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Attributional Style among Youth at Clinical Risk for Psychosis

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Abstract

Aim—A biased attributional style, in which negative events are attributed to external and personal causes, is associated with paranoid delusions in schizophrenia. It is not known whether this biased attributional style also characterizes individuals at clinical risk for psychosis, or if it is associated with their emergent paranoia.

Methods—33 clinical high-risk patients and 15 age- and gender-similar controls were assessed with the Internal, Personal, and Situational Attributions Questionnaire for externalizing and personalizing attributional biases and for potential correlates with suspiciousness and other symptoms.

Results—Both patients and controls had a similar external-personalizing attributional style, which was unrelated to symptoms, including suspiciousness.

Conclusions—Consistent with other studies, a biased attributional style was not associated with subthreshold paranoia. Therefore, a biased attributional style is likely not a trait that contributes to emergent paranoid delusions, but instead a state-dependent correlate of paranoid delusions.

Keywords

attribution; hig	gh risk; psychosis;	schizophrenia;	social cogni	tion	

Introduction

Attributional style refers to the characteristic ways in which individuals explain the causation of events. Studies have examined attribution to external versus internal causes, showing that healthy people have a self-serving bias, and tend to attribute negative events to external causes and attribute positive events to themselves. ^{1,2} Further, healthy people preferentially attribute their own negative behavior to situational factors, constituting an external-situational attributional style, ³ but attribute the negative behavior of others to personal factors, constituting an external-personal attributional style. ⁴ This normal self-serving bias, is attenuated in clinical cohorts with depression and anxiety, ⁵ but is exaggerated in individuals with paranoia, across diagnoses. ^{6,7,8}

Attributional bias was initially measured primarily using the Attributional Style Questionnaire (ASQ), ⁹ a 12-item self-report measure in which respondents read about a situation, write a causal statement about the event, and then rate that statement on

dimensions of internality, stability, and globality, which refers to whether attributions can apply to a broad or narrow range of situations. ¹⁰ The Internal, Personal, and Situational Attributions Questionnaire (IPSAQ) was then developed to further distinguish between attributions of causation to other individuals (external-personal) or to situational factors (external-situational). ¹¹ Initial studies with the IPSAQ found an external-personalizing bias in individuals with paranoia, in which causation for negative events is disproportionately attributed to others. ¹² However, later studies with the IPSAQ found no difference between schizophrenia patients and controls, ^{13,14} or an attenuated self-serving bias comparable to but less severe than that seen in depression. ¹⁵

The exaggerated self-serving bias may be state-specific in schizophrenia patients, seen only in the context of acute paranoia ^{16,17} with concurrent grandiose delusions. ¹⁸ As yet, it is unclear if an exaggerated self-serving bias would be observed with suspiciousness, or with subthreshold paranoia, which might suggest it is a risk factor for paranoid delusions. Studies using the IPSAQ in nonclinical cohorts suggest no difference in attributional style among college students with subclinical paranoia, ^{14,19} in adults (ages 36–65) who endorse psychotic-like experiences in population surveys, or in first-degree adult relatives of people with non-affective psychosis. ²⁰ However, to the best of our knowledge, attributional style has not yet been evaluated in a clinical risk cohort of youths at appreciable risk for schizophrenia (~30%). ²¹ Herein, we characterize causal attribution in a CHR cohort and evaluate its relationship to concurrent symptoms, specifically subthreshold paranoia.

Method

Participants

This study was conducted at the Center of Prevention and Evaluation (COPE), a psychosis-risk clinical research program at the New York State Psychiatric Institute at Columbia University Medical Center. Patients were help-seeking youths considered at clinical high-risk for non-affective psychosis, generally referred from schools and clinicians, or self-referred from the program website (www.copeclinic.com). Participants met criteria for at least one of three prodromal syndromes, as assessed with the Structured Interview for Prodromal Syndromes: 1) attenuated positive symptoms syndrome; 2) genetic risk and deterioration syndrome; or 3) brief intermittent psychotic syndrome.²² Healthy controls were recruited from the same source community using mailings and internet postings. All participants provided written informed consent, or if under the age of 18, written assent with provision of written informed consent by a parent. Data were collected between April, 2005 and July, 2009. This study was approved by the New York State Psychiatric Institute's Institutional Review Board.

Measures

All measures, including demographics, symptoms, and attributional style, were administered at baseline upon enrollment into the longitudinal cohort study.

Demographic data including age, sex and ethnicity, were reported by the participants.

