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Personalizing and externalizing biases in deluded and depressed patients: Are attributional biases a stable and specific characteristic of delusions?

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Objectives. The purpose of this study was to explore whether explicit and implicit attributional styles of delusional patients were associated to their clinical state, and whether attributions biases are specific to delusional psychopathology or also appear in other disorders (i.e. depression).

Design and methods. A cross-sectional design was used. The sample consisted of 136 participants (40 acute deluded participants, 25 remitted deluded participants, 35 depressed patients and 36 normal controls). The Internal, Personal and Situational Attributions Questionnaire (IPSAQ) and the Pragmatic Inferential Test (PIT) were used to assess explicit and implicit attributional style, respectively.

Results. All participants, with the exception of the depressed patients group, showed an externalizing bias (EB) for negative events. Although both acute and remitted deluded patients showed a similar overall pattern of explicit attributions, the personalizing bias (PB) was significantly greater in the acute group. The magnitude of this bias, which was also found in the depressed patients, was significantly related to the patient's degree of severity, as assessed by the total BPRS score ($r = .45$, $p < .001$). The results on the implicit attributions were more equivocal, perhaps due the low reliability of the PIT.

Conclusions. Attributional biases seem to be a stable characteristic of delusions. Yet, the PB might be a rather unspecific characteristic that varies with the degree of the severity of psychopathology. The implications of these findings for understanding the role of attributional biases in depression and delusion formation are discussed.

Researchers are increasingly interested in psychological processes involved in social cognition of psychotic patients (e.g. Penn, Corrigan, Bentall, Racenstein, & Newman, 1997). In the case of delusions, the presence of various cognitive biases is well documented. For example, selective attention to threat-information (Kaney, Wolfenden,

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Dewey, & Bentall, 1992; Kinderman, 1994), difficulties in inferring others' mental states (i.e. deficit on theory of mind, Frith, 1994), a preference for remembering material related to the theme of the delusion or data-gathering biases have all been found in deluded patients (reviewed in Garety & Freeman, 1999; Bentall, 2003).

Attribution biases have also been found to be particularly relevant in the research of delusional thinking. In fact, the social attributional theory has been applied to the study of delusional thinking, showing that paranoid deluded patients tend to externalize causal explanations for negative events. Causal reasoning has been typically studied using tasks in which subjects are required to give a causal explanation for hypothetical events (positive and negative). In a seminal work, Kaney & Bentall (1989) used the Attributional Style Questionnaire (ASQ; Peterson *et al.*, 1982) to study how depressed, deluded and control people inferred causes of hypothetical events. As expected, they found that depressed patients showed an internal, stable and global pattern for negative events. In contrast to depressed patients, deluded participants tended to make global, stable and external attributions for negative events, and internal attributions for positive events, in the same direction as normal people but showing an even more exaggerated *self-serving bias* (Taylor, 1988). This pattern of results for hypothetical negative events has been replicated by some studies also using the ASQ (Candido & Romney, 1990; Kinderman, Kaney, Morley, & Bentall, 1992), although the finding that deluded patients tend to make internal attributions for positive events has not been observed in other studies (Fear, Sharp, & Healy, 1996; Lyon, Kaney, & Bentall, 1994; Sharp, Fear, & Healy, 1997).

All these previous studies focused on assessing explicitly (or overt) causal explanations with the ASQ or similar questionnaires in which subjects are explicitly asked about attributions for positive and negative events. Yet, some authors suggest that the self-serving attributional style found in paranoid patients might act as a defence for an implicit negative self-schema (Bentall, Kinderman, & Kaney, 1994). In an effort to analyse implicit (or covert) attributions, Lyon *et al.* (1994) employed the ASQ-parallel form (ASQpf) to assess explicit attributions and the Pragmatic Inferential Test (PIT; Winters & Neale, 1985) to assess implicit attributions. Whereas with the ASQpf, the results were consistent with previous studies (i.e. deluded and normal subjects showed a self-serving bias for negative events in contrast to depressed patients, who tended to blame themselves), the authors found that both deluded and depressed patients showed a similar depressive attributional pattern for negative events when assessed with the implicit task (i.e. PIT). Despite this interesting finding, there have been few studies that have assessed covert attributions and, furthermore, the results have not been consistent (i.e. Krstev, Jackson, & Maude, 1999; Martin & Penn, 2002; Peters, Day, & Garety, 1997; Winters & Neale, 1985).

