

# The Bern Psychopathology Scale for the Assessment of System-Specific Psychotic Symptoms

Werner Strik<sup>a</sup> Alexander Wopfner<sup>a</sup> Helge Horn<sup>a</sup> Philipp Koschorke<sup>a</sup>  
Nadja Razavi<sup>a</sup> Sebastian Walther<sup>a</sup> Gustav Wirtz<sup>b</sup>

<sup>a</sup>University Hospital of Psychiatry, University of Bern, Bern, Switzerland; <sup>b</sup>MediClin Klinik an der Lindenhöhe, Offenburg, Germany

## Key Words

Affectivity · Brain systems · Cognitive neuropsychiatry ·  
Language · Motor system · Psychopathology ·  
Schizophrenia

## Abstract

The translation from psychiatric core symptoms to brain functions and vice versa is a largely unresolved issue. In particular, the search for disorders of single brain regions explaining classical symptoms has not yielded the expected results. Based on the assumption that the psychopathology of psychosis is related to a functional imbalance of higher-order brain systems, the authors focused on three specific candidate brain circuitries, namely the language, and limbic and motor systems. These domains are of particular interest for understanding the disastrous communication breakdown during psychotic disorders. Core symptoms of psychosis were mapped on these domains by shaping their definitions in order to match the related brain functions. The resulting psychopathological assessment scale was tested for interrater reliability and internal consistency in a group of 168 psychotic patients. The items of the scale were reliable and a principal component analysis (PCA) was best explained by a solution resembling the three candidate systems. Based on the results, the scale was optimized as an instrument to identify patient subgroups characterized by a prevailing

dysfunction of one or more of these systems. In conclusion, the scale is apt to distinguish symptom domains related to the activity of defined brain systems. PCA showed a certain degree of independence of the system-specific symptom clusters within the patient group, indicating relative subgroups of psychosis. The scale is understood as a research instrument to investigate psychoses based on a system-oriented approach. Possible immediate advantages in the clinical application of the understanding of psychoses related to system-specific symptom domains are also discussed.

Copyright © 2010 S. Karger AG, Basel

## Introduction

A new formulation of schizophrenic psychopathology has been repeatedly claimed in order to match the typical psychotic phenomena to known brain functions [1–4]. Despite the broad expert consensus regarding the biological etiology of schizophrenia, there remains a remarkable lack of pathognomonic biological findings for schizophrenia in general, independent of the research approach applied. Further, the high variability in results in biological research still challenges the entity of schizo-

W.S. and A.W. contributed equally to the study.

phrenia in terms of a medical diagnosis with a unique etiopathogenesis [2, 5, 6]. Therefore, there is an urgent need for matching the typical psychotic phenomena to brain functions and vice versa in order to bridge the gap between clinical symptoms and their possible pathophysiology at the level of brain systems.

To increase homogeneity in patient subgroups in terms of their natural boundaries, a number of studies attempted to extract dimensions from psychopathological symptom scales with statistical methods [7–10]. In principle, this is an appealing approach. However, it should be borne in mind that the traditional psychiatric symptoms are not hard facts like physical measures. Instead, their definitions have been historically evolved and implicitly determine the coverage and resolution according to the theoretical framework of their origins [10]. To our knowledge, until now, dimensions derived statistically from symptom scales could not be convincingly linked to alterations in specific neuronal systems. Accordingly, we suggest that existing psychopathological scales, referring to traditional symptom definitions rather than to known functional circuitries, produce noisy measurements regarding their possible relationship with dysfunctions in specific brain systems.

To formulate psychopathology allowing the translation of symptoms or symptom groups to brain functions, it is reasonable to identify candidate neuronal systems which are disturbed in schizophrenia and, on the other hand, can be linked to the phenomenology of psychosis at a descriptive level. In search for the pathophysiology of auditory hallucinations and formal thought disorders in schizophrenia, this approach has been followed consequently by our group, linking clinical symptoms to the structure and function of the language system in empirical studies [11–14]. We underscore the fact that in both hallucinations and formal thought disorders the translation from symptoms to system physiology was successful when the groups were stratified according to the actual presence of the language-related symptoms (acoustic verbal hallucinations or incoherence of speech, respectively).

In addition to these typical schizophrenic symptoms, convincingly linked to the language system, we considered two major psychopathological domains which can be descriptively related to candidate systems. One of them is motor behavior, which is disturbed in various ways in schizophrenia. In catatonia, in particular, motor system dysfunctions have been reported repeatedly [15–18]. The third domain is affectivity, which is often intensely involved in both positive and negative schizo-

phrenia syndromes. Several behavioral and autonomic symptoms of this domain can be ascribed to the activity of the limbic system, which has been frequently associated with the pathogenesis of schizophrenia in general [19–26].

Although there is good evidence that several other brain systems at the basis of cognitive and emotional functions, such as working memory [27] or theory of mind [28], are also involved in schizophrenia spectrum disorders, their relationship with specific schizophrenic symptoms is less evident and cannot be established at a descriptive level without further theoretical assumptions.

Based on these observations, we developed a clinical rating scale defining psychotic symptoms, which helps to distinguish the three psychopathological domains (language, affectivity and motor behavior). The scale is intended as a research tool capable of identifying patients with prominent disturbances in one or more of the psychopathological domains, which can be linked with the activity of the above-mentioned candidate brain systems.

The aims of the present work were (i) to introduce a scale based on the idea of specifying psychotic symptoms putatively related to specific brain systems, (ii) to test the interrater reliability of the instrument and (iii) to understand whether the dysfunctions of these candidate systems examined are intraindividually coherent or whether they can emerge independently from each other. We hypothesized that it is possible to create a clinically applicable and reliable instrument which relates psychotic psychopathology to known brain systems, and that a principal component analysis (PCA) would confirm the validity of the scale to distinguish the symptom domains based on a clinical sample of psychotic patients. The validation of our basic assumption that these symptom domains would be related to natural subgroups of psychosis linked to dysfunctioning candidate brain systems was not the scope of this study and will be addressed in future studies.

## Patients and Methods

### *The Development of the Bern Psychopathology Scale*

#### *General Principles*

The Bern Psychopathology Scale (BPS) is regarded as an instrument for empirical research identifying more homogeneous subject groups in the context of a system-specific physiological model of psychosis. It is neither intended as a diagnostic tool in

terms of the ICD-10 or DSM-IV categories nor does it pretend to offer a complete collection of all pathological phenomena observable in psychoses. According to the findings and observations cited in the Introduction, we grouped and shaped classical descriptions of psychopathological phenomena to fit and distinguish three behavioral and symptom domains: language, affectivity and motor behavior.

It appeared reasonable to define the system-specific symptoms according to their positive or negative quality in terms of hypo-/hyperactivity of the respective psychic function. This allows testing hypotheses about the longitudinal bipolarity of the single symptoms, its temporal dynamics and the excitation state of the respective brain circuitries.

#### *Development and Basic Structure of the Scale*

The symptoms and the basic structure of the rating scale were developed by W.S. based on traditional psychopathological symptoms (AMDP system), on symptoms used by Karl Leonhard and by personal observations. Only symptoms which could be clearly and descriptively attributed to the previously defined domains were taken into account. The scale and its operational criteria were then discussed to improve its consistency and intelligibility by the Board of Clinical Psychiatrists of the University Hospital of Psychiatry in Bern.

