



The affective reactivity of psychotic speech: The role of internal source monitoring in explaining increased thought disorder under emotional challenge



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ABSTRACT

Thought disorder (TD) has been shown to vary in relation to negative affect. Here we examine the role internal source monitoring (iSM, i.e. ability to discriminate between inner speech and verbalized speech) in TD and whether changes in iSM performance are implicated in the affective reactivity effect (deterioration of TD when participants are asked to talk about emotionally-laden topics). Eighty patients diagnosed with schizophrenia-spectrum disorder and thirty healthy controls received interviews that promoted personal disclosure (emotionally salient) and interviews on everyday topics (non-salient) on separate days. During the interviews, participants were tested on iSM, self-reported affect and immediate auditory recall. Patients had more TD, poorer ability to discriminate between inner and verbalized speech, poorer immediate auditory recall and reported more negative affect than controls. Both groups displayed more TD and negative affect in salient interviews but only patients showed poorer performance on iSM. Immediate auditory recall did not change significantly across affective conditions. In patients, the relationship between self-reported negative affect and TD was mediated by deterioration in the ability to discriminate between inner speech and speech that was directed to others and socially shared (performance on the iSM) in both interviews. Furthermore, deterioration in patients' performance on iSM across conditions significantly predicted deterioration in TD across the interviews (affective reactivity of speech). Poor iSM is significantly associated with TD. Negative affect, leading to further impaired iSM, leads to increased TD in patients with psychosis. Avenues for future research as well as clinical implications of these findings are discussed.

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1. Introduction

Thought disorder (TD) is a common (Tan et al., 2014) and enduring feature of psychosis (Marengo and Harrow, 1987, 1997) that is associated with poorer occupational (Racenstein et al., 1999) and social functioning (Bowie and Harvey, 2008), poorer quality of life (Tan et al., 2014) and relapse in patients (Wilcox, 1990), and transition to psychosis in high-risk populations (Bearden et al., 2011). As there is a lack of evidence-based psychological treatments for TD (Beck et al., 2009) there is a pressing need to understand the mechanisms that underlie it.

Hyperpriming in semantic memory (i.e. hyper-activation of semantically-related nodes) has been proposed as one such mechanism (Spitzer, 1997). However, a meta-analysis failed to find significant differences between TD and non-TD patients (Pomarol-Clotet et al., 2008) on this. An alternative theory implicates difficulties with theory-of-mind (ToM, Hardy-Baylé et al., 2003) which could explain

difficulties sharing topics and misalignment in conversation. Indeed impairments in ToM, although not specific, are highly associated with TD (Sprong et al., 2007) but these difficulties alone are unlikely to explain incoherent speech.

1.1. Internal source monitoring (iSM)

iSM refers to the ability to discriminate between self-generated private stimuli such as inner speech, and self-generated speech that is directed to others (Johnson et al., 1993) (iSM is different from external source monitoring implicated in hallucinations, in which the individual distinguishes between inner speech and the *heard* speech of others, Brookwell et al., 2013). Harvey (1985) reported and subsequently replicated (Harvey et al., 1988; Harvey and Serper, 1990) an association between TD in schizophrenia patients and a bias towards over-reporting words as having been verbalized when they had only been thought. Nienow and Docherty (2004) replicated this finding controlling for IQ and working memory and, in a later study, reported a significant association between these biases and communication disturbances (Nienow

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and Docherty, 2005). More recently, Docherty (2012) tested patients using both iSM and an external source-monitoring task. Again, performance on the former was found to be a significant predictor of communication disturbances even after controlling for performance on the external source-monitoring, immediate recall and working memory.

1.2. Affect

The exacerbation of communication difficulties during discussion of affectively-laden topics has been termed *affective reactivity of speech* and has been observed in schizophrenia (Docherty, 1996; Haddock et al., 1995) and bipolar disorder (Tai et al., 2004). For example, Docherty and colleagues tested schizophrenia patients using two speech tasks in which they had to discuss stressful or pleasant experiences; participants displayed more TD in the stressful condition (Docherty et al., 1994a, 1994b).