Focusing on the importance of interpersonal relations in delusional themes, Kinderman and Bentall (1996) developed a causal reasoning assessment instrument to distinguish between other-referent external attributions and external attributions about circumstances or chance. Thus, these authors developed the Internal, Personal and Situational Attributions Questionnaire (IPSAQ), which is based on the ASQ (Peterson *et al.*, 1982). However, whereas the ASQ covers only two causal loci (internal versus external), the IPSAQ covers three (internal, external-personal and external-situational or circumstantial), thus improving one of the limitations of the ASQ (Reivich, 1995). Kinderman and Bentall (1997) used the IPSAQ to assess the causal reasoning pattern in deluded patients, depressed patients and normal population. Their results confirmed the presence of an externalizing bias (EB) in deluded patients for negative events, similar

to the control group and, moreover, they found than only the deluded patients tended to attribute negative events to external-personal causes (i.e. blaming others) instead of making external-situational causal attributions, as did the control group (normal population). Kinderman and Bentall called this causal reasoning pattern the *personalizing bias*.

As a result of the studies on implicit and explicit attributions, a cognitive model of paranoia has been postulated that posits that the EB for negative events shown by deluded patients reflects an attempt to protect self-esteem by avoiding access to implicit negative self-schemata (Bentall, 2003; Bentall, Corcoran, Howard, Blackwood, & Kinderman, 2001; Bentall *et al.*, 1994), which had been suggested in some earlier theories (Zigler & Glick, 1988). Although this model has proven to be fruitful for understanding the delusional phenomena, there are several limitations concerning the empirical support on which this model stands. First, it is surprising that there are only two studies with patients that have used the IPSAQ to assess causal reasoning pattern. Kinderman and Bentall (2000) only used the negative items from the IPSAQ in a sample of students; Blackshaw, Kinderman, Hare, and Hatton (2001) administered the test to patients with Asperger's syndrome; Taylor and Kinderman (2002) used it in a sample of university students, as did Martin and Penn (2001) and Combs and Penn (2004), who used this instrument with a non-clinical sample of students high and low in paranoia. In fact, the Kinderman and Bentall (1997) study is the only one, as far as we know, in which the so-called personalizing bias (PB) is directly associated with delusions and not with some other kind of pathology (i.e. depression). In a second study in which they used the IPSAQ on a clinical population, Martin and Penn (2002) compared the attributional style of a group of deluded patients (paranoid versus non-paranoid), confirming the presence of the PB in both types of delusions. The second limitation is the scant amount of literature that uses the PIT to implicitly assess causal reasoning in deluded patients. Moreover, as mentioned above, the results are inconsistent. Third, we would like to underline that none of these works has consistently examined whether the biases found in deluded patients' causal reasoning (i.e. EB and PB) depends on the patient's clinical state or whether, conversely, these biases are a stable characteristic of delusional phenomenology, regardless of the presence of symptomatology (all the studies reviewed above analyse the pattern of causal reasoning in acute deluded patients). The only exception is the study of Krstev *et al.* (1999) in which the attributional style was examined – using PIT and the ASQpf – in a group of patients who had experienced their first psychotic episode, 6 months after symptom remission, but this study did not include a control group. Although the authors confirmed the presence of the EB when they used explicit measures of causal reasoning, they obtained inconsistent results in attributional style assessed with the PIT because they did not find that deluded patients had a higher tendency of internal attribution for negative events. In fact, the causal reasoning pattern of the PIT is similar to that found in non-clinical control groups in other studies (e.g. Lyon *et al.*, 1994; Sharp *et al.*, 1997).

In the light of these findings, it seems that the only consistent result concerning the causal reasoning pattern in deluded patients is the presence of an EB for negative events. In fact, more recent studies have continued to confirm its presence in deluded patients using other assessment strategies (e.g. qualitative methods; Beese & Stratton, 2004; Craig, Hatton, Craig, & Bentall, 2004; Lee, Randall, Beattie, & Bentall, in press).

