The Effect of State Anxiety on Paranoid Ideation and Jumping to Conclusions. An Experimental Investigation

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Theoretical models of persecutory delusions have emphasized the impact of reasoning biases and negative emotion at the early stages of symptom formation. However, the causal mechanisms remain unclear. This study tests the hypothesis that state anxiety will increase paranoid ideation and that this increase will be moderated by the level of individual vulnerability and mediated by the tendency to jump to conclusions. Healthy participants (n = 90) with varying levels of vulnerability (psychosis symptoms assessed by the Community Assessment of Psychic Experiences) were randomly assigned to either an anxiety or a nonanxiety condition. Anxiety was induced by pictures from the International Affective Picture System and by in-sensu exposure to individual anxiety-provoking situations. During each condition, symptoms of paranoia were assessed by a state-adapted version of the Paranoia Checklist. Jumping to conclusions (JTC) was assessed using a modified version of the beads task. Overall, participants in the anxiety condition reported significantly more paranoid thoughts and showed more JTC than participants in the neutral condition. Participants with higher baseline vulnerability were more likely to show an increase in paranoia as reaction to the anxiety manipulation. Moreover, the association of anxiety and paranoia was mediated by the increased tendency to jump to conclusions in the beads task. The results are in line with a threat anticipation conceptualization of paranoia and provide evidence for an interaction of anxiety and reasoning biases in the development of paranoid beliefs. A combination of meta-cognitive training directed at reasoning biases and promoting emotion regulation skills might prove beneficial in preventing symptoms.

Key words: paranoia/vulnerability/emotion/anxiety/reasoning biases

Introduction

Delusions of persecution and reference are the most frequent symptoms in schizophrenia,1,2 and their continuity across the population is well established.3 Based on basic research findings, Freeman and colleagues have proposed a threat anticipation model, in which paranoid delusions are hypothesized to arise from an interaction of vulnerability, emotional processes, and reasoning biases.4,5

For one, this model proposes that reasoning biases will increase the likelihood of interpreting ambiguous experiences in a delusional manner. This is backed up by empirical evidence linking persecutory delusions to various reasoning biases. In particular, the finding that persons with delusions request fewer pieces of information in decision-making tasks, first identified by Huq et al.,6 is now well replicated.9 Numerous studies using the beads task find approximately 50%–60% of the patients to decide on the basis of 1 or 2 beads compared with 20%–30% in healthy samples.4,8,9 This tendency to jump to conclusions has occasionally also been demonstrated in psychosis-prone healthy individuals.10–13 Nevertheless, its proposed causal role is still a topic of ongoing debate.9

Furthermore, the threat anticipation model proposes that state anxiety will be misinterpreted by the individual as evidence for objective threat. Despite the centrality of emotional processing, there has been a surprising dearth of empirical work examining emotion in schizophrenia,14 and even less work focused on emotions with regard to delusion formation. Some correlation studies provide evidence for associations between anxiety and paranoid thoughts or conviction levels in patients and healthy individuals.15–18 However, this research suffers from the usual problem of assessing causality from correlational data. The few experimental studies conducted so far support the notion that anxiety could play a causal role in delusion formation rather than merely arising as a consequence of symptoms. In a previous study within a healthy sample, we found an increase in paranoia to arise from a noise stressor, in particular for those with high baseline

Footnotes:

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vulnerability. In addition, we found state anxiety following the stressor to be associated with increased paranoid beliefs.\textsuperscript{19} Similarly, the Camberwell walk study\textsuperscript{20} found a brief exposure to an urban environment to increase anxiety and paranoid beliefs in patients with preexisting delusions. Further research found speech of patients with schizophrenia to become more disordered when negative affect was induced.\textsuperscript{21,22} So far though, no study has directly manipulated anxiety to test its impact on paranoid beliefs.