Attributional style was measured using the Internal, Personal, and Situational Attributions Questionnaire, ¹¹ in which the respondent rates positive and negative events as being caused by themselves (internal), other people (external-personal), or the situation (external-situational). Scores for attribution biases were coded according to their guidelines: externalizing bias is coded from –16 to 16, with positive numbers signifying greater external attributions for negative events; personalizing bias is coded from 0 to 1, with scores greater than .5 signifying more external-personal than external-situational attributions for negative

events. The IPSAQ scales have adequate reliability (externalizing bias alpha = .7189, personalizing bias alpha = .7609). ¹¹

Subthreshold psychotic symptoms and modified global assessment of function were rated using the Structured Interview for Prodromal Syndromes/ Scale of Prodromal Symptoms (SIPS/SOPS) by clinicians certified in its administration by Barbara Walsh at Yale University. Interrater reliability is excellent to near-excellent for individual items.²² Depression and anxiety were evaluated using the Hamilton Rating Scale for Depression²³ and Hamilton Rating Scale for Anxiety,²⁴ respectively.

Statistics

The effects of demographic variables on attributional style measures were tested in the combined sample of patients and controls using independent samples t-tests and Spearman's rank order correlation; effect modification was also evaluated. Independent samples t-tests and chi-square tests were used to compare CHR patients and healthy controls for demographics, attribution, and symptoms. Between-group comparisons for attributional style were repeated using analysis of variance with the inclusion of sex as a potential confounder. For the CHR group, Spearman's rank order correlations were calculated for attribution scores with symptoms and function. Linear regression analyses were also conducted with sex and symptoms as predictors and IPSAQ measures as response variables. Alpha was set at .05 for differences between patients and controls for attribution and symptoms, and for tests of association between attributional style and suspiciousness or subthreshold paranoia. Exploratory analyses were done to examine any differences in attributional style among CHR participants who later made a transition to psychosis.

Results

There were 33 CHR patients and 15 healthy controls, who were comparable in age, ethnicity, and gender (Table 1). In the combined group of patients and controls (N=48), age and ethnicity bore no association to attribution, but females had a greater externalizing bias than males, $t_{(45)} = 2.194$, p=.03; there was no age by gender interaction effects on attributional style. CHR patients and healthy controls had nearly identical attributional styles, with both externalizing and personalizing biases (Table 1). Inclusion of gender in the model also did not yield any group effect on attribution. As expected, CHR patients differed from healthy controls in symptoms and function (Table 1). Attributional bias also had no association with subthreshold paranoia (i.e. suspiciousness) or other positive symptoms, or with function, depression or anxiety (Table 2), even in models including gender as a covariate. Attribution was comparable among those CHR patients who did (n=7; mean externalizing score was 1.4, SD 3.0, mean personalizing score was .51, SD .34) and did not (n=26; mean externalizing score was 2.2, SD 4.9, mean personalizing score was .67, SD .26) make a later transition to psychosis (externalizing: $t_{(31)} = -.378$, p=.71; personalizing: $t_{(31)} = -1.284$, p=.21).

Discussion

This initial study is the first to examine causal attributional style in a clinical high risk cohort. We found that the attributional style of the clinical high risk patients was comparable to that of the healthy controls, even accounting for gender differences between the groups. The exaggerated self-serving bias associated with paranoia was not evident in this CHR cohort. It is unlikely that a finding of comparability in externalizing is a consequence of the small sample size and potential Type 2 error, as an N of 2,618 would be necessary to detect a statistically significant difference (calculated using the means and standard deviations yielded herein). Also, among CHR patients, there was no association of attribution with

suspiciousness or subthreshold paranoia (or any clinical measures), or eventual transition to psychosis. Of note, the cohort is similar to other North American CHR cohorts in terms of age, high prevalence of attenuated positive symptom syndrome, and mean positive and negative symptom scores.²⁵

Most studies of paranoia or schizophrenia have measured attribution with either the ASQ or the IPSAQ, facilitating comparison of results across studies. The IPSAQ was used here due to its better reliability and ability to distinguish between personal and situational attributions. ¹¹ The externalizing and personalizing scores in our study are comparable to those found in previous studies (mean externalizing scores: controls=2.6, range 1.9 to 3.1, patients=2.2, range 1.7 to 3; mean personalizing bias: controls=.56, range .40 to .65, patients=.70, range .64 to .75; across 4 studies). ^{11,12,13,19}

Although widely used, the IPSAQ and ASQ have been criticized as having low ecological validity and being difficult to understand for some symptomatic patients, leading to a preference among some investigators for qualitative assessments of attribution^{6,17,26} using the Leeds Attribution Coding System.²⁷ Qualitative studies have provided evidence that attributional biases are state-based¹⁷ and vary depending on whether the respondent sees the delusion as the cause or the outcome of the event.²⁶ These aspects of attribution are difficult to elicit using self-report measures that present hypothetical events, such as the IPSAQ and ASQ. Future qualitative research with CHR samples may allow the identification of associations between attributional style and subthreshold symptoms that were not observed in the present study. Future studies can also evaluate the association of attributional style with IQ, which was not measured in this study.