On the basis of previous findings and of the still limited support of studies on causal reasoning in delusions, the goals of this work are: (a) to determine whether the attributional style (explicit or implicit) of deluded patients reported in the literature is

only associated with their clinical state, or whether it is stable and independent of the presence of symptomatology. For this purpose, we analyse and compare, for the first time in the same study, the pattern of causal reasoning in a group of remitted deluded patients with that of a group of acute deluded patients; (b) to confirm the presence of a personalizing attributional bias in deluded patients, for which we used the same methodology as the one used in the original work of Kinderman and Bentall (1997) in an attempt to be the first authors to replicate their methodology; (c) to analyse whether these biases are specific to deluded symptomatology or whether, contrariwise, they are also present in other kinds of pathologies. For this purpose, a group of depressed patients and a normal non-clinical control group were included in the study.

Method

Participants

There were four groups of participants:

(1) *Acute deluded group*. It comprised 40 people (27 men and 13 women) receiving psychiatric treatment and currently suffering paranoid delusions as assessed by the present state examination (PSE-10, SCAN, Sections 18 and 19, WHO 1992). Participants with delusions of guilt were excluded, as these contents are usually associated with major depressive disorders with psychotic characteristics. Psychiatric diagnoses were obtained from hospital records and clinicians' casenotes. According to the *Diagnostic and Statistical Manual of Mental Disorders* (fourth edition, text revision, American Psychiatric Association, 2000), patients met the diagnostic criteria for the following categories: paranoid schizophrenia ($N = 26$), schizoaffective disorder ($N = 3$), brief psychotic disorder ($N = 9$) and bipolar disorder ($N = 2$). All patients were receiving antipsychotic medication at the time of the study.

(2) *Remitted deluded group*. This group comprised 25 people (21 men and 4 women) who had suffered in the past, but not within the last 6 months, from paranoid delusions according to the PSE interview and clinical records. All participants were recruited from out-patient clinics and from psychosocial rehabilitation centres. According to DSM-IV-TR criteria, the patients had been diagnosed in the following diagnostic categories: paranoid schizophrenia ($N = 19$), schizoaffective disorders ($N = 2$) and bipolar disorder with history of paranoid delusions ($N = 4$). All participants were still receiving antipsychotic medication.

(3) *Depressive disorder group.* This psychiatric control group comprised 35 patients (9 men and 26 women) who met DSM-IV-TR criteria for a current depressive disorder (both in-patients and out-patients) and who had never had a delusional episode. There were 23 patients who met DSM-IV-TR criteria for major depressive disorder (2 participants also had comorbid obsessive-compulsive disorder) and 12 for dysthymia.

(4) *Normal control group*. A non-psychiatric control group of 36 people (21 men and 15 women), basically composed of clinical staff and psychiatry trainees, was recruited via informal contacts to match the deluded groups in terms of gender. No participant had ever suffered either clinical delusions or depressive disorders, as assessed by the computerized version of the Quick Diagnostic Interview Schedule

(Q-DIS; Marcus, Robins, & Bucholz, 1990), and none had ever required psychological assistance for any mental disorder.

All participants in the four groups were between 18 and 65 years of age and were excluded if they had a history of use or abuse of drugs and alcohol. All contacted people in our study accepted to participate with the exception of four active deluded patients. Furthermore, two more patients within that group refused to continue the study after the clinical examination session took place. All these data were excluded from further analyses.

Measures

Brief Psychiatric Rating Scale-Expanded (BPRS; Lukoff, Nuechterlein, & Ventura, 1986). This is a semi-structured interview that assesses the severity of psychiatric symptoms, mainly psychotic ones, during the previous 2 weeks.

Present State Examination (PSE-10, SCAN; WHO, 1992). This is a semi-structured interview used to explore the presence of positive symptoms of psychosis (Sections 18 and 19). Each delusion is assessed in terms of duration, frequency and other relevant dimensions.

Beck Depression Inventory (BDI; Beck, Rush, Shaw, & Emery, 1979). This is a 21-item questionnaire where subjects rate the extent to which they experience cognitive, affective and somatic symptoms of depression. There is much support for its reliability and validity as a measure of depressive symptoms (Beck, Steer, & Garbin, 1988; $\alpha = .93$ in our study).

