

## Original Article

**Cite this article:** González-Ortega I *et al* (2020). Influence of social cognition as a mediator between cognitive reserve and psychosocial functioning in patients with first episode psychosis. *Psychological Medicine* 50, 2702–2710. <https://doi.org/10.1017/S0033291719002794>

Received: 23 January 2019

Revised: 9 August 2019

Accepted: 13 September 2019

First published online: 22 October 2019

**Key words:**


Cognitive reserve; first episode psychosis; psychosocial functioning; social cognition

**Author for correspondence:**

I González-Ortega,

E-mail: [itxaso.gonzalezortega@osakidetza.eus](mailto:itxaso.gonzalezortega@osakidetza.eus)

# Influence of social cognition as a mediator between cognitive reserve and psychosocial functioning in patients with first episode psychosis

I González-Ortega<sup>1,2,3,4</sup> , A González-Pinto<sup>1,2,3</sup>, S Alberich<sup>1,2,3,4</sup>, E Echeburúa<sup>1,5</sup>, M Bernardo<sup>1,6</sup>, B Cabrera<sup>1,6</sup>, S Amoretti<sup>1,6</sup>, A Lobo<sup>1,7</sup>, C Arango<sup>1,8</sup>, I Corripio<sup>1,9</sup>, E Vieta<sup>1,10</sup>, E de la Serna<sup>1,11</sup>, R Rodriguez-Jimenez<sup>1,12</sup>, R Segarra<sup>1,13</sup>, JM López-Ilundain<sup>14</sup>, AM Sánchez-Torres<sup>14</sup>, MJ Cuesta<sup>14</sup>, PEPs Group: I Zorrilla<sup>1,2,3</sup>, P López<sup>1,2,3</sup>, M Bioque<sup>1,6</sup>, G Mezquida<sup>1,6</sup>, F Barcones<sup>7,15</sup>, C De-la-Cámara<sup>1,7</sup>, M Parellada<sup>1,8</sup>, A Espliego<sup>1,8</sup>, A Alonso-Solís<sup>1,9</sup>, EM Grasa<sup>1,9</sup>, C Varo<sup>10</sup>, L Montejo<sup>1,10</sup>, J Castro-Fornieles<sup>1,11</sup>, I Baeza<sup>1,11</sup>, M Dompablo<sup>1,12</sup>, I Torio<sup>1,12</sup>, A Zabala<sup>1,13</sup>, JI Eguiluz<sup>1,13</sup>, L Moreno-Izco<sup>14</sup>, J Sanjuan<sup>1,16</sup>, R Guirado<sup>17</sup>, I Cáceres<sup>18</sup>, P Garnier<sup>18</sup>, F Contreras<sup>1,19,20</sup>, J Bobes<sup>1,21</sup>, S Al-Halabi<sup>1,21,22</sup>, J Usall<sup>1,23</sup>, A Butjosa<sup>1,23</sup>, S Sarró<sup>1,24</sup>, R Landin-Romero<sup>24</sup>, A Ibáñez<sup>1,25</sup> and G Selva<sup>1,26</sup>