The definitions of the symptoms are operational, i.e. with explicitly defined criteria and a description of the assessment procedure. The items were grouped according to the targeted theory into three subscales for the domains language, affectivity and motor behavior. The symptom descriptions as such are atheoretical, i.e. their operational definitions refer exclusively to behavioral observations, subjectively reported phenomena and commonly accepted psychological constructs, but not idiosyncratically to the targeted theory. The items cover detailed domain-related symptoms, while more abstract or summary definitions were excluded. For instance, instead of relating to the concept of psychomotor retardation or agitation, speed of thought and speed of movements are observed separately reflecting thought or motor behavior. Each of the two speed functions is further rated as increased, normal or decreased. The assessment is strictly cross-sectional, i.e. the standard time span to be considered when rating patients is 1 h. This allows measuring the temporal dynamics of the behavioral phenomena even in rapidly changing psychopathology. To include the neurophysiologic principle of inhibition/disinhibition, the symptoms were grouped into mutually exclusive pairs of behavioral hypo-/hyperactivity. Further, the symptoms were explicitly tagged as quantitative or qualitative deviations from normal, in order to distinguish those phenomena which can be defined as an increase or decrease in quantity or speed of normal behavior from those which imply disorders of the sequence of actions.

Only phenomena which could be attributed unambiguously to one of the three domains were included. The classical symptom of delusional ideas was the most critical topic in this endeavor. It is a complex phenomenon of erroneous judgment with many possible origins not directly attributable to one of the three domains. However, there is some evidence that alterations in the limbic system are involved in the genesis of delusions [23, 29], which is supported by clinical observations and classical descriptions; they show that delusions often include an important affective content of existential threat or self-elevation. Consequently, we included

those delusions and hallucinations which contain an unambiguous emotional content, e.g. delusions of danger, threat, unrealistic power or insights, in the affective domain as an indirect sign of the accompanying affective state, but without any a priori assumptions about the direction of causality. This attribution, however, may be considered as a more uncertain category of items; we therefore called this group of affective hallucinatory and delusional contents *indirect signs* within the affective domain. The experience of abnormal, i.e. excessive and sustained, happiness or elevation is observed in acute psychotic states and it fits well in the opposite-pair organization of our scale as the opposite of psychotic anxiety.

#### *Scaling of the BPS*

Originally, the scale consisted of 50 qualitative items. In addition, three quantitative dimensions rated the severity of the pathology in each of the three domains.

Each item consists of one behavioral feature and two mutually exclusive alterations in this feature. For example, the previously mentioned item A23 (Delusions) measures two thematically different kinds of delusions according to the most likely accompanying emotions of paranoia or exaltation. An affectively neutral delusion is not rated.

The subscale for language contained 15 items, each describing an inhibited and disinhibited occurrence of one specific language feature [e.g. the item S5 (Response Latency) can be rated as 'reduced', 'increased' or normal]. Six of them referred to quantitative, 5 to qualitative abnormalities. In addition, 4 items were defined to assess subjective experiences in verbal thoughts.

The affective subscale contained 26 items. Again each item contains two pathological versions of the same behavior as exemplarily described above for delusions. The affective subscale included 11 behavioral and autonomous signs, 10 indirect signs, and 5 items related to the subjective experience.

The subscale for motor behavior consisted of 10 items describing inhibited and disinhibited motor behaviors; e.g. item M4 (Variability of Movements), the specifications 'playful, versatile', 'monotonous', or 'normal' can be rated. Five items of this subscale describe quantitative aspects, 3 items rate qualitative aspects and 2 items are relative to subjective perceptions.

The pathologically inhibited, disinhibited or normal variations of a given behavioral feature are assessed on a three-point scale: -1 = pathologically inhibited; 0 = normal; 1 = pathologically disinhibited; in the affective domain, -1 relates to the signs of anxiety and +1 to the signs of elation.

A global assessment of the severity of disturbance of each domain is rated on a seven-point scale: -3 = severely inhibited or anxious; -2 = moderately inhibited or anxious; -1 = mildly inhibited or anxious; 0 = normal; 1 = mildly disinhibited/elated; 2 = moderately disinhibited/elated; 3 = severely disinhibited/elated. The global assessment does not result from the sum of the items measured within each domain, but is an overall estimation of the severity of the behavioral disorder within each domain. This additional assessment appeared necessary to indicate the overall clinical severity of the disorder since the items of the scale are rated in a purely ordinal way (present/absent), and do not contain information about the severity of each symptom. The sum score of the symptoms reflects therefore only the number of symptoms but not the severity of the disorder. For example, a single but severe symptom like *cereia flexibilitas* would result in a sum score of

**Table 1.** Clinical characteristics of the sample

ICD-10 code	Diagnosis	Patients		Duration of illness, years		Hospitalizations (mean)	
		n	%	mean	SD	frequency n	max. dura- tion, months
F20.0	Paranoid schizophrenia	75	44.6	6.5	6.4	5.0	16.8
F20.1	Hebephrenic schizophrenia	17	10.1	6.9	6.3	5.8	27.4
F20.2	Catatonic schizophrenia	10	6.0	4.7	5.5	5.0	17.4
F20.3	Undifferentiated schizophrenia	5	3.0	8.4	8.1	7.4	13.8
F20.5	Residual schizophrenia	4	2.4	9.7	7.8	8.0	29.0
F21	Schizotypal disorder	1	0.6	3.8		1.0	2.0
F22.0	Delusional disorder	3	1.8	2.3	3.7	2.0	5.7
F23	Acute and transient psychotic disorders	41	24.4	2.6	4.7	2.5	5.5
F25	Schizoaffective disorder, manic type	8	4.8	9.0	7.9	6.1	12.2
Other		4	2.4	3.2	2.9	4.3	19.3

Diagnoses and subtypes were according to ICD-10 (n = 168). Duration of illness, mean frequency of hospitalizations and mean duration of the longest hospitalization in patient history per diagnostic category are shown.

1, while a mild clinical picture presenting 2 or more symptoms would result in a higher score. The original scale used in this study can be found online at [www.puk.unibe.ch/BPS](http://www.puk.unibe.ch/BPS).

### Validation Procedure

#### Subjects

Inpatients of the University Hospital of Psychiatry (n = 168) were assessed using the BPS. The diagnoses were given by board-certified psychiatrists after thorough interviews and review of the available case files according to ICD-10 criteria. Ninety patients (55.9%) were female. Table 1 shows the diagnoses according to ICD-10. Tables 2 and 3 reveal sociodemographic characteristics of the patient group. Inclusion criteria were the presence of a psychotic episode or the diagnosis of schizophrenia, and age between 18 and 65 years. Patients with organic brain disorders and current drug abuse other than nicotine were excluded. All patients provided written informed consent. The study was consistent with the standards of the Declaration of Helsinki.

A manualized semi-structured interview was applied to allow standardized assessment of the necessary information to rate the items of the BPS. Each patient was interviewed for 30–60 min on the ward.

The first 20 interviews were recorded on video. The videos were rated with the BPS by three raters trained in the use of the BPS.