Moving beyond the separate emotional and cognitive routes to paranoia proposed by the threat anticipation model, it is hypothesized that anxiety and jumping to conclusions (JTC) will interact in their impact on paranoia. Higher state anxiety is likely to deter people from extensive processing and evoke “quick and dirty” decisions, such as judging on the basis of little evidence. Accordingly, the Camberwell walk study\textsuperscript{20} found an increase in JTC after exposure to the urban environment, which may have been due to the increased anxiety. Two studies have directly investigated the effect of anxiety on reasoning biases. Mujica-Parodi et al\textsuperscript{23} found that healthy subjects, when aroused, tended to slightly restrict the amount of available information, a pattern they interpreted as being similar to the JTC bias. So et al\textsuperscript{24} investigated the effect of induced anxiety on the data-gathering bias in patients with delusions and healthy controls, finding no increase. However, interpretation was limited due to an insufficient effect of the anxiety induction. No study has investigated interaction effects of anxiety and JTC on paranoia although it seems intuitive to expect that threat-related thoughts, triggered by state anxiety, will be more rapidly accepted in the presence of an increased tendency to jump to conclusions.

The present study tests the impact of state anxiety on paranoia and data gathering in a randomized experimental design. We studied a sample of healthy individuals with varying levels of vulnerability to psychosis, arguing that research in populations that are not yet affected by severe psychotic symptoms and medication is necessary to improve our understanding of the psychological processes involved in the early stages of transition into psychosis. This approach appears justified by the continuity not only of paranoid symptoms but also of associated reasoning biases and emotions in the population.

It is hypothesized that (1) participants will display higher levels of paranoia in the anxiety compared with the neutral condition, (2) the association between anxiety and paranoia will be moderated by the extent of vulnerability, and (3) the association between anxiety and paranoia will be mediated by an increase in JTC.

**Methods**

**Participants**
The sample consisted of 90 students of psychology, educational sciences, or social sciences. The mean age of the sample was 23.5 years (SD = 2.3, range = 19–30), and 56% of the participants were female. Participants were paid or were able to complete curriculum requirements by participating.

**Measures**

Baseline vulnerability to psychosis was assessed with the Community Assessment of Psychic Experiences (CAPE),\textsuperscript{25} the rationale being that low-grade psychotic, negative, and depressive symptoms are frequently used as low-level criterion in high-risk studies.\textsuperscript{26,27} The CAPE is a 42-item self-report instrument developed to rate lifetime psychotic experiences. The CAPE is partly based on the 21-item Peters et al Delusions Inventory\textsuperscript{28} that was derived from the Present State Examination.\textsuperscript{29} The items are rated on 4-point scales with regard to frequency (from never to nearly always) and distress (from not distressed to very distressed). A 3-factor structure of positive, negative, and depressive dimensions has been demonstrated.\textsuperscript{25} The CAPE provides a total score per dimension by adding up the scores on the frequency question, from which a weighted mean is calculated that accounts for partial nonresponse.\textsuperscript{30} Validation studies of the CAPE within large healthy samples demonstrate high correlations between CAPE scores and observer-rated symptom and schizotypy scales and good test reliability.\textsuperscript{30} Studies within healthy and clinical samples have found participants with schizophrenia to obtain higher mean scores in the positive, negative, and depression dimensions,\textsuperscript{31} ranging from 2.1 to 2.4\textsuperscript{32} than healthy controls whose scores were found to range from 1.4 to 1.8.\textsuperscript{30,32} The German version of the CAPE has good-to-excellent internal consistency (Cronbach $\alpha = .94$ for the total scale and .84, .89, .91 for the positive, negative, and depression subscales, respectively) and discriminative validity.\textsuperscript{33}

**Paranoia Checklist.** The Paranoia Checklist\textsuperscript{34} is an 18-item self-report scale developed to measure paranoid ideation. It includes items assessing ideas of persecution (eg, “I need to be on my guard against others”) and reference (eg, “There might be negative comments being circulated about me”) each rated with regard to frequency, conviction, and distress on 5-point Likert scales. The Paranoia Checklist has excellent internal consistency (Cronbach $\alpha > .90$) and good convergent validity, and previous studies have demonstrated its sensitivity to paranoid ideation in healthy populations, with 40% expressing frequent ideas that negative comments are being circulated and about 20% expressing frequent paranoid ideas about being observed or followed.\textsuperscript{34} These rates were 60% and 68% in patients with present delusions.\textsuperscript{35}