The affective reactivity of speech in TD is a well-replicated phenomenon (e.g. Docherty et al., 1994a; Docherty, 1996; St-Hilaire and Docherty, 2005) but its cause is unknown. Here we attempt to explore whether a specific psychological mechanism known to be associated with TD – internal source monitoring, as reviewed above – is also affectively reactive and may therefore explain the affective state-dependent deterioration in social speech observed in thought-disordered patients (i.e. affective reactivity of speech).

2. Materials and methods

2.1. Participants

Eighty participants (see Table 1) were recruited from mental health sites in the UK. The recruitment targeted 18–65 year olds with a diagnosis of schizophrenia-spectrum disorder (WHO, 2004). PS confirmed all the diagnoses with the use of a clinical interview (i.e. PANSS) and the patient's clinical history. All participants provided informed consent according to the Declaration of Helsinki. We excluded participants whose first language was not English, who had severe learning difficulties, recent substance abuse or history of medical disorders that could affect brain function. Antipsychotic medications were converted to chlorpromazine-equivalents as per agreed conventions (Woods, 2003).

For comparison purposes, thirty healthy participants were recruited through advertisements in the community. An attempt was made to

select participants who were approximately comparable for age, gender and ethnicity with participants in the clinical group.

2.2. Materials

2.2.1. Psychotic symptoms

Psychotic symptoms were measured using the Positive and Negative Syndromes Scale (PANSS, Kay et al., 1987) that measures 30 symptoms, comprising a positive, a negative, and a general psychopathology scale. Each item is scored from 1 to 7 with the higher score indicating increased severity. The scale has been found to have good psychometric properties (Kay et al., 1987).

2.2.2. IQ

Intelligence was evaluated using the Quick test (QT, Ammons and Ammons, 1962) in which the participant is presented with four pictures (e.g. a policeman stopping the traffic with a whistle) and is asked to identify fifty words by pointing to the appropriate card where the word referent can be found (e.g. “whistle”). The final score is achieved by summing the number of words correctly identified and scores are converted using standardized guidelines (Ammons and Ammons, 1962).

2.2.3. Interviews

Speech samples were gathered using two interviews that had been previously developed to elicit TD (Tai et al., 2004). The salient interview involved fifteen questions that promoted self-disclosure by asking for negative autobiographical memories, whereas the non-salient interview included fifteen questions about neutral topics (see Appendix 1). Means and standard deviations for duration of the interviews and word-counts can be seen in Table 2.

2.2.1. TD

Speech samples were rated by two independent raters, one of whom was blind to the study hypotheses, using the 18-items of the Scale for the Assessment of Thought, Language and Communication (TLC, Andreasen, 1986). The total is achieved by summing the items scores. The scale has good psychometric properties (Andreasen, 1979, 1986). Table 2 shows the means and standard deviations for the total scores.

Table 1
Clinical and demographic variables.

	Patients	Comparisons	
Sample size	80	30	
Gender (%)	Male 22 (27.5%) Female 58 (72.5%)	21 (70%) 9 (30%)	$\chi^2 = .067; p = .795$
Ethnicity (%)	White British 6 (7.5%) Other 74 (92.5%)	28 (93.3%) 2 (6.6%)	$\chi^2 = .006; p = .936$
Age (years)	39.3 (11.6)	38.4 (13.3)	$t = .33; p = .746$
Years of education	11.2 (1.9)	12.7 (2.3)	$t = -3.35; p = .001$
IQ	98.4 (10.6)	109.5 (8.3)	$t = -5.18; p < .005$
Diagnoses (%)	Schizophrenia (F20) 18 (22.5%) Schizoaffective (F25) 14 (17.5%) Other Psychoses (F29) 48 (60%)	N/A N/A N/A N/A	
Duration of illness (years)	15.2 (10.9)	N/A	
History of admission (yes)	73 (91.3%)	N/A	
FGA (%)	26 (23.6%)	0 (0%)	
SGA (%)	58 (72.5%)	0 (0%)	
'Mood stabilizers' (%)	14 (17.5%)	0 (0%)	
Anti-depressants (%)	31 (38.7%)	0 (0%)	
Equivalent CPZ dose (mg)	469.7 (389.1)	N/A	
PANSS	Positive 17.1 (5.2) Negative 14 (4.7) General 38.6 (9.2) Total 69.8 (16.1)	N/A N/A N/A N/A	

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