Schizophrenia Bulletin vol. 40 no. 2 pp. 269–277, 2014
doi:10.1093/schbul/sbt149
Advance Access publication October 25, 2013

Mood Instability and Psychosis: Analyses of British National Survey Data

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Background: **We used British national survey data to test specific hypotheses that mood instability (1) is associated with psychosis and individual psychotic phenomena, (2) predicts the later emergence of auditory hallucinations and paranoid ideation, and (3) mediates the link between child sexual abuse and psychosis.** ***Methods:*** **We analyzed data from the 2000 and 2007 UK national surveys of psychiatric morbidity (*N* = 8580 and 7403, respectively). The 2000 survey included an 18-month follow-up of a subsample (*N* = 2406). Mood instability was assessed from the Structured Clinical Interview for DSM-IV Axis II (SCID-II) questionnaire. Our dependent variables comprised auditory hallucinations, paranoid ideation, the presence of psychosis overall, and a 15-item paranoia scale.** ***Results:*** **Mood instability was strongly associated in cross-sectional analyses with psychosis (2000: OR: 7.5; 95% CI: I 4.1–13.8; 2007: OR: 21.4; CI: 9.7–41.2), paranoid ideation (2000: OR: 4.7; CI: 4.1–5.4; 2007: OR: 5.7; CI: 4.9–6.7), auditory hallucinations (2000: OR: 3.4; CI: 2.6–4.4; 2007: OR 3.5; CI: 2.7–4.7), and paranoia total score (2000: Coefficient: 3.6; CI: 3.3–3.9), remaining so after adjustment for *current* mood state. Baseline mood instability significantly predicted 18-month inceptions of paranoid ideation (OR: 2.3; CI: 1.6–3.3) and of auditory hallucinations (OR: 2.6; CI: 1.5–4.4). Finally, it mediated a third of the total association of child sexual abuse with psychosis and persecutory ideation and a quarter of that with auditory hallucinations.** ***Conclusions:*** **Mood instability is a prominent feature of psychotic experience and may have a role in its genesis. Targeting mood instability could lead to innovative treatments for psychosis.**

Key words: epidemiology/psychopathology/paranoia/auditory hallucination/child sexual abuse

Introduction

Classification systems in mental health have historically maintained a core division between psychotic conditions and those disorders in which changes to mood or emotion are key. However, affect is now seen as central to understanding all schizophreniform disorders.[1–5] Anxiety and depression are given prominent roles in models of psychosis,[6–10] and are associated with clinical and nonclinical paranoid thinking and with the emergence and persistence of auditory hallucinations.[11–15] Experience sampling methods (ESM) and experimental studies have corroborated these findings.[16–23] The antecedence of affective symptoms is also apparent from the fact that the prodromes of schizophrenia and affective disorders are indistinguishable until the emergence of psychotic symptoms.[24] Consonant with these findings, psychotic-like experiences are more common in individuals with anxiety and depressive disorders,[25] while people at risk of psychosis and those with established psychotic disorder both have high rates of depression and anxiety.[26,27] Finally, the biological vulnerability to psychosis appears to include an enhanced hypothalamic pituitary axis response to stress.[28]

Psychotic disorders and experiences are probably linked to a range of affective symptoms. While anxiety and depression are conceptually and phenomenologically separable, they are strongly correlated and show considerable genetic overlap, indicating a shared negative affectivity factor and shared psychological processes.[29–31]

One of the notable features of psychosis is that its symptoms fluctuate, sometimes markedly.[24,32] Large variations are captured under the rubrics of onset, remission, recovery, and relapse,[33] but most fluctuations are more restricted in amplitude and duration. If affective symptoms are associated with psychotic symptoms, they are likely to covary, making it at least plausible that the ebb

and flow of psychotic symptoms is consequent on similar changes in affect.[19] People who later go on to develop full-blown psychosis may also be temperamentally susceptible, for a variety of reasons, to abnormally unstable mood, ie, to a degree of emotional dysregulation.[34] In its extreme version, this allows a diagnosis of schizoaffective disorder, but less prominent mood instability (MI) may still shape psychotic experiences.