For the purpose of this study, we adapted the Paranoia Checklist by asking participants to rate the extent to which the items apply “at the moment” on a 5-point scale...
ranging from 1 (not at all) to 5 (very strongly). The complete score is obtained by summing up the items and can range from 18 to 90. This state-adapted German version has demonstrated high internal consistency and convergent validity in a previous study, with participants' scores ranging from 18 to 39 (mean $= 24.91$, SD $= 5.39$).19

JTC was assessed using a revised and computerized version of the beads task6 developed by Todd S. Woodward and the final author.36 Participants were shown 2 lakes with red (R) and gray (G) fishes in opposing ratios (80% R and 20% G). Fish were caught one by one from 1 of the 2 lakes only, and it was emphasized that the lakes were never switched. Participants were asked to decide from which lake the fish were caught, keeping in mind that every fish caught would be thrown back into the same lake. Participants were allowed to request as many fish as they wanted before deciding. Instructions were read by the experimenter from the monitor. Conversation between participants and experimenter during the tasks was limited to comprehension questions. In total, 10 fish were drawn in a seemingly random but actually predetermined order, whereby one lake was strongly suggested by the chain of events, and the sequence was R-R-R-G-R-R-R-G-R. Consistent with many of the recent beads task studies,12,24 we used draws to decision (DTD, the number of fish a participant required before deciding) and JTC (making a decision after the first or second fish) as outcome measures for this task. DTD has the advantage of being continuous, whereas the dichotomization of this variable is a more direct measure of JTC as such.

**Design and Procedure**

The experiment was conducted as a randomized group comparison design. The experiment took place at a desk with a computer in a small quiet office without windows. Before the experiment, participants were informed that the study was being conducted to test information processing and were asked to complete the CAPE and the items to assess mood states (as described below). They were then randomized to an anxiety vs nonanxiety condition by the experimenters who drew lots from a jar containing an equal number of lots for each condition (figure 1).

Anxiety was induced in 3 ways: (1) the room was darkened during the complete experiment; however, a desk lamp remained on to secure sufficient light to complete the questionnaires and tasks; in the neutral condition, an additional ceiling light was turned on; (2) the participant viewed 16 anxiety-inducing pictures from the International Affective Picture System (IAPS);37 pictures with negative valence ($M = 2.5$, SD $= 0.87$) and high arousal ($M = 6.8$, SD $= 0.35$) as well as previously demonstrated successful anxiety induction38,39 were selected for this purpose. Each picture was presented for 6 seconds and was then supplemented by an associated anxiety-inducing question (e.g., How many people are murdered in Germany each year?) followed by a black screen for

![Fig. 1. Design.](https://academic.oup.com/schizophreniabulletin/article/36/6/1140/1890653)
3 seconds after the question had been answered by the participant. In the neutral condition, pictures with a neutral valence (M = 5.0, SD = 0.30) and low arousal (M = 3.1, SD = 0.53) were selected, followed by neutral questions (eg, Which is the highest temperature a light bulb can reach?). In each condition, 4 neutral pictures of persons followed by paranoia-relevant questions (eg, Is this person trustworthy?) as well as a brief Implicit Association Test (Greenwald et al 1998)40 were included, with the aim of developing implicit measures of paranoia. The results of these measures will be reported elsewhere (J. Lange, J. Burau, T. M. Lincoln, PhD, in preparation, 2008); (3) participants in the anxiety condition were asked to describe a personally relevant anxiety-inducing situation. They were then instructed to close their eyes and were exposed to this situation in a room by a trained experimenter who used a script of detailed questions aimed at activating different senses (eg, What do you see? hear? smell?). The exposure was terminated after approximately 5 minutes by instructing the participant to open his or her eyes and come back into the presence of the room. In the neutral condition, participants were instructed to imagine biting into a lemon.