#### Statistics

Distribution, standard deviation, skewness and kurtosis of each item were computed. The results are shown in table 4. The items A9 (Sudor) and A10 (Reflexes) could not be accounted for because of assessment difficulties.

On the basis of the data collected from videos recorded from 20 patients by three different raters, the interrater reliability was

**Table 2.** Highest completed education (n = 168)

Highest completed education	n	%
No school completed	6	3.6
Obligatory school (8–9 years)	33	19.6
Apprenticeship	71	42.2
College of higher education	6	3.6
University entrance diploma	10	6.0
University	10	6.0
Unknown	32	14.9

**Table 3.** Sex (n = 168)

Sex	n	%
Male	78	44.1
Female	90	55.9

computed with Kendall's W [30], which allows a non-parametric analysis of >2 interviewers. It was not possible to compute the interrater reliability of items A6–A10 and A12, since they could not be assessed from the videos.

Items with an insufficient interrater reliability were excluded from further analyses. A univariate skewness >2 and kurtosis >7 have been shown to cause significant problems in factor analyses

**Table 4.** Item statistics

Descriptive statistics	n	-	0	+	SD	Skewness	Kurtosis	Kendall's W
GA_L Global Score Language	168	77	42	49	1.40	0.22	-0.54	0.84
GA_A Global Score Affectivity	168	104	28	36	1.33	0.56	-0.04	0.89
GA_M Global Score Motor behavior	168	59	82	27	0.91	0.00	0.09	0.93
L1 Spontaneity of speech	168	52	89	27	0.67	0.18	-0.78	0.71
L2 Spontaneous intermissions of speech pauses	168	37	116	15	0.54	-0.09	0.22	0.79
L3 Speech pace	168	28	124	16	0.51	-0.12	0.84	0.80
L4 Ideas	168	46	93	29	0.66	0.11	-0.71	0.83
L5 Response latency	168	47	106	15	0.58	0.04	-0.26	0.84
L6 Reaction to interlocutor	168	44	95	29	0.66	0.09	-0.66	0.74
L7 Person identification	165	6	137	22	0.40	0.78	2.62	1.00
L8 Coherence of speech	168	30	101	37	0.63	-0.03	-0.47	0.77
L9 Interruptions	168	8	149	11	0.34	0.32	6.02	0.54
L10 Naming	168	11	141	16	0.40	0.24	3.32	0.81
L11 Apprehension of meaning	168	23	122	23	0.53	0.00	0.71	0.77
L12 Stream of thoughts	167	36	98	33	0.64	0.02	-0.56	0.90
L13 Quantity of thoughts	167	42	75	50	0.74	-0.08	-1.17	0.54
L14 Clarity of thoughts	168	26	105	37	0.61	-0.03	-0.30	0.90
L15 Drive to speak	168	50	95	23	0.64	0.16	-0.61	0.93
A1 Emotional sensibility	168	23	124	21	0.51	-0.02	0.88	0.75
A2 Posture	168	35	123	10	0.50	-0.30	0.59	0.74
A3 Movements	168	20	143	5	0.38	-0.97	3.38	0.82
A4 Gesture	168	9	141	18	0.40	0.45	3.23	0.44
A5 Mimic	168	50	103	15	0.59	0.08	-0.36	0.89
A6 Respiration	167	12	151	4	0.31	-1.21	7.31	
A7 Skin color	156	16	134	6	0.37	-0.75	3.99	
A8 Eyes	157	10	130	17	0.41	0.31	2.88	
A9 Sudor	85	3	75	7	0.34	0.80	5.68	
A10 Reflexes	99	10	83	6	0.40	-0.33	3.35	
A11 Prosody	168	37	116	15	0.54	-0.09	0.22	0.93
A12 Muscle tone	161	17	143	1	0.32	-2.11	4.75	
A13 Emotion	168	51	98	19	0.62	0.15	-0.50	0.74
A14 Worry	168	43	112	13	0.55	-0.07	-0.03	0.88
A15 Calmness	168	74	76	18	0.66	0.49	-0.72	0.89
A16 Tension	168	76	79	13	0.63	0.48	-0.64	0.80
A17 Well-being	168	58	104	6	0.54	-0.10	-0.66	0.83
A18 Bodily sensation	168	28	133	7	0.44	-0.61	1.51	0.64
A19 Trust	168	39	113	16	0.56	-0.05	0.06	0.94
A20 Help	168	62	84	22	0.67	0.32	-0.78	0.61
A21 Sureness	168	77	73	18	0.67	0.54	-0.71	0.74
A22 Interpersonal contact	168	66	89	13	0.61	0.30	-0.63	0.93
A23 Delusions	168	48	87	33	0.69	0.12	-0.89	0.97
A24 Hallucinations	168	27	128	13	0.48	-0.23	1.19	0.88
A25 Emotional arousal	168	16	141	11	0.40	-0.24	3.32	0.79
A26 Attitude (feeling)	168	6	136	26	0.42	0.74	1.90	0.81
A27 Contact (behavior)	168	6	157	5	0.26	-0.29	12.67	0.65
M1 Quantity of spontaneous movements	168	49	104	15	0.59	0.06	-0.33	0.82
M2 Movement intermissions	168	38	124	6	0.48	-0.51	0.38	0.78
M3 Movement pace	168	33	131	4	0.44	-0.84	0.86	0.81
M4 Variability of movements	168	17	141	10	0.40	-0.35	3.28	0.62
M5 Stimulatability/excitability	168	35	122	11	0.51	-0.25	0.55	0.76
M6 Motion sequence	168	5	161	2	0.20	-1.88	21.34	0.53
M7 Order of movements	168	1	158	9	0.24	2.90	13.05	0.85
M8 Functionality of movements	168	8	150	10	0.33	0.23	6.55	0.35
M9 Drive to move	168	45	93	30	0.66	0.10	-0.72	0.83
M10 Want to move	168	47	83	38	0.71	0.08	-1.01	0.82

L(x) = Item (x) of the language domain; A(x) = item (x) of the affectivity domain; M(x) = item (x) of the motor behavior domain; n = number of patients in whom the item was applicable; - = number of ratings on the inhibited pole; 0 = number of ratings on the normal range; + = number of ratings on the disinhibited pole; SD = standard deviation. Kendall's W was computed according to 20 filmed patients.

[31]; therefore, items surpassing these values were excluded from further analyses, with the exception of item M6.

To understand whether the underlying factors within the data assessed actually match the structure of the scale and the underlying concept of three behavioral dimensions, we conducted an exploratory PCA. Because we intended to obtain fairly uncorrelated factors we decided to compute a varimax rotated PCA of the remaining 48 items. To determine whether other procedures would lead to meaningful different results, we conducted a principal factor analysis (PFA). Both procedures were done with varimax as well as oblique rotations. Also, a categorical PCA, which does not assume ordinal data, was computed. Due to low Kaiser-Meyer-Olkin measures ( $<0.5$ ) indicating unacceptable sampling adequacy, 2 further items were excluded. The intended number of three factors was confirmed using scree plot, parallel analysis and maximum average power (MAP) test. Accordingly, a three-factor solution was computed. Twelve items with either very low communalities or with high loadings on two factors were again excluded. The latter criterion was chosen to enhance the discriminative power of the scale and its capacity to distinguish between the behavioral domains. For each of the subscales, the scale reliability, the inter-item correlation and the corrected item-total correlation were calculated. Additionally, correlations between all subtotals and global assessments and between these six quantities and every item were computed.