Rosenberg Self-Esteem Questionnaire (RSEQ; Rosenberg, 1965). The RSEQ is a widely used 10-item questionnaire that explicitly measures global feelings of self-worth or self-acceptance. There is a large body of research on its reliability and validity (Blascovich & Tomaka, 1991; $\alpha = .86$ in our study).

Internal, Personal and Situational Attributions Questionnaire (IPSAQ; Kinderman & Bentall, 1996). The IPSAQ has 32 items describing 16 positive and 16 negative situations in the second person (e.g. 'a friend betrays the trust you placed in them'). For each item, participants are required to think of a single, most likely causal explanation, and then to categorize this cause as being internal (i.e. something due to the respondent), external-personal (i.e. something due to another person or persons) or external-situational (i.e. something due to circumstances or chance). Three positive and three negative subscale scores are derived by adding the number of times internal, external-personal or external-situational attributions are chosen for positive and for negative items. Furthermore, two additional scores are derived: EB (the tendency to attribute negative, as opposed to positive events, to external causes) and PB (the tendency to make personal-external, as opposed to situational-external, attributions for negative events).

Pragmatic Inference Test (PIT; Winters & Neale, 1985). The PIT is an instrument that assesses covert causal reasoning. It is presented as an auditory memory task in order to reduce conscious self-serving bias. It is made up of 12 items adapted from the ASQ (Peterson *et al.*, 1982). Half of the items present hypothetical negative events and the other half, positive events. Each item presents a situation and four multiple choice questions. In order to disguise the purpose of the task, only one of the questions concerns causal attribution. Three scores are derived from the scale: internalizing (i.e. attribution of negative events to oneself), externalizing (i.e. attributing negative events

to others) and the self-serving bias (understood as the proportion between internal attributions for positive events and for negative events).

Procedure

All participants volunteered to collaborate in the study. After reading and signing informed consent, participants were assessed by two clinical psychologists in a quiet room. The assessment was carried out during one session lasting approximately 1 hour 30 minutes. The order of administration of the assessment protocol was the same for all participants: evaluation of socio-demographic data, BPRS, PSE, BDI, RSEQ, PIT and IPSAQ for clinical groups. The Q-DIS was only administered to the control group.

Statistical analyses

Data analyses were conducted with SPSS version 11.0. Type I error rate was set at $\alpha = .05$. *Post hoc* analyses of means were tested, following the Bonferroni method when variances were not statistically different, or the Tamhane method when variances were not equal. A series of ANOVAs were performed, using univariate or multivariate statistics depending upon whether sphericity requirements were met or not, respectively. For categorical variables, chi squared analyses were performed.

Results

A summary of participants' characteristics is presented in Table 1. A unifactorial ANOVA of the variable group (acute deluded/remitted deluded/depressed) yielded statistically significant differences in age of participants, $F(2, 99) = 7.15, p < .0001, \eta^2 = .12$, with the depressed group being older. As expected, a test of gender differences among groups revealed a significant difference, $\chi^2(3, N = 136) = 23.28$, C = 0.38, $p < .05$, with the proportion of women being higher in the depressed group than in the other three groups. With regard to the educational level, statistical analyses showed that the

Table 1. Means and standard deviations of participants' demographic and clinical variables

	Group			
Characteristics	AD ($N = 40$)	RD ($N = 25$)	DD ($N = 35$)	NC ($N = 36$)
Mean age in years (*SD*)	33.3 (8.4)	31.1 (4.9)	39.6 (12.2)	30.4 (7.4)
Male (%)	67.5%	84.0%	25.7%	58.3%
Mean age at onset in years (*SD*)	25.2 (7.6)	22.7 (5.7)	33.6 (12.0)	–
Mean duration of illness in years (*SD*)	8.1 (7.0)	8.4 (5.7)	5.8 (8.5)	–
Mean medication (chlorpromazine mg.)	348.65 (184.6)	263.12 (186.3)	–	–
BDI Total score	10.6 (9.0)	8.9 (5.7)	26.3 (11.1)	1.94 (2.3)
RSEQ Total score	31.5 (4.8)	30.5 (4.7)	24.5 (6.0)	35.6 (3.9)
BPRS Total score	19.0 (8.2)	3.8 (3.5)	14.9 (6.7)	0.91 (1.1)