The effect of the anxiety manipulation on emotion was assessed by a brief visual- analog self-report scale of mood states that was completed at 3 time points (after entering the room, after viewing the IAPS pictures, and after the insensu exposure). This scale consisted of five 11-point unipolar and four 11-point bipolar intensity ratings. Anxiety was captured by the items “fear” (labels: frightened, timid, afraid, scared), “tension” (labels: nervous, restless, tense, wound up vs calm, relaxed, placid, at ease), and “subjective impression of heartbeat.” Other negative emotions were captured by the items “anger” (labels: angry, annoyed, mad, sore), “shame” (labels: embarrassed, ridiculed, ashamed, foolish), and “sadness” (labels: sad, depressed, miserable, deserted). Finally, cognitive state was captured by the items “alertness” (labels: alert, attentive, receptive, lucid vs confused, baffled, perplexed), “interest” (labels: curious, interested, motivated vs bored, indifferent, dull), and “activation” (energetic, active, animated, lively vs tired, fatigued, sluggish, exhausted). The items have been validated in other experimental designs.41 In addition, at the end of the experiment, participants completed a feedback questionnaire containing questions on the emotional atmosphere during the experiment (relaxing, eerie, friendly, threatening, agreeable, worrying, trustworthy, hostile) on 8-point Likert scales (eg, “The atmosphere during the experiment was relaxing”) as well as an open question to control whether participants were able to see through the purpose of the experiment (“What do you think was the purpose of the experiment?”).

The experiment lasted for 1 hour and included the anxiety induction procedures, the clinical scales, reasoning task, and the mood and symptom ratings as presented in figure 1. Despite the disadvantage of risking sequence effects, we decided to use a fixed order because the interpretation of results with regard to the implicit measures (J. Lange, J. Burau, T. M. Lincoln, PhD, in preparation, 2008) requires that participants are tested under the same conditions.

**Strategy of Data Analysis**

First, we compared the experimental and control group with regard to baseline differences in symptoms and sociodemographic variables using t tests for independent data. We then tested the success of the anxiety induction by comparing the mood state ratings at baseline and after anxiety inductions between the anxiety and the neutral condition using repeated-measures analysis of variance (ANOVA). t Tests for independent data were applied to test for the effect of the anxiety manipulation on paranoia and reasoning and multiple regression analyses to test for moderating and mediating effects. Post hoc plotting was used to interpret significant interactions,42 and the Sobel test43 was used to test for the final mediation effect. All predictors were centered around the grand mean by subtracting the mean score from each case. The analyses were carried out using SPSS version 17.

**Results**

**Group Differences at Baseline**

The mean CAPE scores for the positive, negative, and depressive dimensions, respectively, were 1.49 (SD = 0.26), 1.97 (SD = 0.36), and 1.89 (SD = 0.33), which are at the higher end of the range found within healthy populations in other studies.30,32 There were no significant differences in regard to the CAPE dimensions, age, or gender between the groups at baseline (all P > .3). The baseline CAPE–positive score was significantly correlated with JTC across both conditions (r = .21, P ≤ .05) but not with the total DTD in the beads task.

**Manipulation Check**

We used a 3 (time: baseline, following IAPS, following insensu exposure) × 3 (mood state: anxiety, other negative moods, cognitive state) mixed ANOVA with experimental condition as between-subject factor to compare the mood states between the conditions at the different assessment times. There was a significant interaction effect for time × group × mood state, F(2,85) = 19.33, P ≤ .001. Post hoc t tests revealed no significant differences in anxiety, other negative emotions, or cognitive state at baseline. After viewing the IAPS pictures, participants in the anxiety condition reported significantly more anxiety (P ≤ .001) than participants in the neutral condition while the groups did not differ in their intensity of other negative emotions or in cognitive states. After the insensu exposure, the participants in the anxiety condition reported not only significantly more anxiety than...
participants in the neutral condition \((P \leq .001)\) but also more other negative emotions \((P \leq .001)\) as well as feeling less activated, motivated, and interested \((P \leq .001)\). Figure 2 displays the reported intensity of anxiety and other negative emotions at the different time points within the neutral and the anxiety condition.