## Results

Because of problematic skewness ( $>2$ ) and kurtosis ( $>7$ ), the items A6 (Respiration), A12 (muscle tone), A27 (Contact) and M7 (Order of Movements) have been excluded from further investigations. Although M6 (Motion Sequence) also features a high kurtosis (21.337), it has been kept because of its importance regarding its content.

### *Interrater Reliability*

The results for Kendall's  $W$  of 20 patients between three raters for every item is shown in table 4. The items A4 (Gesture) and M8 (Functionality of Movements) were eliminated due to a Kendall's  $W < 0.5$ , indicating an unsatisfactory interrater reliability. The interrater reliability of the remaining items ranged from 0.532 to 0.965. The mean was 0.875. The interrater reliability of all remaining items reached the level of significance ( $p < 0.05$ ).

### *Principal Component Analysis*

Due to low Kaiser-Meyer-Olkin measure ( $<0.5$ ), indicating a high non-explicable variance between two correlating variables, the items S7 (Person Identification) and M7 (Order of Movements) were excluded from further analyses.

The number of three factors was confirmed by the parallel test, the MAP test, the scree plot and Kaiser's criterion. According to Hayton et al. [32], the most accurate of these methods is the parallel test (92% correct solutions) followed by the MAP test (85%) and the scree plot (57%). Whereas the parallel test as well as the scree plot tends to overestimate, the MAP test is prone to underestimate the number of factors. The Kaiser criterion suggested thirteen factors, and the scree plot gave hints for a three- or a six-factor solution; the MAP test as well as the parallel test indicated three factors.

A varimax rotated three-factor solution resulted in a component matrix in which the items were to a high degree ordered in factors matching the structure of the scale with its three domains language, affectivity and motor behavior. However, the initial result was still unsatisfactory. First, because of communalities, which were partly very low due to the predetermination of three factors. Thus, items with very low communalities were excluded step by step. Second, the first and third factors contained several items with equal loadings of two domains (language and motor behavior). Since the main goal of the scale is to identify possible subsyndromes related to language, and affective and motor disorders, a further selection of items was done in order to enhance the discriminative power of the scale. For this purpose, items with high intercorrelations between domains were eliminated. According to these two considerations, 11 further items were excluded from the final factor analysis.

Kaiser-Meyer-Olkin's measure regarding all remaining items resulted in 0.818, and the three factors explained 43.05% of the variance. The component matrix and communalities of all remaining items are shown in table 5.

After the elimination of 19 items, the parallel test, the MAP test and the scree plot (fig. 1) still indicated three factors.

The resulting rotated component matrix is shown in table 5. Each of the three factors consists of items of only a single domain. Still the loadings of several items bridge the second and third factor. Figure 2 shows all remaining items as factors composed of the loadings on the three components which span the vector space.

To check if the results depend on the procedure applied (varimax rotated PCA), we conducted further variance-reducing analyses. An oblique rotated PCA (oblimin,  $\delta = 0$ , and promax,  $\kappa = 0.2$ ) as well as PFA again with different rotation procedures (varimax, oblimin and promax) resulted in the identical factor structure, as was the case with a categorical PCA.

**Table 5.** Rotated component matrix, communalities and corrected item-total correlation

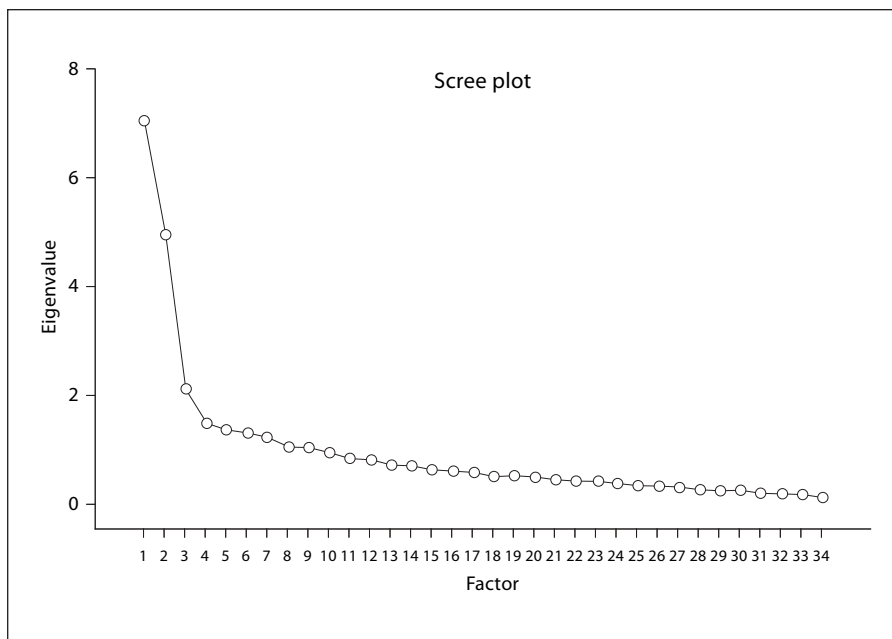
Item	Components			Communalities	Corrected item-total correlation
	1	2	3		
A13 Emotion	0.77			0.61	0.71
A17 Well-being	0.72			0.53	0.67
A19 Trust	0.68			0.46	0.61
A14 Worry	0.68			0.46	0.60
A20 Help	0.67			0.47	0.62
A21 Sureness	0.65			0.44	0.59
A15 Calmness	0.64			0.46	0.57
A23 Delusions	0.64			0.42	0.58
A11 Prosody	0.63			0.44	0.56
A16 Tension	0.62			0.42	0.56
A22 Interpersonal contact	0.60			0.39	0.55
A25 Emotional arousal	0.60			0.38	0.52
A5 Mimic	0.53			0.34	0.48
A24 Hallucinations	0.50		-0.25	0.34	0.43
A26 Attitude (feeling)	0.49			0.27	0.44
A18 Bodily sensations	0.48			0.24	0.42
L5 Response latency		0.73	0.25	0.60	0.70
L11 Apprehension of meaning	0.22	0.70		0.55	0.53
L2 Spontaneous intermissions of speech		0.69	0.35	0.59	0.71
L10 Naming		0.66		0.44	0.44
L3 Speech pace		0.62	0.23	0.44	0.55
L4 Ideas		0.62	0.33	0.49	0.61
L1 Spontaneity of speech		0.60	0.50	0.62	0.68
L8 Coherence	0.22	0.58		0.41	0.50
L15 Drive to speak		0.53	0.36	0.41	0.55
L9 Interruptions		0.52		0.28	0.39
M1 Quantity of spontaneous movements		0.31	0.74	0.64	0.62
M2 Movement intermissions			0.73	0.56	0.49
M5 Stimulatability/excitability		0.36	0.65	0.56	0.60
M4 Variability of movements			0.59	0.35	0.38
M10 Want to move			0.47	0.26	0.41
M9 Drive to move			0.43	0.23	0.45
M6 Motion sequence			0.36	0.13	0.22

L(x) = Item (x) of the language domain; A(x) = item (x) of the affectivity domain; M(x) = item (x) of the motor behavior domain. Component loadings of remaining items in 3 components resulting from varimax rotated PCA; p values <0.2 are omitted. Communalities resulting from PCA with predefined 3-component structure. Corrected item-total correlations regarding the subscales according to the components.