Note. AD = Acute deluded; RD = Remitted deluded; DD = Depressive disorder; NC = Normal control. BDI = Beck Depression Inventory; RSEQ = Rosenberg Self-Esteem Questionnaire; BPRS = Brief Psychiatric Rating Scale.

control group had higher educational levels than the rest of the groups, $\chi^2(18, N = 136) = 61.01$, C = 0.55, $p < .0001$.

A one-way ANOVA revealed statistically significant differences in onset of illness among the clinical groups, Brown-Forsythe $(2, 74.29) = 7.85$, $p < .0001$, $\eta^2 = .21$. As expected, Tamhane *post hoc* tests showed that the depressed group's age at the onset of the illness was higher than that of the two deluded groups. There was no significant effect for the duration of illness in the clinical groups.

With regard to the severity of the symptoms on the BPRS, considering the BPRS total score (the sum of the 18 items), the one-way ANOVA showed a significant effect for group, Brown-Forsythe $(3, 88.57) = 89.81$, $p < .0001$, $\eta^2 = .65$. Tamhane tests further revealed that there were no differences in BPRS overall severity between acute deluded and depressed groups, whereas there were differences in the rest of the comparisons (see Table 1).

Explicit attributions (IPSAQ)

The mean number of attributions made to internal, external-personal and external-situational causes for both positive and negative events for the four groups are shown in Table 2.

Table 2. Mean scores and standard deviations (in parentheses) in attributional biases as assessed by the Internal, Personal and Situational Attributions Questionnaire (IPSAQ)

	Group			
Explicit attributions (IPSAQ)	AD	RD	DD	NC
Positive events				
Internal	7.12 (1.85)	6.68 (1.86)	6.00 (2.04)	6.44 (1.76)
External-personalizing	3.62 (2.30)	3.48 (2.81)	4.25 (2.60)	1.91 (1.55)
External-situational	5.25 (2.36)	5.84 (2.74)	5.71 (2.20)	7.63 (2.25)
Negative events				
Internal	5.10 (3.47)	5.08 (2.90)	6.14 (2.08)	1.61 (1.39)
External-personalizing	7.35 (3.65)	5.12 (3.27)	5.22 (2.34)	4.75 (2.58)
External-situational	3.52 (2.27)	5.80 (3.74)	4.54 (2.50)	9.63 (2.77)
Externalizing bias	2.02 (3.38)	1.60 (3.09)	−0.14 (2.42)	4.83 (1.97)
Personalizing bias	0.65 (0.21)	0.48 (0.28)	0.54 (0.21)	0.33 (0.18)

Note. AD = Acute deluded; RD = Remitted deluded; DD = Depressive disorder; NC = Normal control.

Separate ANOVAs were conducted for the three attributional loci. A two-way ANOVA on the group (4) × type of event (positive/negative) on *internal attributions* showed significant group effects, $F(3, 132) = 10.98$, $p < .0001$, $\eta^2 = .20$, indicating statistical differences in the total number of internal attributions. *Post hoc* Bonferroni test revealed that the normal control group made fewer internal attributions than the other three groups. The type of event was also significant, $F(1, 132) = 74.15$, $p < .0001$, $\eta^2 = .36$, indicating that all the groups made more internal attributions for positive events than for negative events. Lastly, the interaction was significant, $F(3, 132) = 19.58$, $p < .0001$, $\eta^2 = .30$, indicating that the number of internal attributions made for positive and negative events differed for the four groups. *Post hoc* Bonferroni tests revealed that

normal control group made fewer internal attributions for negative events than the other three groups.

