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## CORRESPONDENCE

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To the Editor:

Breslau *et al*. (**32**, 573–576) were critical of our editorial (Neria & Bromet, 2000) on the strengths and limitations of their analysis of the relationship between trauma exposure, post-traumatic stress disorder (PTSD) and depression (Breslau *et al*. 2000). Their research, as they point out, assumes 'the validity of the DSM definition of PTSD'. They argue that our comments were misguided because we do not appreciate the difference between PTSD and trauma-related depression. Our concern is not about the definition of PTSD but rather about whether their secondary analysis was able to address directly the occurrence of 'major depression (or another disorder) associated with traumatic events'. We were especially concerned because the data came from the DSM-III-R version of the Diagnostic Interview Schedule (DIS) and the Composite International Diagnostic Interview (CIDI) used in the National Comorbidity Survey (NCS); in neither case were respondents asked about 'major depression associated with traumatic events'. Moreover, in the NCS analysis, 25% of the exposed sample was excluded because age of onset was unavailable, including those with childhood physical abuse and childhood neglect. Kessler himself has shown that such exposures are associated with subsequent anxiety, mood and addictive disorders (Kessler *et al*. 1997) as well as PTSD (Kessler *et al*. 1995). In general, secondary analyses are often limited because the data are rarely optimal for addressing the new questions being asked.

Epidemiological research has several functions (Morris, 1964). The most basic descriptive function is to estimate the rate of clinical conditions in the general population and the risk factors associated with their occurrence. In this respect, studies like the National Comorbidity Survey (NCS) and the ongoing international World Mental Health (WMH) project in which we are participating produce valuable basic data, and longitudinal spin-offs, like the Baltimore ECA follow-up, provide a platform for examining relationships longitudinally. However, these studies were not designed to address a different and equally important function of epidemiology, and that is 'completing the clinical picture' (Morris, 1964). As we read the Breslau *et al*. (2000) analysis, this was indeed the underlying intent, and their paper is in fact subtitled 'the post-traumatic stress disorder-major depression connection'. Unfortunately, as noted above, respondents were not asked to report on post-trauma symptoms other than those contained in the diagnosis of PTSD. The situation was further biased by the order in which the modules were administered (Stone *et al*. 2000). In the NCS, the CIDI depression module was administered in 'Part 1' of the interview, while PTSD was covered much later in 'Part 2'. One could hypothesize that if trauma exposure had been explored before the depression screen was administered, the actual rate of post-trauma depression might have been different in exposed individuals. Again, our point is simply that statistical analysis cannot substitute for lack of direct data. The relationship between trauma, PTSD and the development of depression and other disorders is a terribly important issue with huge treatment implications, and it deserves to be studied directly in both general population and trauma-exposed samples.

When Breslau *et al*. suggested that we were 'speculating beyond the DSM classification', they were partly correct. While sociologists and epidemiologists have successfully translated existing criteria into structured interview schedules, the field of trauma research is badly in need of investigators who can 'think outside the box' and develop tools that will enable us to complete the clinical picture that arises and evolves over time after a traumatic event. Indeed, in addition to depression (Shalev *et al*. 1998), a number of other symptoms and disorders have been described in trauma survivors



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with PTSD, including substance abuse (e.g. Jacobsen *et al*. 2001), obsessive–compulsive disorder (e.g. Gershuny *et al*. 2002), somatization (Neria & Koenen, 2002), panic attacks or disorder (Chantarujikapong *et al*. 2001) and suicidality (Marshall *et al*. 2001). Once we understand the full range of conditions, and their overlap with PTSD, we will be positioned to design comprehensive clinical interventions and test their effectiveness.