<sup>1</sup>Centre for Biomedical Research in the Mental Health Network (CIBERSAM), Madrid, Spain; <sup>2</sup>Department of Psychiatry, Araba University Hospital, Bioaraba Research Institute, Vitoria, Spain; <sup>3</sup>Department of Neurosciences, University of the Basque Country, Bizkaia, Spain; <sup>4</sup>The National Distance Education University (UNED), Vitoria, Spain; <sup>5</sup>Department of Personality, Assessment and Psychological Treatment, University of the Basque Country, San Sebastián, Spain; <sup>6</sup>Barcelona Clinic Schizophrenia Unit, Neuroscience Institute, Hospital Clinic of Barcelona, Barcelona, Spain; <sup>7</sup>Department of Medicine and Psychiatry, University of Zaragoza, Aragon Institute for Health Sciences (IIS Aragón), Zaragoza, Spain; <sup>8</sup>Child and Adolescent Psychiatry Department, Gregorio Marañón General University Hospital, School of Medicine, Universidad Complutense, IISGM, Madrid, Spain; <sup>9</sup>Department of Psychiatry, Institut d'Investigació Biomèdica-Sant Pau (IIB-SANT PAU), Hospital de la Santa Creu i Sant Pau, Universitat Autònoma de Barcelona (UAB), Barcelona, Spain; <sup>10</sup>Bipolar Disorders Unit, Hospital Clinic, University of Barcelona, IDIBAPS, Barcelona, Spain; <sup>11</sup>Child and Adolescent Psychiatry Service, Hospital Clinic of Barcelona, Barcelona, Spain; <sup>12</sup>12 de Octubre Hospital Research Institute (i+12), Madrid, Spain; <sup>13</sup>Department of Neurosciences, University of the Basque Country, Cruces University Hospital, Biocruces Bizkaia Health Research Institute, Vizcaya, Spain; <sup>14</sup>Department of Psychiatry, Navarre Hospital Complex, IdiSNA, Navarre Institute for Health Research, Pamplona, Spain; <sup>15</sup>Department of Family Medicine, Hospital Universitario Miguel Servet, Zaragoza, Spain; <sup>16</sup>INCLIVA, University of Valencia, Hospital Clínico Universitario de Valencia, Spain; <sup>17</sup>Neurobiology Unit, Department of Cell Biology, Interdisciplinary Research Structure for Biotechnology and Biomedicine (BIOTECMED), University of Valencia, Valencia, Spain; <sup>18</sup>Department of Psychiatry, Hospital del Mar Medical Research Institute (IMIM), Barcelona, Spain; <sup>19</sup>Psychiatry Department, Bellvitge University Hospital-IDIBELL, Barcelona, Spain; <sup>20</sup>Department of Clinical Sciences, School of Medicine, University of Barcelona, Barcelona, Spain; <sup>21</sup>Psychiatry Department, University of Oviedo, Oviedo, Spain; <sup>22</sup>Institute of Neurosciences of the Principality of Asturias, INEUROPA, Oviedo, Spain; <sup>23</sup>Research Unit, Parc Sanitari Sant Joan de Déu, Universitat de Barcelona (UB), Sant Boi de Llobregat, Barcelona, Spain; <sup>24</sup>FIDMAG Hermanas Hospitalarias Research Foundation, Barcelona, Spain; <sup>25</sup>Psychiatry Department, Ramón y Cajal University Hospital, Ramón y Cajal Health Research Institute (IRYCIS), University of Alcalá, Madrid, Spain and <sup>26</sup>Teaching Unit of Psychiatry and Psychological Medicine, Department of Medicine, University of Valencia, INCLIVA Health Research Institute, Valencia, Spain

**Abstract**

**Background.** Social cognition has been associated with functional outcome in patients with first episode psychosis (FEP). Social cognition has also been associated with neurocognition and cognitive reserve. Although cognitive reserve, neurocognitive functioning, social cognition, and functional outcome are related, the direction of their associations is not clear. Therefore, the main aim of this study was to analyze the influence of social cognition as a mediator between cognitive reserve and cognitive domains on functioning in FEP both at baseline and at 2 years.

**Methods.** The sample of the study was composed of 282 FEP patients followed up for 2 years. To analyze whether social cognition mediates the influence of cognitive reserve and cognitive domains on functioning, a path analysis was performed. The statistical significance of any mediation effects was evaluated by bootstrap analysis.

**Results.** At baseline, as neither cognitive reserve nor the cognitive domains studied were related to functioning, the conditions for mediation were not satisfied. Nevertheless, at 2

©The Author(s) 2019. This is an Open Access article, distributed under the terms of the Creative Commons Attribution licence (<http://creativecommons.org/licenses/by/4.0/>), which permits unrestricted reuse, distribution, and reproduction in any medium, provided the original work is properly cited.

years of follow-up, social cognition acted as a mediator between cognitive reserve and functioning. Likewise, social cognition was a mediator between verbal memory and functional outcome. The results of the bootstrap analysis confirmed these significant mediations (95% bootstrapped CI (-10.215 to -0.337) and (-4.731 to -0.605) respectively).

**Conclusions.** Cognitive reserve and neurocognition are related to functioning, and social cognition mediates in this relationship.

