0.40						
Circumstantial	38.3	0.44									−0.52
Restricted thinking	43.6		0.68								
Perseveration	51.1										−0.44
Rumination	39.4	0.50									−0.62
Pressured thinking	36.6	0.89									
Flight of ideas	12.8	0.82									
Tangential	24.5	0.51									−0.59
Blocking	35.1		0.73								
Incoherence	56.4	0.44	0.42								−0.46
Suspiciousness	94.7			0.52							
Delusional mood	95.7			0.65							
Delusional perception	81.9			0.60		−0.42					
Sudden delusional ideas	80.9			0.59							
Delusional ideas	96.8			0.67							
Systematized delusions	87.2			0.51							
Delusional dynamics	98.9			0.78							
Delusions of reference	85.1			0.66							
Delusions of persecution	84			0.61							
Delusions of guilt	20.2							−0.80			
Delusions of grandeur	31.9	0.78									
Other delusions	22.3					−0.72					
Illusions	46.8					−0.47				0.44	
Verbal hallucinations	56.4			0.40					0.55		
Other auditory hallucinations	30.9					−0.42				0.42	
Bodily hallucinations	22.3					−0.74					
Depersonalization	55.3									0.86	
Derealization	56.4									0.82	
Thought broadcasting	38.3								0.86		
Thought withdrawal	20.2								0.80		
Thought insertion	41.5								0.82		
Other feelings of alien influence	38.3					−0.70			0.45		
Perplexity	48.9		0.51								

Table 2 *continued*

Symptoms	Prevalence %	Mania Factor	Psychomotor poverty	Paranoid Factor	Depression Factor	Other delusions Factor	Insight Factor	Anxiety/Guilt Factor	Schneiderian Factor	Depersonalization Derealization	Disorganization Factor
Blunted affect	56.4	–0.51	0.72		0.69						
Feeling of loss feeling	25.5				0.67						
Loss of vitality	48.9				0.78					0.42	
Depressed mood	39.4				0.84						
Hopelessness	19.1										
Anxiety	88.3			0.40				–0.50			
Euphoria	31.9	0.92									
Dysphoria	71.3	0.52				0.44					
Irritability	91.5										–0.61
Inner restlessness	81.9										–0.48
Complaintiveness	16				0.53						
Feelings of inadequacy	17				0.72						
Exaggerated self-confidence	31.9	0.80									
Feelings of guilt	20.2							–0.76			
Ambivalence	22.3										–0.60
Parathymia	36.2					–0.57					
Affective lability	37.2	0.47						–0.52			
Affective incontinence	24.5	0.56									
Affective rigidity	23.7		0.53								
Lack of drive	44.7	–0.41	0.81								
Inhibition of drive	28.7		0.62								
Increased drive	38.3	0.82									
Motor restlessness	70.2	0.68									–0.49
Mutism	22.6		0.52								
Logorrhea	31.9	0.90									
Reduced social contact	74.5	–0.69	0.48								
Excessive social contact	23.4	0.79									
Aggressiveness	54.3										–0.68
Suicidal tendencies	22.3				0.41						
Lack of feeling of illness	88.3						0.81				
Lack of insight	94.7						0.82				
Refusal of treatment	60.6						0.71				
% Variance accounted for	**66.0**	**18.7**	**11.6**	**8.5**	**6.0**	**5.2**	**3.9**	**2.54**	**2.24**	**1.96**	**1.74**
RELIABILITY (α)		**0.84**	**0.90**	**0.82**	**0.80**	**0.71**	**0.82**	**0.66**	**0.82**	**0.71**	**0.81**

* Only item loadings ≥ 0.40 are shown

1998). Kay's five-dimension model resulting from factor analysis of PANSS scale was also present within our model, although its five characteristic dimensions (negative, positive, disorganized thought, excited and anxiety-depression dimensions) merged at different levels of arborification.

The integration of all models within our hierarchical and dimensional structure might help us to understand the clinical 'schizophrenia puzzle', such as it was exemplified in the classic old Indian story called "The blind people and the elephant". In that story, an elephant was presented to a group of blind people and they were allowed to feel and examine it. Different descriptions of the same 'object' were all true but only partially. Following our model it is possible to integrate 'partial knowledge' of each one of the 'blind psychiatric models' within a logic and graphic algorithm (Fig. 1).

There were certain differences between our two studies carried out with a similar statistical strategy (Cuesta and Peralta 2001 and the present study). These differences were probably related to the phase of illness at which the two samples were assessed (chronic versus first-episode phases, respectively). However, a great overlap between both studies in symptoms selected on the basis of its prevalence was found. Sixty-one out of the 70 AMDP symptoms were the same as in our previous study and only 3 symptoms were absent in the present study and entered in our former analysis (visual hallucinations, parakinesis and lack of self care). Seven out of the ten dimensions of final factor solutions represented the same psychopathological constructs in both studies (mania, psychomotor poverty, paranoid, depressive, Schneiderian, insight and disorganization dimensions). Three dimensions were exclusive of the first-episode sample: 'Anxiety-guilt', 'Depersonalization-derealization' and 'Other ego integrity disorders'. Remarkably these dimensions are very close to those symptoms precisely described by Conrad for the early stages of psychosis in his classic book (Conrad 1966). In addition, two catatonic dimensions were extracted in our chronic sample but not in the present study. The latter finding is in agreement with studies reporting higher prevalence of motor features associated with older patients (Peralta and Cuesta 2001b), and consequently with longer time of evolution. There were also certain differences in hierarchical derivation among psychopathological dimensions since, for instance, the disorganization dimension merged from the manic branches in the present study and from the 'non-affective' and subsequently from the 'psychomotor-poverty' branches in our previous study. As another example, the depressive dimension was clearly derived from the affective dimension in our earlier study, while it was shared by the two main branches in the present one.

