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CORRESPONDENCE

To the Editor:

The interesting meta-analysis by Mojtabai (1999) on the influence of chronicity on the structure of schizophrenic symptoms showed that longer duration of illness produced smaller negative–negative symptom-pair correlations and larger negative–positive symptom-pair correlations. These findings led the author to conclude that with increasing chronicity, dimensions of psychopathology become less cohesive and the boundaries between them less clear. The author himself raised doubts about the confidence of the findings because the inherent limitations of the 'review-generated evidence', and suggested that findings from 'study-generated evidence' would be more reliable.

We agree with this methodological issue and we report here on a re-analysis of our data from a large study aimed to address the factor structure of symptoms in schizophrenia and related psychoses (Peralta & Cuesta, 1999). The sample comprised patients with variable duration of illness, which represent the ideal condition to test the effect of chronicity in the symptom structure. We stratified the total sample ($N = 660$) in four groups with different levels of chronicity, which were defined according to the percentiles 25, 50 and 75 of years of duration of illness. On the basis of the findings by Mojtabai, we would expect to find with increasing degrees of chronicity either a blurred factor structure or an association between positive and negative factors.

We conducted four independent factor analyses on ten schizophrenic symptoms using the eigen value > 1 criterion to obtain factors and the oblimin procedure to rotate them. As can be seen in Table 1, the symptom factor structure remained the same across chronicity levels. The internal cohesiveness of the symptoms making the factors (α values) was also basically unchanged. Intercorrelations among factors revealed a consistent significant association pattern between negative and disorganization factors across levels of chronicity. The correlation between psychotic and negative factors evolved from a non-significant association in the psychoses with recent-onset or low chronicity to an inversely significant association in patients with moderate or high levels of chronicity. Lastly, the disorganization and negative factors became inversely correlated in the more chronic group of patients. In conclusion, our data do not support those of Mojtabai and show that the well-known three-factor structure of schizophrenic symptoms is independent of duration of illness, and that with increasing chronicity the psychotic factor becomes inversely correlated with the negative and disorganization factors.

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The Author replies:

Peralta and Cuesta, in their letter, present interesting data on the factor structure of SANS and SAPS from their large study of psychotic inpatients, stratified according to duration of illness. As the authors note, these data are not consistent with the conclusions of my earlier meta-analysis. However, because the samples of the two studies were not comparable, the results cannot be compared either. The meta-analysis was strictly limited to studies of patients with a

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Table 1. *Factor structure of schizophrenic symptoms across different levels of duration of illness*

		Recent-onset (< 2 years, $N = 159$)			Low chronicity (2–5 years, $N = 157$)			Moderate chronicity (6–11 years, $N = 155$)			High chronicity (> 12 years, $N = 189$)		
	Factors	I	II	III	I	II	III	I	II	III	I	II	III
Hallucinations		0·13	0·09	**0·82**	0·10	0·10	**0·84**	−0·02	0·03	**0·85**	0·03	−0·09	**0·85**
Delusions		−0·34	−0·23	**0·75**	−0·14	−0·15	**0·89**	−0·35	−0·10	**0·81**	−0·28	−0·07	**0·81**
Formal thought disorder		0·12	**0·85**	−0·07	0·00	**0·84**	−0·17	0·11	**0·88**	0·05	−0·21	**0·89**	0·11
Bizarre behaviour		−0·04	**0·63**	0·03	0·32	**0·63**	0·26	0·33	**0·57**	0·25	0·16	**0·70**	−0·07
Inappropriate affect		0·03	**0·63**	−0·34	0·33	**0·77**	−0·08	0·44	**0·66**	−0·04	0·45	**0·50**	−0·13
Inattentiveness		0·34	**0·75**	0·15	0·35	**0·70**	0·10	0·19	**0·79**	−0·11	0·32	**0·69**	−0·24
Affective flattening		**0·88**	0·15	−0·12	**0·90**	0·31	0·06	**0·89**	0·38	−0·12	**0·89**	0·17	−0·15
Alogia		**0·78**	0·42	0·05	**0·79**	0·30	−0·03	**0·78**	0·41	−0·23	**0·81**	0·24	−0·28
Avolition–apathy		**0·85**	0·08	0·02	**0·85**	0·35	−0·05	**0·85**	0·26	−0·06	**0·75**	0·28	−0·23
Anhedonia–asociality		**0·81**	−0·14	−0·06	**0·80**	0·11	0·05	**0·84**	0·05	−0·14	**0·77**	0·04	0·08
Internal consistency (α)		0·85	0·71	0·48	0·86	0·73	0·62	0·86	0·72	0·61	0·83	0·64	0·61
Factor intercorrelations†	I	—			—			—			—		
	II	0·13	—		0·30	—		0·29	—		0·21	—	
	III	−0·02	−0·05	—	0·04	0·00	—	−0·12	0·01	—	−0·16	−0·13	—

Bold figures denote factor loadings $\geqslant 0{\cdot}50$.