A two-way ANOVA on the *external-personal attributions* in the four groups for both positive and negative events revealed a significant group effect, $F(3, 132) = 6.66$, $p < .0001$, $\eta^2 = .13$. *Post hoc* Bonferroni tests revealed that the acute deluded and the depressed groups made more external-personal attributions than did the normal control group. The type of events was also significant, $F(1, 132) = 64.42$, $p < .0001$, $\eta^2 = .32$. Overall, more external-personal attributions were made for negative than for positive events for all the groups. Lastly, the interaction was significant, $F(3, 132) = 5.04$, $p < .007$, $\eta^2 = .10$. *Post hoc* Bonferroni test revealed that acute deluded and depressed groups made more external-personal attributions for positive events than did the normal control group. With regard to negative events, the acute deluded group made more external-personal attributions than the other three groups.

A two-way ANOVA on the *external-situational attributions* in the four groups for both positive and negative events revealed a significant effect for group, $F(3, 132) = 29.97$, $p < .0001$, $\eta^2 = .40$. *Post hoc* Bonferroni tests revealed that the non-clinical control group made more external-situational attributions than the other three groups, and that the remitted deluded group made more than the acute deluded group. The type of event factor was non-significant, $F(1, 132) = 0.74$, $p = .137$. Lastly, the interaction was significant, $F(3, 132) = 10.27$, $p < .0001$, $\eta^2 = .20$. *Post hoc* Bonferroni tests revealed that the non-clinical control group made more external situational attributions for positive events than the other three groups. With regard to the number of external-situational attributions for negatives events, the non-clinical control group made more than the other three groups, and the acute deluded group made fewer than the remitted deluded group.

Concerning the bias indices (EB and PB) derived from the IPSAQ scores, a one-way ANOVA revealed a significant difference among the groups in the EB, Brown-Forsythe $(3, 106.75) = 19.48$, $p < .0001$, $\eta^2 = .31$. *Post hoc* Tamhane revealed that the acute deluded group scored higher in EB than the depressed group, but lower than the non-clinical control group, which had the highest score of all the groups. The acute deluded and remitted deluded groups did not differ significantly.

A one-way ANOVA revealed significant differences among groups in *PB,* Brown-Forsythe $(3, 94.54) = 13.14$, $p < .0001$, $\eta^2 = 0.24$. *Post hoc* Tamhane tests showed that the acute deluded group presented a higher bias and the non-clinical control group a lower bias than the other three groups. The remitted deluded group had a lower bias than the acute deluded group but a higher one than the non-clinical control group. It seems that remitted deluded group was in an intermediate position, not significantly different from any of these other groups.

Implicit attributions (PIT)

The mean number of internal attributions for positive and negative events and self-serving bias for all the four groups are shown in Table 3.

A two-way ANOVA on the Group (4) × Type of Event (positive/negative) only revealed significant differences in group, $F(1, 132) = 6.51$, $p < .0001$, $\eta^2 = .12$. *Post hoc* Bonferroni test showed that the acute deluded group made more internal attributions in general than the depressed and normal control groups. The normal control group made fewer internal attributions than the other three groups. There were no significant differences between the remitted deluded group and the other groups.

Table 3. Mean scores and standard deviations (in parentheses) in the Pragmatic Inference Test (PIT)

	Group			
Implicit attributions (PIT)	AD	RD	DD	NC
Internal-positive events	2.8 (1.22)	2.60 (1.19)	2.14 (1.33)	2.13 (1.35)
Internal-negative events	2.55 (1.21)	2.36 (1.18)	2.14 (1.08)	1.66 (0.89)
Internal attributions (Total)	2.7 (0.83)	2.5 (0.91)	2.1 (0.72)	1.9 (0.80)
Self-serving bias	0.25 (1.77)	0.24 (1.50)	0.00 (1.95)	0.47 (1.64)

Note. AD = Acute deluded; RD = Remitted deluded; DD = Depressive disorder; NC = Normal control.

With regard to the self-serving bias, a one-way ANOVA revealed no significant differences among groups, $F(3, 132) = 0.43$, $p < .730$.