We have followed the recommendations of McGorry et al. (1998) in focussing on psychosis rather than on schizophrenia since no definitive validation of any psychopathological entity has been demonstrated. These authors carried out an exploratory factor analysis of dimensional structure on a large sample of first episode psychosis using a different psychopathological instrument. However, both their final 4 and 6 factor solutions can be integrated within our model. Unfortunately they opted for a conventional factor analysis based upon the Scree test to determine the number of factors to extract, which is appropriate from statistical grounds but limited on conceptual and empirical support when it is applied alone. Our results are in greater agreement with those of van Os et al. (1996) who studied a cohort of relatively recent onset subjects with functional psychosis and found a dimensional pattern comprising 7 dimensions. However, not only our results are in agreement with recent studies but also they showed a marked similarity with those found by Lorr et al. (1961) in their seminal contribution more than 40 years ago since 7 out of their 10-syndrome model dimensions are very similar to our final solution both in first-episode (present study) and chronic functional psychosis (Cuesta and Peralta 2001).

Finally, the replication of our model in a first-episode sample added extra value to our results since it avoids bias from chronicity, institutionalization and 'long-term medication' effects. In addition, we re-analyzed our data in the drug-naïve subset of patients (n = 69, 73.4 %) and a very similar hierarchical and multidimensional structure was found. Taken together, longitudinal stability of our hierarchical structure may be inferred from similarity between the two studies comprising 'first-episode' and 'chronic' psychoses.

To our knowledge, a hierarchical analysis of psychotic symptoms was only applied to establish a schizophrenia diagnosis, on the basis of classic criteria (Schneider 1959; Jasper 1963) as well as on current nosotaxias (DSM or ICD classifications), or to identify 'classes of personal illness' (Foulds and Bedford 1975). Common to the above strategies was the classification of patients in categories. On the contrary, our hierarchical model was not developed to classify but to quantify psychopathological dimensions, to show structural interdependence across dimensions and to enable psychiatrists and researchers to set specific levels of hierarchical complexity to undertake their research.

The present 'hierarchical approach' to examine psychopathological dimensions provides a new paradigm to use in biological research in psychosis. It allows for an empirically driven approach by targeting different 'phenotypes' from symptomatological to dimensional levels of complexity within the same data. In this respect, 'candidate phenotypes' can be explored in the same way that geneticists use their 'candidate gene' approach (Leboyer et al. 1998). Setting the level of complexity of phenotype will depend on the hypothesis to test. If researchers are looking for common physiopathological mechanisms setting the level at a low number of dimensions, or at a lower 'branch level', might be required. Likewise, searching for specific mechanisms of any psychopathological dimension will target our phenotype at a high n-dimensional level or 'leaves' level.

In addition, the multidimensional nature of our

model may be devised in a quantitative manner since each of the ten dimensions is reduced to a level of intensity, which allows the estimation of not only cross-sectional but also follow-up assessments of patients. Dimensional phenotypes are useful for practice since one can demonstrate efficacy of treatments on particular dimensions (van Os et al. 1999).

There are limitations to our study that are inherent to the cross-sectional assessment, which did not account for the variability over time of psychopathological structure. Notwithstanding, the striking similarity between our previous study carried out in a large sample of chronic patients suggested that psychopathological dimensions are relatively stable across time. Moreover, two other statistical limitations reducing the strength of our paper should be acknowledged. First, the relatively small size of the sample regarding the number of symptoms to be analyzed. Second, polychoric instead of Pearson correlations should have been employed providing that ordinal data were analyzed.

Finally, an integration of how psychiatrists assess psychopathological symptoms based upon factorial decomposition of an exhaustive psychopathological exploration is presented. Whether these correlate to the classic psychometric postulates (Cattell 1978), assuming that our ten different factors correspond to different pathophysiological mechanisms, deserves future investigation. It is necessary to keep in mind that many efforts to reduce psychopathological syndromes to localizationist interpretations have been unsuccessful since it has still not been demonstrated that there is neurobiological evidence to any psychiatric entity, syndrome or symptom (McGorry 1991). However, focussing on underlying common factors in the manifestations of psychopathological symptoms seems not only to be a promising approach (Vollebergh et al. 2001; Krueger 1999), but also a complementary way of looking at the same data (Goldberg 2000). We hope that our work will stimulate new ways of defining phenotypes in neurobiological research of psychosis.

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