MI has been neglected in psychosis. Where the establishment of psychiatric categories has had regard to the emotional characteristics of individuals, affective symptom constructs have been based on particular dimensions, such as intensity and duration. The fluctuation of mood over time (which enlists frequency and amplitude) has mainly been studied in the context of borderline personality disorder and bipolar disorder.[35,36]

Research focussing on borderline personality disorder suggests childhood adversity (in particular, child sexual abuse [CSA]) may per se result in mood dysregulation,[37,38] forming part of the pathway from abuse to personality dysfunction in adulthood. Similar causal pathways might exist in psychosis, given the established connection between CSA and adult psychosis.[39] The development of psychotic symptoms may be linked to trauma-related cognitive and affective vulnerabilities.[40] Fluctuations in self-esteem and avoidance of emotion have been found to be associated with paranoia, using ESM,[41,42] and features of emotional dysregulation—eg, avoidance of emotional responses and failure to control behaviors when upset—are associated with persecutory ideation.[43] Thus, the association between CSA and psychosis may be mediated by MI.

This article is based on data from two British national surveys of psychiatric morbidity, carried out in 2000 and 2007.[44] The first survey also incorporated an 18-month follow-up. In order to triangulate our hypothesized relationship between subjective emotional dysregulation and psychosis, we used these data to test several linked hypotheses based on complementary measures of psychotic processes. We analyzed the surveys independently, as a way of putting our central hypothesis at greatest risk (we could equally have amalgamated them to increase power).

Our specific hypotheses were as follows: that each of three measures of psychotic disorder and experience would be associated cross-sectionally with MI; that both the emergence and maintenance over time of delusions and hallucinations would be predicted by antecedent MI; and that the separate associations of CSA with a diagnosis of psychosis, with delusions, and with hallucinations would be significantly mediated by MI. Finally, we predicted these associations would persist after controlling for current mood symptoms, thereby implying that MI has an effect over and above the mere presence and severity of anxiety and depression. We tested no hypotheses other than those set out here.

Methods

Setting and Design

Further details of the survey methods are available in the supplementary material and the survey reports.[45,46] Participants were selected using population-based multiphase probability sampling. The targeted age range was 16–74 in the 2000 National survey (N = 8580, response rate 70%), and 16+ in 2007 (N = 7403, response rate 57%). A second phase interview was carried out by clinically trained research interviewers using the Schedules for Clinical Assessment in Neuropsychiatry (SCAN).[47] There was an 18-month follow-up of a subsample (N = 2406) of the 2000 survey.

We had access to three ways of identifying psychotic phenomena: a diagnosis of probable psychosis, specific ratings of paranoid ideation and auditory hallucinations, obtained from the Psychosis Screening Questionnaire (PSQ),[48] and the score on a continuously distributed dimension of paranoia[49] (the latter was available only from the 2000 survey data—see supplementary material).

We identified four levels of severity of depressive/anxiety symptoms (0–5, 6–11, 12–17, and 18+) obtained from the Clinical Interview Schedule Revised (CIS-R).[50] Hypomanic mood was assessed in relation to the past year in the first section of the PSQ. The 2007 survey included screening for posttraumatic stress disorder (PTSD), using the Trauma Screening Questionnaire.[51]

The MI measure was an item from the DSM-IV Borderline Personality Disorder section of the Structured Clinical Interview for DSM-IV (SCID-II).[52,53] Respondents were asked (in relation to "the last several years") "do you have a lot of sudden mood changes?" to which they could answer *yes* or *no*.

The 2007 survey included detailed enquiry about sexual abuse. In our analyses here, we focus on abuse in childhood (age <16) involving sexual intercourse or other physical molestation (see supplementary material).

Analytic Strategy

The data were analyzed using the Statistical Package for the Social Sciences (version 19 for Windows) and Stata (version 11.2 for Windows). To assess the association of our measures of psychosis with MI, we used binary logistic regression, ordered logistic regression, and linear regression, as appropriate. Data were weighted to allow for design and response rates in order to render the results representative of the national household population.[45,46]

Regression was carried out in four stages designed to be maximally informative in relation to our hypotheses. We initially produced unadjusted ORs. We next adjusted for sociodemographic characteristics (age, sex, marital status, employment status, and ethnicity [see supplementary material]). We then adjusted for the total score on the

CIS-R, in order to distinguish the contribution of instability from that of current dysphoric mood disturbance.

The final analysis included controlling for hypomanic mood and (only in the 2007 survey) for the avoidance and numbing aspects of PTSD. People who endorse enquiries about hypomanic mood are likely to experience mood fluctuations from elation to depression. Such instability may be conceptualized as a trait or, in the context of bipolar disorder and schizoaffective disorder, as a symptom.[54] Controlling for current hypomanic symptoms counters the argument that MI might merely be the reflection of a manic process. PTSD is known to be associated with psychosis,[55,56] and there is good evidence that severe (life-threatening) events may generate psychotic symptoms.[57] As MI might occur only in people with psychosis who also screen positive for PTSD, we controlled for PTSD in the 2007 data set.