We understand just how difficult a task we are proposing. However, we are more and more convinced of the importance of this task. For example, a recent post 9–11 study found similar rates of PTSD and depression (Galea *et al*. 2002). In the same vein, clinical research centres and practitioners in Manhattan are treating trauma victims with no prior psychiatric history who present with a broad array of problems, needs, grief reactions and other symptoms. Some of these patients meet criteria for PTSD, while others meet criteria for different DSM disorders. Some meet criteria for multiple conditions, and others do not meet criteria for any specific disorder. While clinicians make the link between exposure and its consequences in order to provide appropriate treatment, epidemiologic instruments are unable to do so (beyond the three symptom clusters defined in the DSM) and further are rarely designed to capture what Breslau *et al*. (2002) so eloquently refer to as the 'coalescence' of post-exposure symptoms.

In short, we see a gap between epidemiological studies that focus only on PTSD symptoms in the wake of trauma exposure, and cumulative clinical experience showing that trauma exposure leads to a broad spectrum of clinical symptoms and conditions that evolve over time and vary from culture to culture. In his last talk at the New York State Psychiatric Institute given more than a decade ago, the late Professor Joseph Zubin, the father of psychopathology research, chastized the audience for no longer listening to their respondents. Our present instruments prejudge what is salient for community respondents who experience traumatic events, be they individual traumas or community-wide disasters. While they are useful tools for establishing the prevalence of PTSD, they restrict our ability to fully explore what people undergo in the days and years after trauma exposure.

## REFERENCES


Breslau, N., Davis, G. C., Peterson, E. L. & Schultz, L. R. (2000). A second look at comorbidity in victims of trauma: the post-traumatic stress disorder-major depression connection. *Biological Psychiatry* **48**, 902–909.

Breslau, N., Chase, G. A. & Anthony, J. C. (2002). The uniqueness of the DSM definition of post-traumatic stress disorder: implications for research. *Psychological Medicine* **32**, 573–576.

Chantarujikapong, S. I., Scherrer, J. F., Xian, H., Eisen, S. A., Lyons, M. J., Goldberg, J., Tsuang, M. & True, W. R. (2001). A twin study of generalized anxiety disorder symptoms, panic disorder symptoms and post-traumatic stress disorder in men. *Psychiatry Research* **103**, 133–146.

Galea, S., Ahern, J., Resnick, H., Kilpatric, D., Bucuvalas, M., Gold, J. & Vlahov, D. (2002). Psychological sequelae of September 11 terrorist attacks in New York City. *New England Journal of Medicine* **346**, 982–987.

Gershuny, B. S., Baer, L., Jenike, M. A., Minichiello, W. E. & Wilkelm, S. (2002). Comorbid post-traumatic stress disorder: impact on treatment outcome for obsessive–compulsive disorder. *American Journal of Psychiatry* **159**, 852–854.

Jacobsen, L. K., Southwick, S. M. & Kosten, T. R. (2001). Substance use disorders in patients with post-traumatic stress disorder: a review of the literature. *American Journal of Psychiatry* **158**, 1184–1190.

Kessler, R. C., Sonnega, A., Bromet, E., Hughes, M. & Nelson, C. B. (1995). Post-traumatic stress disorder in the National Comorbidity Survey. *Archives of General Psychiatry* **52**, 1048–1060.

Kessler, R. C., Davis, C. G. & Kendler, K. S. (1997). Childhood adversity and adult psychiatric disorder in the US National Comorbidity Survey. *Psychological Medicine* **27**, 1101–1119.

Marshall, D. R., Olfson, M., Hellman, F., Blanco, C., Guardino, M. & Struening, L. E. (2001). Comorbidity, impairment, and suicidality in sub threshold PTSD. *American Journal of Psychiatry* **158**, 1467–1473.

Morris, J. N. (1964). *Uses of Epidemiology, 2nd edn*. Livingstone: London.

Neria, Y. & Bromet, E. J. (2000). Comorbidity of PTSD and depression: linked or separate incidence. *Biological Psychiatry* **48**, 878–880.

Neria, Y. & Koenen, K. (2002). Self-reported physical health in Israeli war veterans with combat stress reaction: an eighteen year follow-up study. *Anxiety, Stress, and Coping* (in the press).

