



Emotion processing in schizophrenia is state and trait dependent



Arija Maat^{a,6}, Simone J.T. van Montfort^{b,6}, Jessica de Nijs^b, Eske M. Derks^a, René S. Kahn¹, Don H. Linszen², Jim van Os^{3,5}, Durk Wiersma⁴, Richard Bruggeman⁴, Wiepke Cahn^{*1}, Lieuwe de Haan², Lydia Krabbendam³, Inez Myin-Germeys³, GROUP Investigators⁷

¹ Department of Psychiatry, Rudolf Magnus Institute of Neuroscience, University Medical Center Utrecht, Utrecht, The Netherlands

² Department of Psychiatry, Academic Medical Center, University of Amsterdam, Amsterdam, The Netherlands

³ South Limburg Mental Health Research and Teaching Network, EURON, Maastricht University Medical Center, Maastricht, The Netherlands

⁴ Department of Psychiatry, University Medical Center Groningen, University of Groningen, Groningen, The Netherlands

⁵ King's College London, King's Health Partners, Department of Psychosis Studies, Institute of Psychiatry, London, United Kingdom

^a Department of Psychiatry, Academic Medical Center, University of Amsterdam, Meibergdreef 5, 1105 AZ Amsterdam, The Netherlands

^b Department of Psychiatry, Rudolf Magnus Institute of Neuroscience, University Medical Center Utrecht, Huispostnummer A 00.241, Postbus 85500, 3508 GA Utrecht, The Netherlands

ARTICLE INFO

Article history:

Received 5 May 2014

Received in revised form 18 November 2014

Accepted 22 November 2014

Available online 24 December 2014

Keywords:

Emotion processing
Cognition
Psychosis
Remission
Schizophrenia
Social cognition

ABSTRACT

Background: Substantial evidence exists about emotion processing (EP) impairments in schizophrenia patients. However, whether these deficits are present primarily during psychosis (i.e., state dependent) or an integral part of the disorder (i.e., trait dependent) remains unclear.

Methods: EP was assessed with the degraded facial affect recognition task in schizophrenia patients ($N = 521$) and healthy controls ($N = 312$) at baseline (T1) and after a three year follow-up (T2). In schizophrenia patients symptomatic remission was assessed with the Positive and Negative Syndrome Scale (PANSS) remission tool. Patients were divided into four groups: remission T1 and remission T2 (RR); remission T1 and non-remission T2 (RN); non-remission T1 and non-remission T2 (NN) and non-remission T1 and remission T2 (NR). Factorial repeated measures ANCOVA was used to compare EP performance over time between groups. Age, gender and general cognition were included as covariates.

Results: Schizophrenia patients performed worse than healthy controls on EP at T1 ($p = 0.001$). The patients that were in symptomatic remission at both time points (the RR group) performed worse than the healthy controls at T2 ($p < 0.001$). Significant group \times time interactions were found between RR and RN ($p = 0.001$), and between NR and RN ($p = 0.04$), indicating a differential EP performance over time. No group \times time interaction was found between NN and NR.

Conclusion: The results show relatively poor EP performance in schizophrenia patients compared to healthy controls. EP performance in schizophrenia patients was associated with symptomatic remission. The results provide support for the hypothesis that EP deficits in schizophrenia are both state and trait dependent.

© 2014 Elsevier B.V. All rights reserved.

* Corresponding author at: Department of Psychiatry, Rudolf Magnus Institute of Neuroscience, University Medical Center Utrecht, Huispostnummer A 00.241, Postbus 85500, 3508 GA Utrecht, The Netherlands. Tel.: +31 88 7556003; fax: +31 88 7555466.

E-mail address: w.cahn@umcutrecht.nl (W. Cahn).

⁶ Both authors contributed equally to the study.

⁷ Group Investigators: René S. Kahn (a), Don H. Linszen (b), Jim van Os (c,e), Durk Wiersma (d); Richard Bruggeman (d), Wiepke Cahn (a), Lieuwe de Haan (b), Lydia Krabbendam (c), Inez Myin-Germeys (c) (a) Department of Psychiatry, Rudolf Magnus Institute of Neuroscience, University Medical Center Utrecht, Utrecht, The Netherlands; (b) Department of Psychiatry, Academic Medical Center, University of Amsterdam, Amsterdam, The Netherlands; (c) South Limburg Mental Health Research and Teaching Network, EURON, Maastricht University Medical Center, Maastricht, The Netherlands; (d) Department of Psychiatry, University Medical Center Groningen, University of Groningen, Groningen, The Netherlands; (e) King's College London, King's Health Partners, Department of Psychosis Studies, Institute of Psychiatry, London, United Kingdom.