Finally, to test the role of MI as a mediator of the relationship linking CSA with psychosis, auditory hallucinations, and paranoid ideation, we applied the Karlson Holm Breen command in Stata to the 2007 data set. This method of mediation analysis decomposes the total effect of a variable into direct and indirect effects.[58]

Results

Cross-Sectional Analyses

In table 1, we present separate analyses of the relationship between MI in psychosis based on the national surveys carried out in 2000 and 2007. Endorsement of MI greatly increased the (weighted) prevalence of psychosis (from 0.3% to 2.0% in 2000 and from 0.1% to 2.2% in 2007). MI was a very common concomitant of psychosis, being present in 53% of people with psychosis in 2000 and in 77% in 2007 (this discrepancy between surveys is significant, $P < .025$). In both surveys, the equivalent general population rate was around 14%. The corresponding ORs were large and highly significant in each year (7.5 (CI: 4.1–13.8) and 21.4 (CI: 9.7–47.2), respectively), and remained so (6.4 (CI 3.4–11.9) and 16.2 (CI 7.4–35.6), respectively) after adjustment for age, sex, marital status, employment status, and ethnicity. Adjustment for current affective state as indicated by the CIS-R total score reduced but did not eliminate the extent and significance of the association. Further control, for hypomanic mood in the 2000 data set, and for hypomanic mood and PTSD screen status in the 2007 data set, had little additional effect on the ORs linking instability and psychosis. The greater size of the unadjusted association in the 2007 survey meant that adjustment had relatively less impact.

In tables 1 and 2 of the supplementary material, we provide the equivalent cross-sectional analyses relating to auditory hallucinations and paranoid ideation, as derived from the PSQ. The ORs were not so large because we were using ordered logistic regression on variables with 3 and 4 levels, respectively. However, they remained highly significant: in all cases, they closely paralleled the results based on a diagnosis of probable psychosis.

Our next cross-sectional analysis was of the distribution of paranoia scores in the 2000 data (figure 1; table 2). The curves for people who endorsed the MI item and for those identified as having probable psychosis were compared with that for the total population. In both cases, the curves were, as predicted, shifted to the right, indicating a greater preponderance of higher scores in these groups. The unadjusted coefficient linking MI with the paranoia scale was 3.63 ($P < .001$), and this was scarcely changed by sequential adjustments, for sociodemographic characteristics, for CIS-R score, and for the symptom of hypomanic mood.

Longitudinal Analyses

In table 3, we present the longitudinal ordered logistic regression analysis of the effect of MI on the emergence and maintenance of paranoid ideation. In people who were not initially paranoid, baseline MI predicted the development of paranoid ideation over the 18-month follow-up period. If the baseline CIS-R score was controlled,

Table 1. The Cross-Sectional Relationship Between Mood Instability and Psychosis

	OR (95% Confidence Limits)	*t*	*P* > *t*
National Psychiatric Morbidity Survey 2000			
Unadjusted ($\chi^2 = 57.5$; $P < .001$)	7.51 (4.1–13.8)	6.54	<.001
Controlling for sociodemographic variables[a]	6.35 (3.4–11.9)	5.75	<.001
Controlling for the above *plus* CIS-R total score	2.03 (1.1–3.7)	2.26	.024
Controlling for the above *plus* hypomanic mood	1.99 (1.1–3.7)	2.19	.029
Adult Survey of Psychiatric Morbidity 2007			
Unadjusted ($\chi^2 = 100.6$; $P < .001$)	21.43 (9.7–47.2)	7.64	<.001
Controlling for sociodemographic variables[a]	16.2 (7.4–35.6)	6.98	<.001
Controlling for the above *plus* CIS-R total score	9.07 (3.4–24.5)	4.36	<.001
Controlling for above *plus* PTSD symptoms and hypomanic mood	8.11 (2.8–23.5)	3.87	<.001

Note: CIS-R, Clinical Interview Schedule Revised; PTSD, posttraumatic stress disorder.
[a]Age, sex, marital status, employment status, and ethnicity.