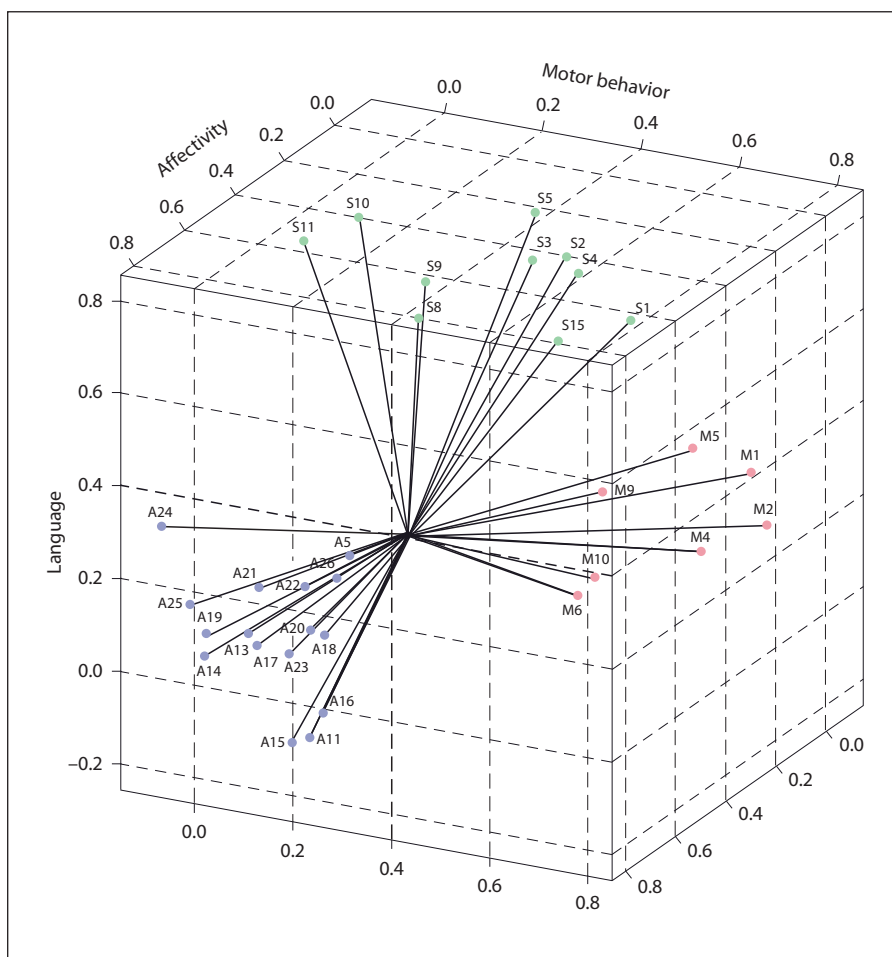
### Subscales

The remaining items of each factor are summarized to three subscales of language, affectivity and motor behavior. The sum of all items within one subscale is further defined according to the subtotal. Cronbach's  $\alpha$  of the first subscale containing 16 affective items was 0.894. The deletion of none of the items led to a further increase in Cronbach's  $\alpha$ . In the second subscale containing 10 items regarding speech behavior, Cronbach's  $\alpha$  was 0.857. Again, the deletion of none of the items would lead to an

increase in Cronbach's  $\alpha$ . The third subscale contained 7 motor-behavioral items. Cronbach's  $\alpha$  was 0.731. Deletion of item M6 resulted in an increase in Cronbach's  $\alpha$  to 0.745, but we decided to keep the item in the final version due to its importance regarding its content. Corrected item-total correlations of every item according to the respective subscale were conducted and are given in table 5. To examine the relationships between the three factors, an interscale correlation was computed (table 6). To gain a better understanding of the relationship between



**Fig. 1.** Scree plot of PCA of the BPS factors (see online suppl. material).



**Fig. 2.** Component plot in rotated space: vectors representing the remaining 33 items in the three-dimensional factor space. Each dimension represents one of the resulting components interpreted as Language, Affectivity and Motor behavior. The item loadings in each component account for components of each vector. The lines meet in the origin. The colors indicate the affiliation to the resulting subscales: Language (green), Affectivity (blue) and Motor behavior (red). The items are clearly separated according to their attribution to the psychopathological domains of the system.



subtotals and the global assessments, correlations were calculated (table 6). To further validate the internal consistency, correlations between items and subtotals and global assessments, respectively, were computed. The results are shown in table 7.

## Discussion

A psychopathological rating scale is presented, intended to map clinical symptoms relevant for psychoses on brain systems known to be involved in the genesis of psychotic symptoms [11–26]. It is neither designed as a diagnostic instrument nor as a complete assessment tool of all possible psychotic phenomena or of all higher brain functions possibly involved in psychoses. Rather, it allows to identify patients who show altered activity in one or more of these systems for scientific purposes, e.g. to study physiological equivalents of the brain or the temporal dynamics of hypo- or hyperactivity in relevant behavioral domains.

The scale was validated and improved in four steps. First, in a broad discussion with the board of residents, consistency, intelligibility and applicability of the single items was addressed and enhanced. Then, the interrater reliability was tested and improved eliminating 2 unreliable items. The average interrater reliability of the items included in the final version was excellent. Third, PCA was performed on the data of 168 psychotic patients to validate one of our basic assumptions, namely that the theoretically driven grouping of the items into domains can be statistically identified as distinct, partially independent factors. Finally, ambiguous items were eliminated to further enhance the domain-specific discriminative power of the scale. After this process, 33 of the original 52 items remained. Internal consistency as well as the item discrimination were found moderate to excellent and thus fulfilled in general the statistical requirements for a psychometric scale. Regarding the internal consistency, one must consider the differences in the numbers of items per subscale. An overestimation especially for the affective subscale with a comparatively high number of items cannot be excluded.

Both, the initial and the final PCA identified three consistent factors within the psychotic symptoms which well matched the domains of language, affectivity and motor behavior. This result did not change if other variance-reducing procedures (oblique rotated PCA or PFA) were applied to the data of the final item pool. This shows that the definitions of the items enable to discriminate

**Table 6.** Pearson correlations of global assessment with subtotals

	L <sub>Sum</sub>	GA_L	A <sub>Sum</sub>	GA_A	M <sub>Sum</sub>	GA_M
L <sub>Sum</sub>	1					
GA_L	0.85**	1				
A <sub>Sum</sub>	0.16*	0.09	1			
GA_A	0.19*	0.14	0.84**	1		
M <sub>Sum</sub>	0.52**	0.42**	0.19*	0.31**	1	
GA_M	0.47**	0.42**	0.16*	0.27**	0.79**	1

\* p < 0.05, \*\* p < 0.01, 2-tailed test.