Shalev, Y. A., Freedman, S., Peri, T., Brandes, D., Sahar, T., Orr, S. P. & Pittman, R. K. (1998). Prospective study of post-traumatic stress disorder and depression following trauma. *American Journal of Psychiatry* **155**, 630–637.

Stone, A. A., Turkkan, J. S., Bachrach, C. A., Jobe, J. B., Kurtzman, H. S. & Cain, V. S. (2000). *The Science of Self-report: Implications for Research and Practice*. Erlbaum Associates Inc.: Mahwah, NJ.



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The Authors reply:

This letter builds upon a scholarly and (we hope) amicable exchange between two research groups, each sharing a common interest in the human

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response to traumatic events, post-traumatic stress disorder, and the possibility that major depression might be a consequence of exposure to traumatic events of the type that meet diagnostic criteria for such events. Interested readers may appreciate a numbered chronology of entries.

1 An original report of new evidence from an ongoing prospective and longitudinal epidemiological study being conducted in Detroit, Michigan, and from ancillary analyses of data from the United States National Comorbidity Study (NCS), for which Breslau, Davis, Peterson and Schultz were the authors, and which appeared in *Biological Psychiatry* (cited below as Breslau *et al*. 2000).

2 An editorial commentary on the original research report, authored by Neria & Bromet, also published in *Biological Psychiatry* (Neria & Bromet, 2000).

3 An editorial on these same topics, authored by Breslau, Chase & Anthony (2002), subsequently published in *Psychological Medicine*, with attention to some of the points covered by Neria & Bromet (2000).

4 Neria and colleagues' letter to the editor concerning our editorial, published in this issue of *Psychological Medicine* (Neria *et al*. this issue).

5 This reply.

In this reply, we seek to correct a puzzling error or possible misunderstanding that appears in Neria and colleagues (this issue) regarding the original studies reported by Breslau *et al*. (2000) and to restate crucial methodological and conceptual points in our editorial (Breslau *et al*. 2002), which Neria and colleagues have ignored.

**Correction.** Neria and colleagues (this issue) state that their main concern is not with diagnostic definitions but with whether the 'secondary analysis (in Breslau *et al*. 2002) was able to address directly the occurrence of major depression associated with traumatic events'. They continue with a discussion of the limitations of the NCS.

This allusion to 'secondary analysis' and their focus on the NCS may convey the idea that Breslau *et al*. (2000) is a secondary analysis of NCS data. This is not the case. The report by Breslau *et al*. (2000) is a presentation of original evidence from on-going prospective and longitudinal studies of PTSD in the Detroit, Michigan area, which are 'primary' rather than 'secondary' analyses. Ancillary NCS analyses were presented for comparison with the Detroit results.

**Major depression after a traumatic event.** The subject matter in this exchange involves a possibility that risk of major depression might be elevated after exposure to a traumatic event. Breslau *et al*. (2000) reported that in both the prospective and retrospective data from the Detroit area a markedly increased risk for major depression was observed in exposed persons whose traumatic exposure was followed by PTSD. Risk of major depression was not elevated in trauma-exposed persons in the absence of PTSD, compared with persons unexposed to trauma. NCS data were then used to examine this finding, with a stratification of traumatic events by type.

This pattern of a markedly higher occurrence of major depression in victims with PTSD than in victims without PTSD can be observed in reports from other studies, including post-disaster studies (e.g. the Oklahoma City bombing), in which the links between questions about depression (and other disorders) and the recent disaster were perfectly clear to the respondents. To supply a very recent example, we have done hand calculations of numbers reported by Galea *et al*. (2002), cited by Neria and colleagues as showing similar rates of depression and PTSD in the aftermath of the attacks on the World Trade Center of 11 September 2001. Our hand calculations, kindly checked by S. Galea, reveal that the prevalence of major depression in persons with PTSD was 49% and in the absence of PTSD, only 6·5%. Galea noted that the latter figure is not far from what could be expected for past 30-day major depression in the general population, according to the NCS.