1. Introduction

Schizophrenia is characterized by positive symptoms (e.g., delusions and hallucinations), negative symptoms (e.g., flat or blunted affect and emotion), and cognitive impairments (e.g., deficits in working memory, attention and social cognition) (American Psychiatric Association, 2000). Social cognition represents how people think about themselves and others (Penn et al., 2008) and is necessary for successful social interactions between people (Baron-Cohen et al., 1985). Emotion processing (EP) is an important domain of social cognition (Green et al., 2005) and has been described as the ability to infer emotional information from prosody or facial expressions, the latter being the focus of the current study (Couture et al., 2006). Not surprisingly, EP is found to be related to social problem solving and community functioning in

schizophrenia (Hofer et al., 2009; Irani et al., 2012). Moreover, impairments in EP may even exceed the value of general cognition in explaining outcome in schizophrenia (Fett et al., 2011).

Schizophrenia patients differ as for the genetic patterns that predispose them to the illness (Gershon et al., 2011). A factor complicating the search for genes that underlie the disorder is that the course of the disease is usually characterized by different states of illness that fluctuate over time (i.e., a patient returns to a non-psychotic state in between psychotic episodes). An emerging area of genetic research in schizophrenia is that of so-called 'trait markers'. Trait markers refer to processes that play an antecedent, possibly causal, role in the susceptibility to the disease (Chen et al., 2009). These markers may be closer to the genotype than the symptoms of the illness (van Os and Kapur, 2009), and, therefore, can be useful targets for genetic studies. In addition, trait markers can be relevant if they have a high diagnostic specificity. A behavioral trait is an enduring characteristic that is associated with illness in the population. Numerous studies have shown deficits in EP performance in schizophrenia patients compared to healthy controls (Marwick and Hall, 2008; Penn et al., 2008; Chan et al., 2010; Kohler et al., 2010). Cross-sectional studies showed EP deficits to be present at the first onset of schizophrenia and to be stable over the course of illness in chronic patients (Pinkham et al., 2007; Green et al., 2012). Trait markers are most useful when they are also present in clinically unaffected relatives (Chen et al., 2009). Indeed, some studies found EP deficits in unaffected siblings of schizophrenia patients (Eack et al., 2009; de Achaval et al., 2010). Siblings performed worse on recognizing facial emotion compared to healthy controls, suggesting a trait dependent deficit in EP in schizophrenia.

However, results on EP deficits in unaffected siblings of schizophrenia patients are inconsistent and the presence of EP deficits at a prodromal stage of the illness remains questionable (Kee et al., 2004). Although some studies found EP deficits in people at clinical high risk for psychosis (Amminger et al., 2012; Green et al., 2012), other studies found that subjects at increased risk for psychosis performed similarly to healthy controls (Pinkham et al., 2007). Moreover, EP performance might change over time according to an increase or decrease in clinical symptoms, as one study reviewing 24 studies on EP suggested that individuals in remission outperform individuals at an acute phase of the disorder (Edwards et al., 2002). In addition, several studies showed that poor EP performance in schizophrenia was related to more severe schizophrenia symptoms (Kohler et al., 2000, 2010; Marwick and Hall, 2008; Laroi et al., 2010; Huang et al., 2013; Tseng et al., 2013; Ventura et al., 2013). A longitudinal study by Kucharska-Pietura et al. showed EP deficits in schizophrenia patients to worsen with progression of illness (Kucharska-Pietura et al., 2005). Although it remains uncertain if the decline in EP ability seen in patients over time was entirely due to an increase in illness severity, the results at least indicate that EP deficits in schizophrenia patients fluctuate over time.