Attributional biases and psychological symptoms

With regard to the IPSAQ scores, we found a negative correlation between the BDI and EB, $r = -.42$ ($p < .001$) and a positive correlation between the BDI and PB, $r = .23$ ($p < .001$). As expected, BDI scores were found to be positively correlated to internal attributions for negative events ($r = .42$, $p < .001$) and negatively correlated to external-situational attributions for negative events ($r = -.37$, $p < .001$). The severity of the disorder was also related to the magnitude of the attributional biases. In fact, the BPRS total score was significantly related both to the EB, $r = -.38$, $p < .001$, and to the PB, $r = .45$, $p < .001$. Lastly, there was no significant correlation between the PIT scores and the level of depression as measured by the BDI.

Concerning the RSEQ, we logically found the opposite pattern of correlations to those of the BDI. Thus, the RSEQ had positive correlations with external bias, $r = .51$ ($p < .001$), and with external-situational attributions for negative events, $r = .34$ ($p < .001$) and negative correlations with PB, $r = -0.18$ ($p < .005$), and internal attributions for negative events, $r = -.45$ ($p < .001$). Moreover, positive correlations were found between the RSEQ and self-serving bias in the PIT, $r = .25$ ($p < .001$), and between the RSEQ and internal attributions for positive events in the PIT, $r = .27$ ($p < .001$).

Discussion

The chief goal of this work was to find out whether attributional biases (i.e. externalizing or self-serving biases and PB) found in previous studies on delusions (Kinderman & Bentall, 1997; Kinderman *et al.*, 1992; Lyon *et al.*, 1994) are determined by the presence of delusional symptomatology or whether, conversely, they are stable characteristics of individuals with delusional beliefs. Moreover, the study was designed to provide information about the specificity of the results by including two additional control groups (depressed group and normal group).

The pattern of results of the acute deluded and the remitted deluded participants was fairly similar. However, some interesting group differences were found. Regarding the number of explicit internal attributions (as measured by the IPSAQ), we found no differences between the acute and remitted deluded groups either for positive or for

negative events. Nor did we find differences in the EB. Both the acute and remitted deluded groups had a tendency to attribute positive events to internal causes and negative events to external causes. Thus, this causal reasoning bias could be considered a stable characteristic associated with delusions. As expected, and according to the literature on causal attributions in depression (Mezulis, Abramson, Hyde, & Hankin, 2004; Sweeney, Anderson, & Bailey, 1986), in the group of depressed patients, this EB was absent. Although the direction of the bias in deluded participants was the same as in the normal control group, contrary to the results of other studies (Fear *et al.*, 1996; Kinderman & Bentall, 1997; Sharp *et al.*, 1997), its magnitude was significantly lower in the control group.

Regarding the PB, in our study, the normal control group showed the lowest bias of all the groups, which is congruent with the study of Kinderman and Bentall, (1997). Concerning deluded patients, the acute deluded patients showed more bias than the remitted deluded group, which suggests that the magnitude of the bias is related to the patient's clinical state. However, the most relevant finding is that the bias seems to have a non-specific association with pathology. In effect, this bias is also present in the sample of depressed patients, who showed no differences with either the acute or the remitted deluded groups. Although the PB has been described in the literature on causal reasoning and delusions (Kinderman & Bentall, 1997), the finding of a similar bias in depressed patients has not been reported in previous studies.

The IPSAQ was designed to extend the assessment domain of external attributions by permitting the identification of two different kinds of external causes (personal and situational). Although originally designed for use in the field of the study of delusions, the notion of distinguishing between different subtypes of external attributions may be very relevant for other psychological problems. For example, although the predominant attributional models of depression underscore the role of what has been called a *depressogenic attributional style* (Abramson, Seligman, & Teasdale, 1978); that is, an internal, global and stable style of making causal attributions for negative events. There is also clinical and experimental evidence that depression is frequently associated with interpersonal problems and a tendency to blame others for one's own situation. Blaming one's past and significant others is a very frequent characteristic of depressed patients (Bolton *et al.*, 2003; Lewinsohn & Rosenbaum, 1987; Parker, 1979), and may play a causal role in the development of the disorder (Nathan *et al.*, 2003). In fact, although our study confirms the expected internalizing bias (or, more precisely, the absence of an EB) in the depressed group, the finding of a simultaneous PB seems highly relevant. However, the existence of a PB in the depressed group is not incongruent with studies that indicate a high level of interpersonal dysfunction in depression (Joiner, 2002).