L<sub>Sum</sub> = Sum of the language subscale; GA\_L = global assessment of language; A<sub>Sum</sub> = sum of the affectivity subscale; GA\_A = global assessment of affectivity; M<sub>Sum</sub> = sum of the motor behavior subscale; GA\_M = global assessment of motor behavior.

between these domains, and that the domains do have a certain degree of independence. In other words, if the abnormalities in terms of inhibition/disinhibition had a similar and common impact on all domains in each patient, the domains would not have been separated as independent factors.

Interestingly, some interscale correlations between the subscales and global assessments of motor behavior and language were found even after several items bridging the respective factors were eliminated; in contrast, intercorrelations of affectivity with the other two domains were weak. Since spoken language can be understood, in formal neurophysiological terms, as a highly specialized motor behavior, there may be some common physiological determinators for both language and motor behavior. Affectivity, on the other hand appears to be more complex with distinct features since it relies, on the sensory side, on several specialized cortical and subcortical modules for the analysis of relevant stimuli, e.g. faces [33], prosody [34], posture [35], archaic visual or acoustic patterns like snakes or looming sounds [36]. On the behavioral side, it essentially relies on subcortical motor patterns for mimic and gestures rather than on voluntary cortical motor behavior, and on autonomous reactions which generate involuntary non-verbal signals of the emotional state like skin perfusion, sweat secretion, the opening of the eyelids, size of the pupil, muscle tonus and prosody.

However, the fact that some items of the original item pool loaded significantly on more than one factor indicates that the behavior described by these items is not

**Table 7.** Pearson correlations of each item with global assessments and subtotals

	GA_L	GA_A	GA_M	L <sub>Sum</sub>	A <sub>Sum</sub>	M <sub>Sum</sub>
L1 Spontaneity of speech	0.67**	0.23**	0.42**	0.77**	0.17*	0.54**
L2 Spontaneous intermissions of speech	0.667**	0.10	0.39**	0.78**	0.11	0.42**
L3 Speech pace	0.52**	0.07	0.36**	0.64**	0.01	0.32**
L4 Ideas	0.64**	0.10	0.32**	0.71**	0.03	0.42**
L5 Response latency	0.71**	0.10	0.32**	0.78**	0.03	0.39**
L6 <i>Reaction to interlocutor</i>	0.60**	0.17*	0.40**	0.68**	0.09	0.49**
L7 <i>Person identification (n = 165)</i>	0.03	-0.01	0.07	0.08	-0.01	0.15
L8 Coherence	0.50**	0.23**	0.34**	0.62**	0.24**	0.30**
L9 Interruptions	0.38**	-0.00	0.15*	0.46**	-0.03	0.16*
L10 Naming	0.43**	0.04	0.22**	0.53**	0.01	0.21**
L11 Apprehension of meaning	0.55**	0.18*	0.14	0.63**	0.23**	0.18*
L12 <i>Stream of thoughts (n = 167)</i>	0.44**	0.06	0.27**	0.39**	0.07	0.33**
L13 <i>Quantity of thoughts (n = 167)</i>	0.42**	-0.02	0.13	0.27**	-0.03	0.19*
L14 <i>Clarity of thoughts</i>	0.23**	-0.06	0.07	0.16*	-0.07	0.06
L15 Drive to speak	0.54**	0.16*	0.35**	0.66**	0.15*	0.40**
A1 <i>Emotional sensibility</i>	0.19*	0.38**	0.20**	0.19*	0.44**	0.26**
A2 <i>Posture</i>	0.12	0.30**	0.06	0.10	0.30**	0.21**
A3 <i>Movements</i>	-0.08	0.33**	-0.05	-0.01	0.32**	-0.01
A4 <i>Gesture</i>	0.11	0.36**	0.04	0.14	0.43**	0.12
A5 <i>Mimic</i>	0.24**	0.41**	0.157*	0.27**	0.55**	0.20*
A6 <i>Respiration (n = 167)</i>	-0.01	0.33**	0.04	0.02	0.38**	0.06
A7 <i>Skin color (n = 156)</i>	0.07	0.29**	0.20*	0.11	0.28**	0.22**
A8 <i>Eyes (n = 157)</i>	0.08	0.36**	0.24**	0.09	0.35**	0.22**
A9 <i>Sudor (n = 85)</i>	0.02	0.29**	0.23*	0.13	0.37**	0.25*
A10 <i>Reflexes (n = 99)</i>	0.09	0.18	0.12	0.09	0.23*	0.08
A11 Prosody	-0.05	0.53**	-0.01	-0.04	0.62**	0.11
A12 <i>Muscle tone (n = 161)</i>	0.09	0.27**	0.36**	0.14	0.27**	0.31**
A13 Emotion	0.13	0.67**	0.13	0.17*	0.77**	0.19*
A14 Worry	0.00	0.53**	0.05	0.03	0.66**	0.05
A15 Calmness	-0.07	0.50**	0.05	-0.04	0.65**	0.05
A16 Tension	-0.01	0.55**	0.06	0.02	0.63**	0.13
A17 Well-being	0.07	0.58**	0.10	0.13	0.72**	0.13
A18 Bodily sensation	-0.03	0.39**	0.11	0.06	0.48**	0.12
A19 Trust	0.01	0.53**	0.04	0.07	0.67**	0.02
A20 Help	0.08	0.61**	0.21**	0.16*	0.69**	0.22**
A21 Sureness	0.10	0.57**	0.18*	0.17*	0.67**	0.15*
A22 Interpersonal contact	0.14	0.51**	0.18*	0.19*	0.63**	0.17*
A23 Delusions	0.03	0.64**	0.17*	0.07	0.66**	0.18*
A24 Hallucinations	-0.01	0.42**	-0.02	0.08	0.50**	-0.04
A25 Emotional arousal	0.06	0.46**	-0.05	0.05	0.57**	-0.01
A26 Attitude (feeling)	0.16*	0.41**	0.13	0.18*	0.50**	0.17*
A27 <i>Contact (behavior)</i>	-0.04	0.24**	0.07	-0.04	0.32**	0.05
M1 Quantity of spontaneous movements	0.39**	0.27**	0.74**	0.49**	0.15	0.77**
M2 Movement intermissions	0.35**	0.18*	0.63**	0.38**	0.07	0.64**
M3 <i>Movement pace</i>	0.32**	0.12	0.57**	0.37**	0.03	0.38**
M4 Variability of movements	0.28**	0.08	0.43**	0.26**	0.04	0.53**
M5 Stimulatability/excitability	0.45**	0.30**	0.69**	0.51**	0.19**	0.73**
M6 Motion sequence	0.01	0.05	0.27**	0.09	0.04	0.30**
M7 <i>Order of movements</i>	-0.09	-0.01	0.00	-0.05	-0.01	-0.06
M8 <i>Functionality of movements</i>	0.07	0.03	0.29**	0.06	0.07	0.18*
M9 Drive to move	0.18*	0.15	0.37**	0.29**	0.09	0.667**
M10 Want to move	0.17*	0.25**	0.36**	0.23**	0.19*	0.65**

\*  $p < 0.05$ , \*\*  $p < 0.01$ , 2-tailed test.  $n = 168$ , if not otherwise indicated. L<sub>Sum</sub> = Sum of the language subscale; GA\_L = global assessment of language; A<sub>Sum</sub> = sum of the affectivity subscale; GA\_A = global assessment of affectivity; M<sub>Sum</sub> = sum of the motor behavior subscale; GA\_M = global assessment of motor behavior. Items not included in the final version of the scale are presented in italics.

specific for one of the domains and therefore does not discriminate; rather, their presence blurs the contours, but might also be interpreted as an indication for additional dimensions.