In the feedback questionnaire at the end of the experiment, participants in the anxiety condition described the atmosphere during the experiment as significantly less relaxing \((P \leq .01)\), more eerie \((P \leq .01)\), more threatening \((P \leq .01)\), less agreeable \((P \leq .01)\), more worrying \((P \leq .01)\), and less trustworthy \((P \leq .01)\), while groups did not differ with regard to perceived hostility or friendliness. Also, 6 participants in the neutral condition and 3 in the anxiety condition concluded that the experiment was somehow connected to do with paranoia. This difference was not significant \((\chi^2 = 1.11, df = 1, P > .2)\).

In sum, the anxiety condition was effective in increasing anxiety. However, as other emotions also increased, the effect of these emotions on the dependent variables ( DVs) will need controlling.

### Direct Effect of Anxiety on Paranoia and Cognitive Biases

A direct effect of the anxiety induction could be demonstrated by the significantly higher mean score of the state-adapted Paranoia Checklist in the anxiety \((M = 31.7, SD = 11.0)\) compared with the neutral condition \((M = 25.8, SD = 6.2, T = -3.2, df = 69.3; P \leq .001; d = 0.66 [confidence interval = 0.24–1.09])\). In addition, participants in the anxiety condition showed more JTC \((51\%)\) than participants in the neutral condition \((24\%, \chi^2 = 6.8, P \leq .01)\) but not fewer DTD \((M = 3.6, SD = 2.6, range = 1–11, vs M = 4.6, SD = 3.0, range = 1–11, respectively; T = 1.7, df = 88; P = .103)\).

In order to assess whether the intensity of anxiety after the in sensu exposure is uniquely associated with paranoia and JTC, rather than other negative emotions or cognitive states, we conducted 2 regression analyses (Method: STEPWISE) using the Paranoia Checklist score, JTC and DTD as DVs, and anxiety, other negative emotions, and cognitive state as predictors. Anxiety emerged as single significant predictor for paranoia \((\beta = .54, t = 6.06, P \leq .001)\) and JTC \((\beta = .35, t = 3.45, P \leq .001)\); neither emotional nor cognitive states predicted DTD.

### Moderating Effect of Vulnerability

To assess whether the increase in paranoia in the anxiety condition is moderated by baseline vulnerability, we conducted a linear regression analysis (Method: ENTER) with the Paranoia Checklist score as DV, entering the experimental condition (anxiety vs neutral), the CAPE baseline scores, and the interaction term condition \(\times\) CAPE. The results are displayed in the top part of table 1. The regression equation explained a significant amount of the variance of the DV, with each predictor contributing significantly. In order to help with the interpretation of the interaction, we calculated the effects of condition on paranoia at different levels of the moderator (CAPE) for which we used the bottom quartile (low range), the second and third quartiles (medium range), and the fourth quartile. The mean scores on the Paranoia Checklist in the anxiety and the neutral condition for these 3 levels of the CAPE are depicted in figure 3. The anxiety manipulation resulted in higher scores in the Paranoia Checklist in individuals with higher baseline vulnerability \((\beta = -.59, t = 3.39, df = 22, P \leq .01)\) but not in individuals with medium \((\beta = .252, t = 1.66, df = 42, P = .104)\) or low levels of baseline vulnerability \((\beta = .10, t = 0.45, df = 22, P > .3)\).
Mediating Effect of Cognitive Biases