Nevertheless, future studies should explore further this finding and the explanatory mechanisms of this reasoning pattern. The PB found in all the groups of patients of our study may originate, at least partially, from a generalized difficulty to take into account certain signs or situational information appropriately, which may exacerbate this dysfunctional attributional style. It has been argued that the analysis of situational parameters, compared with the analysis of personal cues, is more cognitively demanding and therefore patients may have more difficulties to take into account situational information (Bentall, 2003; Freeman *et al.*, 2004). A second possibility that could be addressed in future studies is whether a similar cognitive product (i.e. a PB) may be the result of different underlying cognitive processes in different psychopathological groups. When examining the resulting attributional pattern, the normal control group is seen to have a higher tendency to focus on situational

explanations, especially for negative events, which may be associated with a more benevolent self-and other-schema, which represents the typical style of a state of positive mood (Isen, 2000). Although we cannot suggest a common explanatory hypothesis of the PB in the samples of patients in our study, our results show that the magnitude of the bias is directly related to the severity of the pathology (as measured with the BPRS), whether it be deluded (the degree of suspicion was the best predictor of the number of internal attributions for negative events in the study of Krstev *et al.*, 1999) or – and we believe this finding is novel – depressive. In the only study published to date that has compared performance on the IPSAQ in a group of acute deluded patients and a group of depressed patients, the depressed patients' PB was closer to that of the normal group (Kinderman & Bentall, 1997). In the case of the work of Kinderman and Bentall, they used a group of 20 patients who met the DSM-IV criteria of major depressive episode, although the diagnostic labels of these patients were not reported. The different diagnostic composition of the patients with a depressive episode in both studies may explain the differences found. In any case, more studies are required to extend and replicate the analysis of the relations between PB and psychopathology.

Concerning the implicit attributional measure (PIT), we found no differences between the acute and remitted deluded patients, either in the number of internal attributions or in the self-serving bias. According to the literature reviewed, we expected that both groups of deluded patients and the group of depressed patients would show an internal attributional pattern for negative events in the implicit attribution task (Lyon *et al.*, 1994). However, interpretation of these results does not seem relevant because of the low internal consistency of the PIT that we found. The general alpha indexes obtained in our study were .27 for positive events and − .09 for negative events. Although the original study of Winters and Neale (1985) showed higher alpha coefficients (.59 for positive events and .69 for negative ones), other works (Krstev *et al.*, 1999) that are similar to the present study also show very low Cronbach's alpha internal consistency indexes (.16 for negative events and .36 for positive ones). In fact, theoretically, as the PIT indirectly assesses attributional patterns that are assumed to be closely related to self-concept and self-schemata, it is surprising that we found no correlations between any of the PIT subscales and the BDI scores. Furthermore, there were no significant correlations between the PIT and the RSEQ scores with only some exceptions (.27 for internal attributions for positive events and .25 for the self-serving bias). Our study therefore confirms the need to find alternative assessment methods to evaluate implicit attributions, which are key features of current explanatory models of paranoia (Bentall, 2003).

In sum, our study offers some intriguing results on the pattern of changes in attributional reasoning in delusion. Whereas both acute and remitted deluded patients show a similar EB, acute deluded patients show a higher PB than remitted patients do. Given that blaming bad events on other people rather than on circumstances seems to be central for the understanding of delusions, and especially paranoia (Bentall, 2003; Bentall *et al.*, 2001; Kinderman *et al.*, 1992), this finding may be relevant to both explain changes in the patients' clinical state and to guide clinical interventions to modify attributional patterns (e.g. Kinderman, 2001; Kinderman & Benn, 2002). However, future studies should analyse the relations between changes in patterns of causal explanations and changes in psychopathology by using longitudinal designs, or even studies that, as have been used in the research on vulnerability to depression (e.g. Persons & Miranda 1992), activate latent negative self-schemata. Furthermore, future research on causal attributions should be refined. Firstly, it would be necessary, as

Martin and Penn (2002) have pointed out, to analyse patterns of attributions for actual rather than hypothetical events. Secondly, researchers should also develop psychometrically sound instruments that allow them to adequately measure implicit cognitions that are central for new models of deluded reasoning.

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