## Introduction

Neurocognition, cognitive reserve, and social cognition have all been related to functioning in first-episode psychosis (FEP). Specifically, numerous studies have found that cognitive deficits are present early in psychosis, manifesting in multiple cognitive domains including working memory, executive function, attention, processing speed, and/or learning and memory (Zabala *et al.*, 2010; González-Ortega *et al.*, 2013; Bora and Murray, 2014). These deficits have been related to poorer psychosocial functioning (Leeson *et al.*, 2011; González-Ortega *et al.*, 2013; Green and Harvey, 2014; Torgalsbøen *et al.*, 2015), but some studies have not found this association (Kravariti *et al.*, 2003; Stirling *et al.*, 2003). Overall, there is a lack of consensus on how cognitive functioning changes over the course of the disease and its relation to functional outcome.

Cognitive reserve has been associated with the functional course of FEP, patients with a higher cognitive reserve showing a better outcome (Leeson *et al.*, 2011; Amoretti *et al.*, 2016). Cognitive reserve has been defined as the ability of a brain to cope with brain pathology and thereby minimize symptoms (Stern, 2002) and variables used as measures of cognitive reserve include premorbid IQ, educational level, and occupational attainment (Anaya *et al.*, 2016).

Social cognition has also been associated with functional outcome. Indeed, it has been suggested that social cognition is a better predictor of functional outcome of FEP patients than neurocognition (Fett *et al.*, 2011; Ohmuro *et al.*, 2016). Social cognition includes theory of mind, social perception, social knowledge, attributional biases, and emotion processing, areas that are affected in FEP patients (Horan *et al.*, 2012; Bora and Pantelis, 2013; Bliksted *et al.*, 2014). It has also been associated with neurocognition and premorbid IQ (Bliksted *et al.*, 2014) and there is evidence of a relationship between social cognition and neurocognition and that this may have a negative influence on functional outcome of FEP (Schmidt *et al.*, 2011).

Although cognitive reserve, neurocognitive functioning, social cognition, and functional outcome are probably related, the direction of their associations is not clear (Fett *et al.*, 2011; Schmidt *et al.*, 2011; Pinkham and Harvey, 2012; Hedman *et al.*, 2013; Lin *et al.*, 2013). In a systematic review, Schmidt *et al.* (2011) found that social cognition acts as a mediator between neurocognition and functioning in schizophrenia (Schmidt *et al.*, 2011), though in individuals at clinical high risk of psychosis this was not observed (Barbato *et al.*, 2013).

Therefore, for predicting the outcome of FEP patients after the onset of the disease, it is important to explore the potential mediating role of social cognition in the association between neurocognition and functioning. In this context, our study had the following objectives: (1) to assess changes from baseline in cognitive functioning, social cognition, and functioning at 2 years of follow-up in FEP patients; (2) to analyze the influence of cognitive reserve and cognitive domains on social cognition and functioning at baseline

and at 2 years of follow-up; (3) to analyze the influence of social cognition on functioning; and (4) to analyze the influence of social cognition as a mediator between cognitive reserve and cognitive domains on functioning.

## Method

### Subjects

This work was part of the 'Phenotype-genotype and environmental interaction. Application of a predictive model in first psychotic episodes' study (PEPs study, from its acronym in Spanish) (Bernardo *et al.*, 2013).

Patients were aged between 18 and 35 years old at the time of first evaluation and were required to have fluent Spanish and to give written informed consent. If patients were not capable of giving consent or minors, a legally authorized representative was asked to provide consent on their behalf. Moreover, it was required that patients had a less than 12-month history of psychotic symptoms.

Exclusion criteria for patients were: mental retardation according to the DSM-IV (American Psychiatric Association, 1994) (including not only an IQ below 70, as determined using the test described in Table 1, but also poor functioning), history of head trauma with the loss of consciousness, and organic illness affecting mental health. The study was approved by the clinical research ethics committees of all participating centers in the PEPs Group.

Initially, 335 FEP patients were included in the study. Of these, 53 patients were excluded for not having seven or more neuropsychological tests completed. Thus, the final study sample consisted of 282 patients.

### Data collection

Sociodemographic and clinical data were collected at baseline and at 2 years, while cognitive assessment was performed at 2 months of follow-up, in order to ensure the psychopathological stability of patients, and at 2 years of follow-up. If patients were actively psychotic at the time of the 2-year follow-up, we delayed the assessment until their condition was stabilized. The assessment protocol was fully described by Bernardo *et al.* (2013).