It is quite likely that there is an elevated risk for major depression in persons exposed to traumatic events *versus* those not exposed. In Breslau's study, there was an odds ratio of 1·7. However, the crucial finding in Breslau *et al*. (2000) is that the elevated risk for major depression is not attributable to exposure *per se*, because there was no excess risk of major depression in persons who were exposed but did not succumb to PTSD, compared to persons who were not exposed. The excess risk of major

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depression was restricted to trauma victims who had succumbed to PTSD. This finding narrows down potential explanations of the relationship linking PTSD and the onset of major depression: PTSD might be a causal determinant of major depression; or, these two disturbances might have a shared diathesis or shared environmental causes other than the exposure to trauma; or, perhaps there is some confounding process, with a resulting spurious association between PTSD and major depression. Evidence that family history of major depression and preexisting major depression are associated with increased risk for PTSD following trauma suggests that shared diathesis or genetic susceptibility might play a role (see Breslau *et al*. 2000).

**Methodological issue.** In our editorial published in *Psychological Medicine* (2002), we sought to clarify a methodological issue that had been raised by Neria & Bromet (2000). Neria & Bromet (2000) seem to want a research approach in which trauma victims are asked whether the trauma has caused them to experience the clinical features of major depression. That is, there may be something less than even-handed about an approach that asks trauma victims whether their trauma has been followed by clinical features like re-experiencing the trauma or nightmares about the trauma, but that fails to ask trauma victims whether their trauma has been followed by clinical features such as dysphoria or anhedonia. They describe the absence of the even-handed approach as an introduction of a 'procedural difference' that 'will inevitably reduce the strength of the relationship found between extreme stress and these other disorders relative to PTSD'.

In our editorial (Breslau *et al*. 2002), we pointed out that this is not a procedural difference but a definitional difference. A link with a traumatic event is required in the DSM for PTSD only and not for major depression. In consequence, the risk of PTSD in trauma victims is measured by a conditional probability or conditional risk (risk for those exposed to the stressor), whereas the association between trauma exposure and subsequent occurrence of major depression is measured by a ratio of risks, or by a relative risk estimate (risk of major depression among those exposed to the stressor divided by risk among those not exposed); hence, the two measurements cannot be directly compared. For PTSD, a link with a stressor is a necessary criterion for diagnosis; a relative risk with respect to the presence or absence of a stressor cannot be estimated. In contrast, there is no link with a stressor in the definition of major depression and the risk for major depression associated with exposure to stressors is evaluated relative to a reference group unexposed to stressors. Without such a comparison of risks, we could not test whether exposed persons are in fact at a higher risk for major depression than they would have been if they were not exposed. In PTSD, there is no such alternative.

Did Breslau *et al*. (2000) lack 'direct data' to evaluate the risk for major depression in trauma victims? Readers can judge for themselves. What the study does not have is information on respondents' attributions of their depressive complaints to traumatic events. But, this is not necessarily a deficiency. Such attributions are required for PTSD, but not for major depression. Reliance on such attributions may well take the field backward if the goal is to make any valid inferences on the question of the relative risk for major depression associated with trauma in victims with and without PTSD.

Our comments in the editorial (Breslau *et al*. 2002) apply to studies that have used the DSM definition of PTSD (i.e. nearly all published studies since 1980). In this sense, we 'assume' the validity of the PTSD definition. There is much to critique about the DSM approach to the specification of PTSD but the explicit link to a traumatic experience is difficult to challenge. Without exposure to trauma, what is 'post-traumatic' about the ensuing syndrome of traumatic memories and associated distressing symptoms? Rather than advocate for a new DSM category of 'Post-Traumatic Major Depression', we think it might be better to use the architecture of clinical and epidemiological research to illuminate whether the risk of major depression is increased after exposure to a stressor, and whether the occurrence of major depression depends upon presence of PTSD. This goal does not require a new category in the DSM, neither does it require reliance upon what may well be faulty causal attributions made by victims trying to understand their experiences in the aftermath of traumatic events.