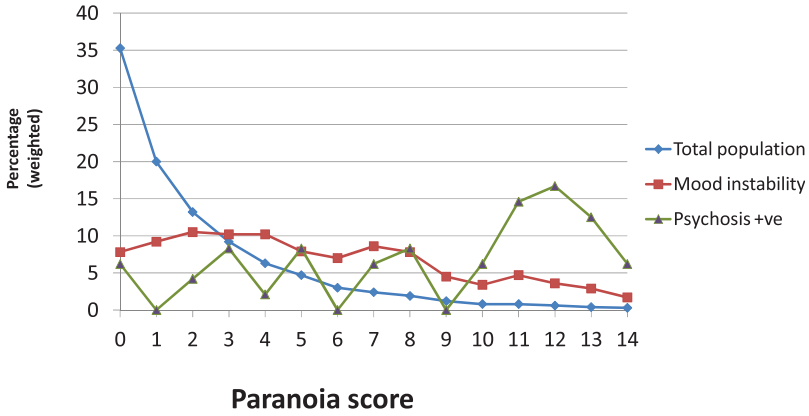

Fig. 1. The cross-sectional associations of mood instability and psychosis with paranoia score.

Table 2. The Cross-Sectional Relationship Between Mood Instability and Paranoia Score in the General Population

	Coefficent (95% Confidence Limits)	*t*	*P* > *t*
National Psychiatric Morbidity Survey 2000			
Unadjusted	3.63 (3.34–3.92)	24.42	<.001
Controlling for sociodemographic variables[a]	3.47 (3.18–3.76)	23.39	<.001
Controlling for the above *plus* CIS-R total score	4.49 (3.55–5.43)	9.71	<.001
Controlling for above *plus* hypomanic mood	4.44 (3.50–5.39)	9.63	<.001

Note: CIS-R, Clinical Interview Schedule Revised.
[a]Age, sex, marital status, employment status and ethnicity.

Table 3. The Effect of Mood Instability on Inceptions of Paranoid Ideation and Maintenance of Existing Paranoid Ideation (Psychosis Screening Questionnaire)

	OR	95% CI	*t*	*P* > *t*
New paranoid ideation emerging at T2				
Mood instability	2.25	1.55–3.26	4.27	<.001
Controlling for CIS-R score at T1				
Mood instability	1.60	1.06–2.42	2.24	.026
CIS-R score				
0–5 (reference)				
6–11	2.87	1.88–4.39	4.91	<.001
12–17	3.01	1.69–5.34	3.76	<.001
18+	3.79	2.20–6.53	4.81	<.001
Initial paranoid ideation maintained at T2				
Mood instability	2.45	1.80–3.34	5.67	<.001
Controlling for CIS-R score at T1				
Mood instability	2.05	1.47–2.87	4.22	<.001
CIS-R score				
0–5 (reference)				
6–11	1.18	0.67–2.07	0.57	.57
12–17	1.36	0.74–2.48	1.00	.32
18+	2.24	1.23–4.09	2.64	.009

the OR reduced from 2.3 (CI: 1.6–3.3) to 1.6 (CI: 1.1–2.4) but remained significant. In participants who initially acknowledged paranoid ideas, baseline endorsement of MI predicted the maintenance of paranoia at follow-up (OR: 2.45; CI: 1.8–3.3). Again this was little affected by controlling for initial CIS-R score.

The emergence of auditory hallucinations appeared to be predicted by MI in the same way (table 4), although controlling for baseline CIS-R score reduced the OR, from a highly significant 2.6 (CI: 1.5–4.4) to 1.7 (CI: 0.97–2.9): the *P* value then fell short of conventional significance, at 0.063. However, MI did not predict the maintenance

Table 4. The Effect of Mood Instability on Inceptions of Auditory Hallucinations and Maintenance of Existing Auditory Hallucinations (Psychosis Screening Questionnaire)

	OR	95% CI	*t*	*P* > *t*
New auditory hallucinations emerging at T2				
Mood instability	2.58	1.52–4.38	3.51	<.001
Controlling for CIS-R score at T1				
Mood instability	1.67	0.97–2.88	1.86	.063
CIS-R score				
0–5 (reference)				
6–11	2.43	0.99–5.96	1.94	.053
12–17	3.83	1.50–9.74	2.83	.005
18+	5.28	2.10–13.25	3.55	<.001
Initial auditory hallucinations maintained at T2				
Mood instability	1.59	0.68–3.72	1.08	.28

Note: CIS-R, Clinical Interview Schedule Revised.

of auditory hallucinations. It is possible that hallucinations are more sporadic than paranoid ideation; if so, this would reduce the ability to predict their emergence and maintenance.