Adult patients were diagnosed with FEP using the Structured Clinical Interview for DSM-IV Axis I and II Disorders (First *et al.*, 1997), while the Schedule for Affective Disorders and Schizophrenia for school-age children-Present and Lifetime version (Kaufman *et al.*, 1997) was used for those under 18 years old, following DSM-IV criteria.

Functional impairment of patients was evaluated using the functional assessment short test (FAST) (Rosa *et al.*, 2007), for which high scores suggest poorer functioning. The FAST is a measure of functioning that is valid, reliable, and sensitive to change and has been validated and widely used in bipolar

**Table 1.** Description of test and measures used for cognitive domain summary scores

Cognitive domain	Neuropsychological subtests
Premorbid IQ	Vocabulary subtest of WAIS-III <sup>a</sup> /WISC-IV <sup>b</sup>
Processing speed	Trail Making Test <sup>c</sup> -form A
	Stroop Test word and color condition <sup>d</sup>
Attention	CPT-II <sup>e</sup>
Verbal memory	TAVEC <sup>f</sup>
Working memory	Digit Span subtest of the WAIS-III <sup>a</sup> /WISC-IV <sup>b</sup>
	Letter and number sequencing of the WAIS-III <sup>a</sup> /WISC-IV <sup>b</sup>
Executive function	FAS test <sup>g</sup>
	Animal words <sup>h</sup>
	Trail Making Test <sup>c</sup> -form B
	WCST <sup>i</sup>
	Stroop Test <sup>d</sup> -interference condition

<sup>a</sup>Wechsler Adult Intelligence Scale III (WAIS-III) (Wechsler, 1997).

<sup>b</sup>Wechsler Intelligence Scale for Children-IV (WISC-IV) (Wechsler, 2003).

<sup>c</sup>Trail Making Test (Reitan and Wolfson, 1985).

<sup>d</sup>Stroop Test (Golden, 1978).

<sup>e</sup>Conners' Continuous Performance Test (CPT-II) (Conners, 2004).

<sup>f</sup>Test de Aprendizaje Verbal España-Complutense (TAVEC) (Benedet and Alejandre, 1998).

<sup>g</sup>Controlled Oral Word Association Test (FAS test) (Loonstra *et al.*, 2001).

<sup>h</sup>Animal words (Peña-Casanova, 1990).

<sup>i</sup>Wisconsin Card Sorting Test (WCST) (Heaton *et al.*, 1993).

disorder (Rosa *et al.*, 2011; Bonnín *et al.*, 2018; Vieta *et al.*, 2018), attention-deficit hyperactivity disorder (Rotger *et al.*, 2014), and FEP (González-Ortega *et al.*, 2010).

The substance use was assessed using the European Adaptation of a Multidimensional Assessment Instrument for Drug and Alcohol Dependence (EuropASI) and patients were categorized according to EuropASI scores into four groups (no use, use, abuse, and dependence) (Kokkevi and Hartgers, 1995).

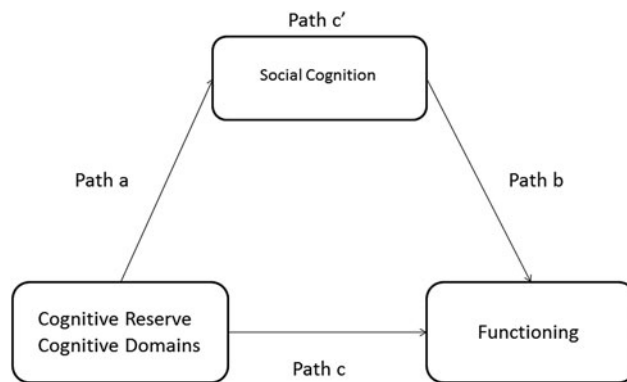
The following cognitive domains are assessed: processing speed, attention, verbal memory, working memory executive function, and premorbid IQ. The tests and measures used for domain summary scores are described by Bernardo *et al.* (2013) and Cuesta *et al.* (2015) (Table 1).

Estimated premorbid IQ, educational level, and occupational attainment were used as measures of cognitive reserve (Anaya *et al.*, 2016). IQ was evaluated with the Vocabulary subtest of Wechsler Adult Intelligence Scale (Wechsler, 1997) or Wechsler Intelligence Scale for Children-IV (Wechsler, 2003), while educational level and occupational attainment variables were assessed with the Hollingshead-Redlich Scale (Hollingshead and Redlich, 1958).