A mediation effect occurs when (1) the independent variable (IV) significantly affects the mediator, (2) the IV significantly affects the DV in the absence of the mediator, (3) the mediator has a significant effect on the DV, and (4) the effect of the IV on the DV shrinks upon the addition of the mediator to the model (Muller et al. 2005).44 The required regression analyses to test the mediation effect are depicted in table 1. It can be seen, that the experimental condition (IV) had a significant effect on JTC (mediator). Thus, precondition (1) is fulfilled. The significant beta coefficient for condition (IV) as a predictor of paranoia (DV) demonstrates precondition (2), and the significant beta coefficient for JTC in predicting paranoia demonstrates precondition (3). In the multiple regression of condition and JTC on paranoia, it can be seen that the direct effect of condition on paranoia, although significant, was reduced by entering JTC into the model, which demonstrates precondition (4). In addition to demonstrating the heuristic conditions, we used the Sobel test to formally assess the mediation effect. The Sobel test statistic was significant (z = 2.92, P ≤ .01), supporting an indirect effect of anxiety on paranoia via JTC. The mediation effect is depicted in figure 4.

Additional Analyses

The primary analyses testing the effect of the experimental condition on paranoid beliefs and reasoning were repeated excluding the 9 participants who inferred that the study was about paranoia. The group differences remained significant for the Paranoia Checklist (P ≤ .01) and JTC (P ≤ .001). In addition, the group difference in DTD reached significance in this sample (P ≤ .05).

Discussion

The study set out to investigate the causal links proposed by the threat anticipation model of persecutory delusions5 as well as interaction effects of anxiety and reasoning biases on paranoia. Several significant findings emerged: Induced state anxiety lead to an increase in state paranoia and JTC in healthy individuals. The association between anxiety and paranoia was moderated by the extent of baseline symptoms and mediated by an increase in the tendency to jump to conclusions.

The notion that anxiety provides fertile soil for paranoid thoughts fits in well with results from basic research demonstrating that anxiety tends to narrow attention to the emotionally relevant cues.45,46 The findings are also in line with studies using the experience sampling method47 and other experimental studies demonstrating affective state and affective reactivity in patients to be associated with the severity of psychotic symptoms.19,21,48 In contrast to these study designs, our study directly manipulated anxiety. By doing so, we could demonstrate that the increase in paranoid thinking seems to arise from anxiety and that anxiety is not merely a reaction to paranoid thoughts.

The finding that persons with higher baseline symptoms were more likely to react to state anxiety with an increase in paranoia is consistent with a vulnerability stress notion of psychosis.49 It also provides further evidence for the relevance of subclinical symptoms as a vulnerability marker, as demonstrated in previous studies, in which vulnerable individuals were more likely to react to stress with an increase of paranoia.19
As hypothesized, participants in the anxiety condition jumped to conclusions more often than participants in the non-anxiety condition, which extends findings from the Camberwell walk study20 and Mujica-Parodi et al.23 Despite this, the total number of DTD was not significantly affected by the experimental condition, although the trend in the expected direction reached significance when participants who realized that the experiment was about paranoia were excluded. Possibly, these participants were characterized by more conservative response styles. In addition, it can be speculated that within some individuals the increase in general negative affect, which has been found to be characterized by more systematic, detail-oriented information processing strategies,50 might have had a counterbalancing impact on the reasoning produced by anxiety. However, a valid test of differential effects of mood states on DTD would require a direct comparison of specific mood inductions. The data clearly underline the relevance of taking interactions between emotions and reasoning processes into account in explaining the development of delusions. The significant mediating effect of JTC in the association of anxiety and paranoid thoughts is in accord with our assumption that threat-related thoughts, triggered by state anxiety, will be more rapidly accepted in the presence of a tendency to jump to conclusions.

So far, few studies have integrated emotion and cognition in schizophrenia research.51 Findings from basic research have demonstrated state anxiety to impair performance accuracy on a variety of tasks due to decreased allocation of attentional resources and a reduced range of cue utilization.46 The interplay of reasoning biases and emotions also makes sense if one considers that regulating emotional responses as well as developing and adjusting strategies for the cognitive reasoning tasks both involve monitoring and correction processes. Although the neural basis of emotion regulation is less well understood than the neural basis of cognitive control, similar frontolimbic networks are implicated. Specifically, efforts at downregulating anxiety responses recruit both the prefrontal cortex and the anterior cingulate cortex,52 and these regions have also been demonstrated to be closely involved in implementing cognitive control.53 Accordingly, studies investigating error monitoring and stress regulation have supported the notion that cognitive control and emotion regulation depend on common or interacting systems.54 Thus, it can be assumed that under the condition of heightened state anxiety vulnerable individuals will be unable to concurrently maintain cognitive and emotional control. They will thus be at risk for paranoid ideation due to limited information processing capacity.