Mood Instability as a Mediator

In table 5, we use data only available in the 2007 survey to analyze the extent to which MI might mediate the associations of CSA with psychosis, paranoid ideation, and auditory hallucinations. The indirect route via MI was highly significant, accounting for over a third of the total effect for psychosis and persecutory ideation, and a quarter of that for auditory hallucinations.

Discussion

Findings

In this article, we examined interlinked hypotheses about the influence of MI in psychosis, exploiting the data from two separate surveys to provide replication. Our use of multiple measures (a diagnosis of psychosis, the presence of auditory hallucinations or of paranoid ideation, and a continuous measure of paranoia) should be seen as a form of sensitivity analysis. In all cases bar one, our hypotheses were strongly supported.

MI was strongly associated in cross-sectional analyses with all of our chosen measures, remaining so after adjustment for sociodemographic variables and for current mood state. This implied a contribution from MI independent of the mere presence of anxiety, depression, and hypomania.[27] Although unpleasant and threatening psychotic experiences and thoughts might well lead to proportionate fluctuations in mood, our findings argue that MI is part of the psychotic process, whether as antecedent, concomitant, or consequence.

Although limited to the specific symptoms of auditory hallucinations and paranoid ideation, our prospective analyses from the 18-month follow-up of the 2000 survey allow more secure causal inference. This is an unusually long interval over which to demonstrate relationships between psychological attributes,[59] but MI significantly predicted *new inceptions* of both paranoid ideation and auditory hallucinations though the finding for the latter was reduced to trend level after controlling for baseline CIS-R score. MI also predicted the *maintenance* of paranoid ideation, but not of auditory hallucinations.

Our hypothesis that MI substantially mediates the association of CSA with psychosis, paranoid ideation, and auditory hallucinations was also supported. Others have found that dysphoric mood states and mood changes may shape the emergence and persistence of psychotic phenomena in response to different forms of stress.[39,59–63]

Limitations

It is difficult to achieve full enumeration of cases of psychosis in large epidemiological surveys. Some cases in this study were identified from indirect information obtained at the screening stage rather than from the full phase 2 clinical interview. However, using the screening results in this way would in any case have identified 19 of the 23 individuals diagnosed as having psychosis by using SCAN in the 2007 survey.[39] Our category of psychosis did not distinguish affective, schizoaffective, and nonaffective varieties. For this reason, we controlled for hypomanic mood, and, significantly, this did not change the relationship between MI and psychosis.

While the relationship between MI and diagnosed psychosis was significant in both the 2000 and the 2007 surveys, it was significantly stronger in the latter. As the methods of the surveys were identical in relation to this analysis, methodological differences cannot explain this discrepancy nor can routine sociodemographic differences because controlling for a range of sociodemographic

Table 5. Mood Instability as a Mediator of the Link Between Child Sexual Abuse and Psychotic Phenomena

Effect	OR	Robust SE	*z*	*P* > *z*	95% CI
Probable psychosis					
Reduced	11.09	4.95	5.39	.0001	4.62–26.62
Full	4.83	2.22	3.42	.001	1.96–11.90
Difference	2.30	0.42	4.54	.0001	1.60–2.39
34.6% of the link is mediated by mood instability					
Paranoid ideation					
Reduced	4.10	0.80	7.25	.0001	2.80–6.00
Full	2.52	0.50	4.66	.0001	1.71–3.72
Difference	1.63	0.14	5.63	.0001	1.37–1.93
34.5% of the link is mediated by mood instability					
Auditory hallucinations					
Reduced	3.94	1.24	4.37	.0001	2.13–7.29
Full	2.79	0.87	3.28	.001	1.51–5.14
Difference	1.41	0.10	4.90	.0001	1.23–1.63
25.3% of the link is mediated by mood instability					

variables did not much affect the discrepancy, and the data were weighted for design and nonresponse in each survey in order to approximate to the national population. The prevalence of diagnosed psychosis is relatively low in representative population surveys (here around half of 1%), and this will introduce a degree of instability; in this context, our findings can be seen as notably consistent.