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**Closing question.** We can imagine a line of research that seeks to be 'even-handed' in the approach to assessment of PTSD and mood disorders. It would ask trauma victims whether the trauma has been followed by the experience of dysphoria, anhedonia, and associated clinical features of mood disorders, and the experience of nightmares or other clinical features of PTSD. Would this be a line of research about major depression, as this disturbance currently is defined in either the DSM or the ICD? Or would it be a line of research about a different form of mood disturbance, akin to the 'Reactive Depression' formulation of earlier diagnostic systems? It is possible that the shared public health-oriented goals of our research groups will be served best by drawing a distinction between a 'Major Depression' that can be defined without reference to stressors, *versus* a 'Reactive Depression' that, like PTSD, is defined in terms of response to a traumatic event.

Our own standard approach is to investigate suspected cause-effect relationships in the manner of general epidemiology. Notwithstanding our compassion for disease victims and the degree to which we listen to them, in order to probe cause-effect relationships we typically would not choose to draw causal inferences by asking the lung cancer victim whether it was asbestos exposure, tobacco smoking, or something else that caused the disease. PTSD research is an exception, because the enquiry is guided by a disease construct that is specific with respect to cause (exposure to a specific class of stressors) and in which the content of the disturbance is linked tightly with the traumatic experience. By its definition and clinical nature, PTSD occurs only in the aftermath of a stressor. 'Reactive Depression' research also could be one of these exceptions.

Is aetiological research on major depression a similar exception? We think not, but let us ask for new evidence rather than an appeal to authority before we put this issue to rest. We believe that Neria, Bromet and Marshall are asking for a new line of research about 'Reactive Depression,' for which exposure to a traumatic event is a required criterion. This line of research would be different from what we are doing, not necessarily worse, not necessarily better – just different. We wish them well, and await evidence from this new line of research that will compel us to change our point of view.

## REFERENCES

Breslau, N., Davis, G. C., Peterson, E. L. & Schultz, L. R. (2000). A second look at comorbidity in victims of trauma: the post-traumatic stress disorder – major depression connection. *Biological Psychiatry* **48**, 902–909.

Breslau, N., Chase, G. A. & Anthony, J. C. (2002). The uniqueness of the DSM definition of post-traumatic stress disorder: implications for research. *Psychological Medicine* **32**, 573–576.

Galea, S., Ahern, J., Resnick, H., Kilpatric, D., Bucuvalas, M., Gold, J. & Vlahov, D. (2002). Psychological sequelae of September 11 terrorist attacks in New York City. *New England Journal of Medicine* **346**, 982–987.

Neria, Y. & Bromet, E. J. (2000). Comorbidity of PTSD and depression: linked or separate incidence. *Biological Psychiatry* **48**, 878–888.

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To the Editor:

Pillmann *et al*. (**32**, 525–533) reported that 76% (29/38) of patients with ICD-10 acute transient psychotic disorder (ATPD) experienced at least one relapse by the time of follow-up, which was conducted 2·2 years on average after the index episode. These results from Germany may at first appear to be at odds with findings by Susser *et al*. from samples of patients with non-affective acute remitting psychosis (NARP) from India (Susser *et al*. 1998) and the US (Susser *et al*. 1995). In the study from India only 18% (3/17) of patients relapsed throughout a 12-year follow-up period and in the US study, only one (14%) out of the seven cases of NARP followed for up to 2 years had a relapse.

The differences in relapse rates between the Pillman *et al*. study (2002) on the one hand, and the Susser *et al*. studies (1998, 1995), on the other, are puzzling. It is unlikely that these differences are attributable to differences between the criteria used to define NARP and the ICD-10 ATPD. Similar to ATPD, NARP is defined as a non-affective remitting psychosis with acute onset, the major difference being that the maximum duration criterion for NARP is 6 months and is thus less restrictive than the 1–3 months duration criteria for the ATPD. The clinical

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status at follow-up of ATPD and NARP also appear to be similarly favourable. Eighty-two per cent (32 of 38) of the patients in the Pillman study were in remission and had an 'excellent' social outcome at the time of the follow-up assessment, as were 94% (16 of 17) of the patients in the Susser *et al*. study from India and all of the patients in the Susser *et al*. study from the US.