Our data suggest attributional bias is not a promising trait-like candidate risk factor for the development of paranoid delusions. This is consistent with studies in other "risk" cohorts, including first-degree relatives of individuals with schizophrenia, and adults and college students who endorse psychotic-like experiences when queried, ^{14,19,20} and with evidence that attributional biases are state-specific in schizophrenia. ^{16,17} However, given that attributional bias is associated with paranoia in clinical cohorts, future studies in larger cohorts should examine the evolution of attributional patterns and paranoid symptoms over time to better understand their relationship.

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Table 1Descriptive statistics and between-group comparisons for CHR patients and controls on demographic, attribution, and symptom measures.

	CHR n=33	Controls n=15	Statistics
Age	18.7 (3.4)	19.9 (3.7)	$t_{(46)} = -1.032, p=.31$
Sex (% female)	18	50	$\chi^2_{(1, n=47)} = 3.510, p=.06$
Race (% Caucasian)	48	69	$\chi^2_{(1, n=46)} = .890, p=.35$
IPSAQ:			
Externalizing score	2.0 (4.7)	2.1 (4.3)	$t_{(46)} =073, p = .94$
Personalizing score	.64 (.28)	.61 (.29)	t ₍₄₆₎ = .314, <i>p</i> =.76
Scale of Prodromal Symptoms	s:		
Suspiciousness	2.7 (1.4)	0.4 (.7)	t _(44.3) = 7.631, <i>p</i> <.001 *
Unusual Thought Content	3.5 (1.2)	.21 (.43)	t _(43.8) = 13.177, <i>p</i> <.001 *
Grandiosity	1.7 (1.5)	0 (0)	t _(32.0) = 6.600, <i>p</i> <.001 *
Perceptual Disturbances	2.6 (1.5)	.14 (.36)	t _(39.4) = 8.626, <i>p</i> <.001 *
Conceptual	1.9 (1.3)	.14 (.36)	t _(41.4) = 6.918, <i>p</i> <.001 *
Disorganization			
Total positive	12.4 (4.4)	0.9 (.9)	t _(35.3) = 13.924, <i>p</i> <.001 *
Total negative	13.0 (5.7)	1.8 (2.0)	t _(44.0) = 9.991, <i>p</i> <.001 *
Global Function	44.5 (7.1)	77.4 (6.5)	t ₍₄₅₎ = -14.9, <i>p</i> <.001 *
Hamilton:			
Depression	12.1 (6.8)	1.5 (1.7)	t _(33.2) = 7.824, <i>p</i> <.001 *
Anxiety	10.9 (6.8)	1.5 (1.5)	$t_{(32.2)} = 7.018, p < .001$ *

All data listed are means, with standard deviations in parentheses. Alpha=.05 for all t-tests and χ^2 , with degrees of freedom in parentheses.

^{*}p<.001

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Table 2

Correlations between measures of attributional bias and measures of symptoms and functioning among the CHR sample.

	Z	IPSAQ: Ex	IPSAQ: Externalizing	IPSAQ: Personalizing	rsonalizing
		rho	d	rho	d
Age	33	0.01	96.0	0.21	0.25
Sex	33	0.385	* 720.	0.34	90.0
Race	33	-0.19	0.30	-0.04	0.82
Scale of Prodromal Symptoms:					
Suspiciousness	33	-0.15	0.41	0.00	0.99
Unusual thought content	33	0.14	0.45	-0.22	0.21
Grandiosity	33	0.14	0.43	-0.12	0.52
Perceptual disturbances	33	-0.17	0.34	-0.33	90.0
Conceptual disorganization	33	0.09	0.62	0.20	0.27
Total positive	33	0.07	0.71	-0.01	0.95
Total negative	33	-0.01	0.94	-0.25	0.17
GAF-m	33	0.04	0.81	0.25	0.17
Hamilton					
Depression	28	-0.18	0.35	-0.02	0.92
Anxiety	28	-0.09	0.65	0.01	0.98

Spearman's rank correlations,

* P<.05

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