There is no accepted and validated definition or method of measuring MI.[64] Although it does have concomitant behavioral manifestations, MI is primarily perceived subjectively. It is therefore appropriate to base its evaluation on self-report. Our measure was based on responses to a single unelaborated question. However, the question appeared readily comprehensible to participants and had good face validity: very few declined to answer it. In 2007, 48 people could not or would not respond, while in 2000 only 25 were in this category.[39] The capacity of the measure to substantiate hypotheses like those tested here also gives it a degree of construct validity. More detailed enquiry would improve precision, as individual responses will reflect different frequencies and amplitudes of mood variation. In some cases, these might indicate occasional days of mood disturbance, in others more frequent disturbance, and in yet others sizeable week- or month-long swings. The concept of MI could therefore be seen as the integrated molar equivalent of the more molecular (or atomic) level of daily life stress reactivity.[18,20] Finally, it is not clear if the "yes" answer to our question about MI would be phenomenologically equivalent in borderline personality disorder, affective psychosis, and nonaffective psychosis; this needs to be investigated further. It is noteworthy that responses to a very similar question were capable of predicting the onset of bipolar disorder in an at-risk sample.[65]

Theoretical and Clinical Implications

A range of putative mechanisms might explain links between MI and psychosis. The additional effect of instability may arise merely by providing more frequent experiences of dysphoric mood. However, MI may equally foster mental environments in which abnormal beliefs and psychotic experiences emerge. Thus, the repetition of dysphoric mood may have cumulative effects, for instance by encouraging metacognitions such as the unsettling belief that equanimity can never be relied upon. Bursts of anxiety may be taken as evidence of imminent danger; given a context of prior abuse, this sense of threat may become attached to people nearby and the world comes to seem persistently unsafe. The sense that emotional experiences are out of one's personal control may prompt a search for meaning that may find explanations in terms of external influence.

The effect of MI may also correspond to neurobiological changes rendering individuals more vulnerable to psychotic experiences. For example, emotional dysregulation in borderline personality disorder has been associated with changed central dopaminergic and serotinergic functioning,[38] and a dopamine transporter polymorphism has been linked specifically to borderline personality disorder.[66]

Our analysis of MI in relation to the continuously distributed paranoia score corroborated our findings for identified cases of psychosis but suggested additionally that the processes involved are equally characteristic of nonclinical paranoid experience. Psychosis comprises a range of distinct experiences, including paranoia, grandiosity, hallucinations, anhedonia, and thought disorder,[67–69] and these form continua in the general population.[11,49,70–73] Only the rare individuals at the severe end of a number of these dimensions are diagnosable in terms of current

classifications of psychotic disorders. However, the relationship between the continua and frank psychosis is substantiated by the empirical resemblance of their correlates.[73,74]

Emotional instability may provide routes linking prior experience to the development of psychosis. Our finding that an external event (CSA) may be linked to subsequent psychosis by its capacity to induce MI is of particular interest. There is evidence for this mechanism from ESM studies: enhanced reactivity to current stresses is associated with increased psychotic experiences, and current reactivity is enhanced in people with a history of early adversity.[60,63] Mood dysregulation may also lie behind the inconsistent behavior of abused people that leads to revictimization and further risk of abuse[39,75] and to the increased propensity for suicide in people with psychosis.[76]

Our findings have potential clinical relevance. Thus, direct therapeutic targeting of MI may reduce the propensity to recrudescence of psychotic symptoms. In particular, cognitive behavioral techniques aimed at emotional regulation in psychosis should be investigated further. Examples might include decatastrophizing fluctuations in mood, learning to tolerate acute negative emotions, switching attention away from the self to external activity, improving sleep, developing regular meal times, and improving diet.

Conclusions

Our results indicate a strong and consistent signal linking MI to psychotic experience. This association adds to the boundary problems and comorbidity between nonaffective psychosis, schizoaffective disorder, bipolar conditions, and borderline personality disorder.[77–79] However, it is not clear what the signal actually portends. MI may be an epiphenomenon, but it might equally have a central role in the formation and maintenance of symptoms of psychosis. If so, it could provide explanations both for the origins of psychotic phenomena and for the fluctuations so commonly observed in them. Our findings are sufficiently interesting and robust to justify detailed investigation of the phenomenology of MI in people with psychosis, the temporal relationship between mood changes and psychotic experiences, and whether the relationship is limited to particular psychotic symptoms. Mechanisms can then be examined more closely and the role of emotional reactivity disentangled by trials of targeted cognitive behavioral treatments.

Supplementary Material

Supplementary material is available at http://schizophreniabulletin.oxfordjournals.org.

Funding

The 2000 survey was funded by the English Department of Health, the Scottish Executive, and the National Assembly for Wales. The 2007 survey was funded by the NHS Information Centre for Health and Social Care.

Acknowledgments

None of the authors has conflicts of interest to declare, financial or otherwise. P.B. is emeritus, the other authors are employed by their respective universities. E.K. acknowledges the support of the Biomedical Research Centre, South London and Maudsley NHS Foundation Trust.

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