We believe that the best explanation for the differences in relapse rates among these studies might be a difference in sampling methods. Specifically, the Susser *et al*. samples were ascertained either at first treatment contact (1998) or first-admission (1995) (i.e. incidence samples), whereas the Pillman *et al*. sample was comprised of consecutive admissions (i.e. a prevalence sample). Patients in the Susser *et al*. samples had no previous treatment contacts, whereas 45% of the ATPD patients in the Pillman *et al*. study had previous treatment episodes, with the sample having an average of 1·6 previous episodes at intake (range 0–16). Thus, the Susser *et al*. samples represent incidence cases whereas the Pillman *et al*. sample overrepresents patients with a recurrent type of illness. As a result, the course of ATPD in the Pillman *et al*. sample would appear to be worse than it is in the community.

Similarly, a review of follow-up studies of schizophrenia noted that a larger proportion of patients in first-admission studies have reasonably good outcomes compared to consecutive admission studies (Ram *et al*. 1992). The discrepancy in the results of the two types of studies is likely to be larger for a condition with more variability in course than schizophrenia. A large proportion of patients with acute remitting psychosis (however defined) never require further treatment, whereas a small proportion require multiple treatment contacts. This latter group would predominate in a prevalence sample based on consecutive admissions.

It is also interesting to note that the classic descriptions of cycloid psychosis (Perris, 1974) and reactive psychosis (McCabe, 1975) – psychoses with features similar to NARP and ATPD – depict these conditions as highly recurrent. Data on the long-term course of these conditions are mostly based on consecutive samples (e.g. Perris, 1974; McCabe, 1975) and thus might also overrepresent cases with high recurrence rates.

Careful large-scale studies based on consecutive admissions, such as the study by Pillman *et al*. (2002), provide valuable information on the prevalence of disorders and demand for services. However, the least biased method for assessing illness course is to use incidence samples, particularly samples that are representative of the areas from which they are drawn.

## REFERENCES

McCabe, M. (1975). Reactive psychoses: a clinical and genetic investigation. *Acta Psychiatrica Scandinavica Supplementum* **259**, 1–133.

Perris, C. (1974). A study of cycloid psychoses. *Acta Psychiatrica Scandinavica Supplementum* **253**, 1–77.

Pillman, F., Haring, A., Balzuweit, S., Blöink, R. & Marneros, A. (2002). The concordance of ICD-10 acute and transient psychosis and DSM-IV brief psychotic disorder. *Psychological Medicine* **32**, 525–533.

Ram, R., Bromet, E. J., Eaton, W. W., Pato, C. & Schwartz, J. E. (1992). The natural course of schizophrenia: a review of first-admission studies. *Schizophrenia Bulletin* **18**, 185–207.

Susser, E. S., Fennig, S., Jandorf, L., Amador, X. & Bromet, E. (1995). Epidemiology, diagnosis, and course of brief psychoses. *American Journal of Psychiatry* **152**, 1743–1748.

Susser, E. S., Varma, V. K., Mattoo, S. K., Finnerty, M., Mojtabai, R., Tripathi, B. M., Misra, A. K. & Wig, N. N. (1998). Long-term course of acute brief psychosis in a developing country setting. *British Journal of Psychiatry* **173**, 226–230.