† Correlations coefficients $> 0{\cdot}11$ are significant after the Bonferroni correction ($0{\cdot}05 \div 12 = 0{\cdot}004$).

diagnosis of schizophrenia. Peralta & Cuesta's (1999) sample, on the other hand, included a mixture of psychotic disorders (including mood disorders with psychotic features, delusional disorder, psychotic disorder not otherwise specified, etc.) and only slightly over half of the 660 patients in their study had a DSM-III-R diagnosis of schizophrenia. A subsample of the patients with a schizophrenia diagnosis from this study was included in the meta-analysis.

This difference in the nature of samples might prove crucial. Although there is some evidence that the *cross-sectional* structure of symptoms in non-schizophrenic psychotic disorders might be similar to that of schizophrenia, there is no evidence that the *evolution* of symptom structure in non-schizophrenic psychoses is the same as in schizophrenia. Combining subjects with schizophrenia and non-schizophrenic psychoses, therefore, might obscure patterns of the evolution of symptom structure that are specific to schizophrenia. This problem is compounded by the probably uneven distribution of different diagnoses across the duration strata. Psychotic diagnoses such as brief reactive psychosis and schizophreniform disorder in DSM-III-R are, by definition, of short duration. Such disorders, therefore, will be found only among cases with < 2 years duration of illness in Peralta & Cuesta's study.

Replication of Peralta & Cuesta's analyses in the subsample of patients with a diagnosis of schizophrenia would be much more relevant to the results of the meta-analysis. I am also hopeful that this exchange of ideas would encourage other researchers with similar data to examine the impact of the duration of illness on the structure of symptoms.

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To the Editor:
I was interested in the twin study of disordered eating published in your journal recently (Wade *et al*. 1999), but surprised by the conclusions. In the third wave of the study 325 twins including 94 monozygotic (MZ) and 57 dizygotic (DZ) pairs agreed to be interviewed. Twenty-three of the 325 (7·1%) had a past diagnosis of bulimia nervosa. However, these 23 included only one twin pair concordant for lifetime bulimia nervosa, but as this was an MZ pair the authors concluded, after mulivariate analysis, that disordered eating has a heritability of 60%. They concluded that the most likely influence determining individual variation in disordered eating is 'additive genetic'; but this conclusion is based entirely on the one co-twin who had the same problem as her sister. As the great majority of the twins were discordant it might be wiser to conclude that there is no significant genetic influence in bulimia nervosa and that environmental factors should be the focus of further investigation.

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The Authors reply:
Dr Harding is correct in noting that only one pair of twins (MZ) was concordant for bulimia nervosa at the third wave assessment.

However, the statement that our conclusions are therefore 'based entirely on the one co-twin who had the same problem as her sister' represents a considerable misunderstanding of the methodology that was utilized. We provide the salient points of this methodology for those readers similarly confused by these analyses.

1 We used multivariate model fitting to analyse the correlations between three con-

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tinuous or polychotomous measures of disordered eating: (1) number of lifetime eating problems at Wave 1; (2) a 5-point measure of lifetime bulimic symptomatology at Wave 2; and, (3) the Wave 3 Eating Disorder Examination score summed from four subscales.

2 Use of continuous measures introduces considerably more power for genetic analyses than the use of binary measures (Neale *et al*. 1994).

3 Use of polychotomous measures assumes a liability-threshold model (Pearson, 1901), where such measures represent the latent or unobserved normal distribution of liability to illness. So while few women meet criteria for the threshold trait of bulimia nervosa, all women are represented on a normally distributed curve of liability to eating problems and concerns (Kendler, 1993).

Our results, using this powerful design, indicate that both genetic and environmental factors are important in the development of disordered eating, and as such, both should continue to be a focus of further research.

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