To the best of our knowledge, no longitudinal study to date has investigated whether EP impairments are either present primarily during psychosis (i.e., state dependent) or form an integral part of the disorder (i.e., trait dependent), or a combination of the two (i.e., state as well as trait dependent). Typically, though not necessarily, a state characteristic is transient and a trait characteristic is enduring (Chen et al., 2009). Therefore, longitudinal research is essential to elucidate whether EP deficits are state or trait dependent in schizophrenia, because these studies follow the natural course of illness within the same patient, whereas cross-sectional studies do not. The present study was outlined to examine EP performance, i.e., facial emotion recognition ability, longitudinally in a large cohort of schizophrenia patients and healthy controls over three years' time. Assessments of EP, general cognition (IQ) and schizophrenia symptoms were performed at baseline and after a three year follow-up. First, EP performance was compared between schizophrenia patients and healthy controls. Second, schizophrenia patients were divided into four groups, based on their symptomatic state of illness at both measurements,

i.e. remission or non-remission at baseline and remission or non-remission at follow-up. The severity of schizophrenia symptoms served as a basis for defining state of illness within the patients. EP scores were compared between the four patient groups over time. In the literature to date there is still debate concerning emotion specific EP deficits in schizophrenia patients. Although some studies suggest a negative-emotion specific deficit (Edwards et al., 2001; Bediou et al., 2005; van 't Wout et al., 2007), other more recent studies suggest that facial emotion recognition impairment in schizophrenia may reflect a more generalized deficit (Mendoza et al., 2011; Huang et al., 2013).

In the context of previous evidence of social cognitive impairments in schizophrenia patients, we expected the EP scores to be different between patients and healthy controls. Besides being related to the disorder (trait dependent), we also expected EP performance to vary within the patient group depending on state of illness, in other words, for the patient groups we hypothesized EP performance to be state dependent. Specifically, 1) for the patients in non-remission at baseline we expected an improvement on EP performance over time if they achieved a remission state at follow-up, and 2) for patients in remission at baseline, we expected a decrease in EP performance over time, if they returned to a non-remission state at follow-up.

2. Method

2.1. Procedure and sample

The data originate from measures of the ongoing longitudinal multicenter study 'Genetic Risk and Outcome in Psychosis' (GROUP). Assessments were performed at baseline and after a three year follow-up. The procedure of recruitment, informed consent, approval by the accredited Medical Ethics Review Committee (METC) and population characteristics of the participants have been described in a previous report on the GROUP study (Korver et al., 2012). The full GROUP sample at baseline consisted of 1120 patients with a non-affective psychotic disorder, 1057 of their siblings, 919 of their parents, and 590 healthy controls. For this study, we included patients and healthy controls for whom assessments were available at baseline and follow-up.

The patient group had to meet the criteria for non-affective psychotic disorder of the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM IV) (American Psychiatric Association, 2000), as assessed by the Comprehensive Assessment of Symptoms and History (CASH) interview (Andreasen et al., 1992). Further inclusion criteria for patients were: age between 15 and 60; good command of the Dutch language; ability and willingness to give informed consent and having had the first psychotic episode up to ten years before baseline.

For the healthy control group inclusion criteria were: not having any diagnosis according to DSM IV (American Psychiatric Association, 2000), as assessed by the CASH (Andreasen et al., 1992); age between 15 and 60; good command of the Dutch language; ability and willingness to give informed consent and no first degree family members with a psychotic disorder at baseline.

2.2. Measures

All measures used in the GROUP project were selected on the basis of established validity, reliability and on their feasibility for use in multisite studies.

2.2.1. The Positive and Negative Syndrome Scale (PANSS)

In the GROUP project, current severity of symptoms was measured with the PANSS (Kay et al., 1987). The PANSS consists of 30 items. Each item is scored on a scale ranging from 1 (absent) to 7 (extreme), the behavioral effect of symptoms and their severity are incorporated in item rating. Three domains are described for the PANSS, measuring positive, negative or general symptoms.

متن کامل مقاله

دریافت فوری ←

ISIArticles

مرجع مقالات تخصصی ایران

- ✓ امکان دانلود نسخه تمام متن مقالات انگلیسی
- ✓ امکان دانلود نسخه ترجمه شده مقالات
- ✓ پذیرش سفارش ترجمه تخصصی
- ✓ امکان جستجو در آرشیو جامعی از صدها موضوع و هزاران مقاله
- ✓ امکان دانلود رایگان ۲ صفحه اول هر مقاله
- ✓ امکان پرداخت اینترنتی با کلیه کارت های عضو شتاب
- ✓ دانلود فوری مقاله پس از پرداخت آنلاین
- ✓ پشتیبانی کامل خرید با بهره مندی از سیستم هوشمند رهگیری سفارشات