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The Authors reply:

We agree with Dr Mojtabai and colleagues that the inclusion of patients with multiple episodes in our sample might potentially lead to an overrepresentation of recurrent cases. However, the actual impact of this bias on the results reported in our study seems rather small. We recalculated relapse rates for the subsample of patients that entered the study at first episode. The proportion of patients with a relapse during follow-up decreased slightly from 29/38 (76·3%) to 14/20 (70·0%), and the number of episodes during the follow-up period decreased from $1{\cdot}28 \pm 0{\cdot}65$ to $1{\cdot}1 \pm 0{\cdot}75$ (differences between first and multiple admission not significant; for proportion with relapse: $P = 0{\cdot}454$ with Fisher's exact test, for number of episodes during follow-up period: $t = 0{\cdot}510$, df = 36, $P = 0{\cdot}613$). Thus,

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even when only first-episode patients are considered, the relapse rate in our study is far higher than the figures of 18% and 14% reported by Susser *et al*. (1995, 1998). We submit that the discrepancy is related to differences in the diagnostic criteria employed, and possibly to population differences.

In our study we used ICD-10 and DSM-IV criteria for acute and transient psychotic disorder (ATPD) and brief psychotic disorder (BPD) without modification. The two studies by Susser *et al*. (1995, 1998) used criteria differing from ICD-10 and DSM-IV as well as from each other. In the Indian study (Susser *et al*. 1998), the criteria of acute brief psychoses included freedom from relapse at 2-year follow-up (otherwise a diagnosis of acute relapsing psychosis was assigned). The fact that only 18% of the patients relapsed within 12 years is remarkable. Obviously, in this sample, freedom from relapse after 24 months (despite discontinuation of pharmacotherapy in most cases) strongly predicted freedom of relapse after 12 years. However, these results cannot be generalized to all cases of ATPD or BPD, the definitions of which do not contain a criterion of relapse-free survival. In fact, the majority of patients in our sample (including most of the first-episode patients) would not fulfil the criteria of Susser *et al*. (1998). In addition, prevalence differences between industrialized and developing countries could also contribute to the difference between the studies. Brief and acute psychoses appear to be more frequent in developing countries and may carry a better prognosis (Hopper & Wanderling, 2000).

The diagnostic criteria used in the North American study of Susser *et al*. (1995) are more similar to those used by us, but they contain the requirement of a state of complete remission at (not within) 6 months after initial evaluation. This criterion might have excluded some patients with early relapse. If, in addition, the small size of this sample ($N=7$) is considered, the difference from our results seems less puzzling.

Meanwhile, one Indian study (Sajith *et al*. 2002), using a first-episode sample of patients with ATPD (acute polymorphic subtype), reported a relapse rate – 47% relapse within 3 years – intermediate between the data of Susser *et al*. (1998) and our data. In conclusion, we agree with Dr Mojtabai and colleagues that single episodes of acute brief psychoses without further relapse exist and that their incidence is best estimated by the analysis of first-episode samples (or subsamples). We maintain, however, that our results imply that the frequency of single-episode ATPD is much lower than 50%, although the figure may be higher in developing countries. Clearly, further studies are needed to resolve the issue. Preferentially, such investigations should employ standardized criteria as implemented by ICD-10 and DSM-IV to ensure comparability across studies. The use of 'prognostic' criteria requiring a certain status and/or freedom from relapse at a specified future time carries theoretical problems and may impair clinical applicability.

## REFERENCES

Hopper, K. & Wanderling, J. (2000). Revisiting the developed versus developing country distinction in course and outcome in schizophrenia: results from ISoS, the WHO collaborative follow-up project: International Study of Schizophrenia. *Schizophrenia Bulletin* **26**, 835–846.

Sajith, S. G., Chandrasekaran, R., Sadanandan Unni, K. E. & Sahai, A. (2002). Acute polymorphic psychotic disorder: diagnostic stability over 3 years. *Acta Psychiatrica Scandinavica* **105**, 104–109.

Susser, E., Fennig, S., Jandorf, L., Amador, X. & Bromet, E. (1995). Epidemiology, diagnosis and course of brief psychoses. *American Journal of Psychiatry* **152**, 1743–1748.

Susser, E., Varma, V. K., Mattoo, S. K., Finnerty, M., Mojtabai, R., Tripathi, B. M., Misra, A. K. & Wig, N. N. (1998). Long-term course of acute brief psychosis in a developing country setting. *British Journal of Psychiatry* **173**, 226–230.

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