We interpret the initial results of the PCA as a first internal validation of the concept of domain-specific psychotic syndromes, while its goal was not to conduct an explorative search for new dimensions within psychotic symptoms. Rather, we used the results to identify those items which best fit to our concept of system-specific symptom domains. Therefore, we eliminated the items with high loadings on two domains in order to enhance the discriminative power of the scale and to better confine domain-specific psychotic syndromes.

The 3 items for global assessment correlated significantly with the respective subtotals. Again, there was also a significant correlation between the language and the motor domain. It should be kept in mind, that the subtotal only describes the number of symptoms present, but not their severity. Therefore, we decided to keep the global assessment in spite of the high correlation with the subtotal in order to allow measuring the severity of the present syndrome.

Every single item correlated significantly with the respective global assessment as well as with the subtotal. Several items of the language and the motor-behavioral domain correlated less but still significantly with the other global assessment and subtotal.

A limitation of this study was that the predefined bipolar structure and the resulting organization of each item containing two mutually exclusive behaviors did not allow testing the assumption that both behaviors could occur in the 1-hour time span assessed. Further, some of the bipolar items may not actually represent true contrasts so that the structure of the items might not represent identical dimensions.

Some results are worth to be discussed in detail. We attributed delusions and hallucinations according to their emotional content as indirect signs to affectivity rather than to the cognitive domain of language. This idea was supported by the results of the PCA, since the respective item pairs loaded clearly on the affectivity factor with insignificant intercorrelations to other factors. Furthermore, from 3 items regarding qualitative abnormalities of motor behavior only 1 was included in the final version. The excluded items described typical catatonic signs such as *cerea flexibilitas* or mannerisms. These phenomena, however, were rare and the descriptive bipolar arrangement of the scale was not intelligible for these phenomena. These symptoms will be reinvestigated in future studies.

Within the language domain, 3 of 4 items regarding subjective experiences were excluded due to low communalities. In future studies, it would be worth to further investigate the relationship of disturbed subjective thinking and spoken language.

It must be kept in mind that there are several pathogenetic hypotheses of psychoses which are not addressed by the scale, among them global neurotransmitter dysregulations [37] or other neurophysiological and neuropsychological concepts like subcortical information processing [38], working memory [27, 39] or theory of mind deficits [28, 40]. Our results do not falsify any of them. Rather, they show that there are patients with abnormalities in the behavioral domains of language, affectivity and motor behavior, and that the alterations between these domains can occur with some degree of mutual independence. This encourages comparative functional brain imaging studies between prototypical patients focusing on regions of interest within the here addressed candidate brain systems, and indicates our scale as a suitable instrument to stratify patient groups accordingly for statistical comparisons.

In future studies, we aim at a biological validation of the concept with empirical studies on the activity of the possibly involved brain systems. This approach includes the possibility that not all psychotic patients fit the concept, be it because their symptomatology is not covered sufficiently by the three dimensions of the scale, or because their symptoms involve all domains indistinctively. However, the BPS allows to challenge its assumptions, e.g. whether inhibitory and disinhibitory phenomena are, at a given moment in time, mutually exclusive, and to understand the incidence of specific and mixed syndromes in terms of the domains involved.

During the application of the scale, we made a surprising clinical observation, which, in retrospective, appears even trivial. The three domains at the same time represent the most important domains for interpersonal communication. Therefore, the most disturbed communication channel in single patients can be identified. It was intriguing then to invert the problem and to identify the individually intact communication domains, understanding them as intact resources of the patient. This invited us to a systematic resource-oriented clinical approach to the patients: the intact and not the disturbed domains should be used for communication and conflict management with the patient, e.g. a patient with formal thought disorder has deficits in verbal communication and will be further irritated by verbal reasoning and arguments, unveiling and enhancing the deficit. Conse-

quently, verbal arguments should be avoided and, instead, affectivity as an effective non-verbal channel of communication should be employed and fostered. The inverse is true in patients with psychotic anxiety who tend to misunderstand positive as well as negative affective messages. Often, however, they remain accessible for rational, respectful reasoning, as long as the affective communication remains unambiguous and neutral. Finally, an akinetic, trembling catatonic patient may appear affectively tense and provoke fear and aversive reactions in others, who would force him to move away. Instead, relaxed emotional and verbal communication may be possible. This immediate practical application of the approach appears to us clinically and didactically helpful and will be investigated in empirical studies.

In conclusion, the presented psychopathological scale is reliable and statistically valid to identify subsyndromes

of psychoses, characterized by symptom domains which can be matched to the activity of three important brain systems. Neurophysiological and brain imaging studies are required to understand whether this allows to circumscribe homogeneous patient groups, which can be compared with regard to a specific activation of the specific brain systems. Clinical studies are required to validate the observation of a domain-specific treatment of psychoses.

### Acknowledgments

We express our thanks to the residents of the University Hospital of Psychiatry in Bern for ongoing critical discussions and suggestions for the development of the scale, and Pietro Ballinari for statistical support.