Strengths and Limitations

The study design is characterized by a number of strengths, including the use of a randomized design and the manipulation of state anxiety. Unfortunately, the in sensu induction method increased negative emotions in general, not just anxiety. The increase in other negative mood states, such as sadness, might also explain why participants in the anxiety condition reported significantly less activation than controls, which contradicts the physiology of anxiety. Although the regression on paranoia and reasoning biases clearly underlined the relevance of anxiety over and above other emotional states, a unique impact of our induction method on anxiety would have provided a more straightforward interpretation.

The sample of healthy participants, rather than participants with psychosis, was purposely chosen in order to demonstrate basic mechanisms on the pathway from vulnerability to psychosis. Limitations might be seen in the use of a student sample due to the concern that there will be insufficient variance of psychosis symptoms. However, students have been found to reveal slightly higher levels of subclinical psychotic symptoms,55 and in this sample the CAPE subscale scores were at the high end of the range found in population samples.30 Thus, there was a representative number of high scorers in the sample, although these scores were still below scores obtained in patient samples.32 The use of the CAPE as a measure of subclinical psychotic symptoms to assess vulnerability seems justifiable because attenuated subthreshold psychotic symptoms have been described as risk factors, emerging before first episodes or psychotic relapse.56,57 Moreover, low-grade psychotic, negative, and depressive symptoms, as assessed in the CAPE, are frequently used as low-level criterion in high-risk studies.26,27 Nevertheless, it is agreed that using multiple indicators of risk, eg, by adding attenuated positive symptoms, functional decline, and genetic risk, is likely to yield higher predictive values (close-in strategy).58

Another question is whether paranoid beliefs as assessed in the Paranoia Checklist are comparable to those experienced by patients. Given the pervasiveness of subclinical paranoia in the general population, it is plausible that only a unique subset of individuals experience the type of paranoia that is continuous with paranoia in schizophrenia. Nevertheless, the Paranoia Checklist has been demonstrated to be a fairly valid self-report measure of paranoia in schizophrenia. A correlation of r = .52 between the Paranoia Checklist and observer-rated delusions in the Positive and Negative Syndrome Scale59 (Lincoln, Ziegler, Lüllmann, Müller, and Rief, PhD, 2009) underlines the assumption that the same or at least strongly overlapping constructs are being assessed.

Implications

This study investigated the processes at a subclinical level, seeking to identify mechanisms that are involved in transition from healthy to psychotic states. It must be noted that the effects of anxiety might be different in people with psychosis. For example, there might be
ceiling effects of the higher rates of anxiety typical of psychotic populations so that further increases do not have the same impact on paranoia or reasoning. Also, limited cognitive capacity might have more impact on reasoning biases in clinical samples and possibly render the additional effect of state anxiety nonsignificant. Thus, whereas this study cannot provide answers to symptom exacerbation in persons who already have schizophrenia, it provides us with an idea about how symptoms may evolve at earlier stages and with better knowledge about how these processes are helpful in order to develop specific preventive interventions.

The results suggest that meta-cognitive training targeted at reasoning biases can be one effective way to prevent symptom development and exacerbation. In addition, if abnormalities in the capacity to downregulate negative affect are crucial aspects of vulnerability to psychopathology, it follows that more effective regulation of anxiety could be another key to symptom prevention. This could be promoted by encouraging a stronger acceptance of negative affect as proposed by mindfulness. Although effective emotion regulation also involves the ability to refocus attention to less overwhelming stimuli, the use of distraction techniques has not been proven helpful in other emotional disorders. Rather, successful emotion regulation involves reappraisal. Thus, supporting vulnerable individuals to question and challenge dysfunctional appraisals of negative mood states might prove helpful.

References