### References

- Andreasen NC: Schizophrenia: the fundamental questions. *Brain Res Brain Res Rev* 2000;31:106–112.
- Honey GD, Bullmore ET: Functional neuroimaging and schizophrenia. *Psychiatry* 2002;1:26–29.
- Allardyce J, Gaebel W, Zielasek J, van Os J: Deconstructing Psychosis Conference February 2006: the validity of schizophrenia and alternative approaches to the classification of psychosis. *Schizophr Bull* 2007;33:863–867.
- Shenton ME, Dickey CC, Frumin M, McCarley RW: A review of MRI findings in schizophrenia. *Schizophr Res* 2001;49:1–52.
- Fujii DE, Ahmed I: Is psychosis a neurobiological syndrome? *Can J Psychiatry* 2004;49:713–718.
- Sallet PC, Elkis H, Alves TM, Oliveira JR, Sassi E, de Castro CC, Busatto GF, Gattaz WF: Rightward cerebral asymmetry in subtypes of schizophrenia according to Leonhard's classification and to DSM-IV: a structural MRI study. *Psychiatry Res* 2003;123:65–79.
- Grube BS, Bilder RM, Goldman RS: Meta-analysis of symptom factors in schizophrenia. *Schizophr Res* 1998;31:113–120.
- Lindenmayer JP, Brown E, Baker RW, Schuh LM, Shao L, Tohen M, Ahmed S, Stauffer VL: An excitement subscale of the Positive and Negative Syndrome Scale. *Schizophr Res* 2004;68:331–337.
- Emsley R, Rabinowitz J, Torremans M: The factor structure for the Positive and Negative Syndrome Scale (PANSS) in recent-onset psychosis. *Schizophr Res* 2003;61:47–57.
- Peralta V, Cuesta MJ: How many and which are the psychopathological dimensions in schizophrenia? Issues influencing their ascertainment. *Schizophr Res* 2001;49:269–285.
- Hubl D, Koenig T, Strik W, Federspiel A, Kreis R, Boesch C, Maier SE, Schroth G, Lovblad K, Dierks T: Pathways that make voices: white matter changes in auditory hallucinations. *Arch Gen Psychiatry* 2004;61:658–668.
- Horn H, Federspiel A, Wirth M, Muller TJ, Wiest R, Wang JJ, Strik W: Structural and metabolic changes in language areas linked to formal thought disorder. *Br J Psychiatry* 2009;194:130–138.
- Strik W, Dierks T, Hubl D, Horn H: Hallucinations, thought disorders, and the language domain in schizophrenia. *Clin EEG Neurosci* 2008;39:91–94.
- van de Veen VG, Formisano E, Roder CH, Prvulovic D, Bittner RA, Dietz MG, Hubl D, Dierks T, Federspiel A, Esposito F, Di Salle F, Jansma B, Goebel R, Linden DE: The spatio-temporal pattern of auditory cortical responses during verbal hallucinations. *Neuroimage* 2005;27:644–655.
- Scheuerecker J, Ufer S, Käpernick M, Wiesmann M, Brückmann H, Kraft E, Seifert D, Koutsouleris N, Möller HJ, Meisenzahl EM: Cerebral network deficits in post-acute catatonic schizophrenic patients measured by fMRI. *J Psychiatr Res* 2009;43:607–614.
- Northoff G, Braus DF, Sartorius A, Khoram-Sefat D, Russ M, Eckert J, Herrig M, Leschinger A, Bogerts B, Henn FA: Reduced activation and altered laterality in two neuroleptic-naive catatonic patients during a motor task in functional MRI. *Psychol Med* 1999;29:997–1002.
- Northoff G, Steinke R, Czyczenka C, Krause R, Ulrich S, Danos P, Kropf D, Otto H, Bogerts B: Decreased density of GABA-A receptors in the left sensorimotor cortex in akinetic catatonia: investigation of in vivo benzodiazepine receptor binding. *J Neurol Neurosurg Psychiatry* 1999;67:445–450.
- Northoff G: Catatonia and neuroleptic malignant syndrome: psychopathology and pathophysiology. *J Neural Transm* 2002;109:1453–1467.
- Radulescu AR, Mujica-Parodi LR: A systems approach to prefrontal-limbic dysregulation in schizophrenia. *Neuropsychobiology* 2008;57:206–216.
- Hoptman MJ, D'Angelo D, Catalano D, Mauro CJ, Shehzad ZE, Kelly AM, Castellanos FX, Javitt DC, Milham MP: Amygdala-frontal functional disconnectivity and aggression in schizophrenia. *Schizophr Bull* 2009, E-pub ahead of print.
- Fujiwara H, Hirao K, Namiki C, Yamada M, Shimizu M, Fukuyama H, Hayashi T, Murai T: Anterior cingulate pathology and social cognition in schizophrenia: a study of gray matter, white matter and sulcal morphometry. *Neuroimage* 2007;36:1236–1245.

- 22 Kumari V, Das M, Taylor PJ, Barkataki I, Andrew C, Sumich A, Williams SC, Ffytche DH: Neural and behavioural responses to threat in men with a history of serious violence and schizophrenia or antisocial personality disorder. *Schizophr Res* 2009;110:47–58.
- 23 Hall J, Whalley HC, McKirdy JW, Romaniuk L, McGonigle D, McIntosh AM, Baig BJ, Gountouna VE, Job DE, Donaldson DI, Sprengelmeyer R, Young AW, Johnstone EC, Lawrie SM: Overactivation of fear systems to neutral faces in schizophrenia. *Biol Psychiatry* 2008;64:70–73.
- 24 Brunet-Gouet E, Decety J: Social brain dysfunctions in schizophrenia: a review of neuroimaging studies. *Psychiatry Res* 2006;148:75–92.
- 25 Lane RD: The neural substrates of affect impairment in schizophrenia. *Am J Psychiatry* 2003;160:1723–1725.
- 26 Williams LM, Das P, Harris AW, Liddell BB, Brammer MJ, Olivieri G, Skerrett D, Phillips ML, David AS, Peduto A, Gordon E: Dysregulation of arousal and amygdala-prefrontal systems in paranoid schizophrenia. *Am J Psychiatry* 2004;161:480–489.
- 27 Meyer-Lindenberg AS, Olsen RK, Kohn PD, Brown T, Egan MF, Weinberger DR, Berman KF: Regionally specific disturbance of dorsolateral prefrontal-hippocampal functional connectivity in schizophrenia. *Arch Gen Psychiatry* 2005;62:379–386.
- 28 Andreasen NC, Calage CA, O’Leary DS: Theory of mind and schizophrenia: a positron emission tomography study of medication-free patients. *Schizophr Bull* 2008;34:708–719.
- 29 Schlagenhaut F, Sterzer P, Schmack K, Ballmaier M, Rapp M, Wrase J, Juckel G, Gallinat J, Heinz A: Reward feedback alterations in unmedicated schizophrenia patients: relevance for delusions. *Biol Psychiatry* 2009;65:1032–1039.
- 30 Siegel S, Castellan NJ Jr: *Nonparametric Statistics for the Behavioral Sciences*, ed 2. New York, McGraw-Hill, 1988.
- 31 Curran PJ, West SG, Finch JF: The robustness of test statistics to nonnormality and specification error in confirmatory factor analysis. *Psychol Methods* 1996;1:16–29.
- 32 Hayton JC, Allen DG, Scarpello V: Factor retention decisions in exploratory factor analysis: a tutorial on parallel analysis. *Organ Res Methods* 2004;7:191–205.
- 33 Vuilleumier P, Pourtois G: Distributed and interactive brain mechanisms during emotion face perception: evidence from functional neuroimaging. *Neuropsychologia* 2007;45:174–194.
- 34 Bach DR, Herdener M, Grandjean D, Sander D, Seifritz E, Strik WK: Altered lateralisation of emotional prosody processing in schizophrenia. *Schizophr Res* 2009;110:180–187.
- 35 Hadjikhani N, de Gelder B: Seeing fearful body expressions activates the fusiform cortex and amygdala. *Curr Biol* 2003;13:2201–2205.
- 36 Seifritz E, Neuhoff JG, Bilecen D, Scheffler K, Mustovic H, Schachinger H, Elefante R, Di Salle F: Neural processing of auditory looming in the human brain. *Curr Biol* 2002;12:2147–2151.
- 37 Kapur S: Psychosis as a state of aberrant salience: a framework linking biology, phenomenology, and pharmacology in schizophrenia. *Am J Psychiatry* 2003;160:13–23.
- 38 Hazlett EA, Buchsbaum MS, Kemether E, Bloom R, Platholi J, Brickman AM, Shihabuddin L, Tang C, Byne W: Abnormal glucose metabolism in the mediodorsal nucleus of the thalamus in schizophrenia. *Am J Psychiatry* 2004;161:305–314.
- 39 Silver H, Feldman P, Bilker W, Gur RC: Working memory deficit as a core neuropsychological dysfunction in schizophrenia. *Am J Psychiatry* 2003;160:1809–1816.
- 40 Frith CD: Schizophrenia and theory of mind. *Psychol Med* 2004;34:385–389.