Social cognition was assessed with understanding emotions, managing emotions, and total emotional intelligence scores of the Mayer-Salovey-Caruso Emotional Intelligence Test, which rate the helpfulness of certain moods and assess the effectiveness of strategies to manage emotions (Mayer *et al.*, 2009).

### Statistical analyses

Demographic variables are described with means ( $\pm$ S.D.) and percentages. *t* Tests for paired samples were used to analyze

**Fig. 1.** Hypothesized mediation model.

differences in cognitive performance and functioning of patients between baseline and 2 years of follow-up.

To assess the cognitive domains, we calculated the average of the *z*-scores of the neuropsychological variables which correspond to each domain (Table 1). As the scores of the Trail Making Test (Reitan and Wolfson, 1985) are inverse, they were multiplied by  $-1$  to facilitate the interpretation (higher scores indicating better performance). The same approach was taken for cognitive reserve, that is, averages of the *z*-scores were calculated for estimated premorbid IQ, educational level, and occupational attainment.

To explore whether social cognition mediates the influence of cognitive reserve and cognitive domains on functioning, a three-step procedure was used (Frazier *et al.*, 2004). Figure 1 represents the hypothesized mediation model. Firstly, we analyzed the relationship between cognitive reserve and cognitive domains with social cognition with a linear regression (path a). Secondly, linear regression models were built to explore the association of cognitive reserve and cognitive domains (independent variables) with functioning (dependent variable) (path c). All models were adjusted for potential confounders (namely, age, sex, civil status, socio-economic level, educational level, diagnosis, substance use, and family history) with a stepwise procedure which only included variables in the final model if they produced a significant change in the coefficient of the independent variable. Finally, social cognition was included as a mediator in the multiple regressions found to be significant in the second step (paths b and c'). If the relation of social cognition with functioning was significant and the path c association became non-significant, conditions for mediation would be satisfied, this suggesting that social cognition is a mediating variable. The statistical significance of the mediation was examined by bootstrap analysis (95% bootstrapped confidence interval (CI)).

Data analyses were conducted with Stata 12.1. The level of significance was set at  $p < 0.05$ , except for the analyses of the cognition domains for which we applied the Bonferroni correction for multiple comparisons. For these, the level of significance was set at  $p < 0.01$ , calculated by dividing 0.05 by five, the number of cognitive domains.

## Results

### Sociodemographic characteristics, cognitive, and functional course of the sample

Baseline characteristics of the sample are summarized in Table 2. The total sample comprised 192 men (68.1%) with a mean age of

**Table 2.** Baseline characteristics of the sample

Variable	Total sample (n = 282)
Sex	
Male	192 (68.1%)
Age, years	23.64 (5.93)
Civil status	
Single	245 (86.9%)
Married	20 (7.1%)
Other	17 (6%)
Socioeconomic level	
Low	124 (44.6%)
Medium	69 (24.8%)
High	85 (30.6%)

23.64 (5.93) years. Of these, 123 (43.6%) had a diagnosis of schizophrenia and 59 (20.9%) had a diagnosis of bipolar disorder. The drug most frequently used was tobacco (66.4%) and most patients were single (86.9%).

During 2 years of follow-up, all the cognitive domain scores improved significantly, except working memory and executive function. Social cognition, cognitive reserve, and functioning were also significantly better at 2 years (Table 3).

Correlations between cognitive and functional variables are presented in a Supplementary Table.

### Relationship between cognitive reserve, social cognition, general cognition, and functioning

Table 4 reports the results of the analysis of the basic path models at both baseline and 2 years. At baseline, cognitive reserve was significantly associated with social cognition. Regarding baseline cognitive domain scores, after the Bonferroni correction, only processing speed and verbal memory showed a significant association with social cognition at baseline (path a). Social cognition was not significantly associated with functioning at baseline (path b). Neither cognitive reserve nor the cognitive domains were related to functioning at baseline (path c).

At 2 years of follow-up, in addition to baseline cognitive reserve and verbal memory, baseline executive function was also associated with social cognition at 2 years, while processing speed became non-significant at this time point (path a). Furthermore, social cognition at both baseline and 2 years was significantly associated with functioning at the end of the follow-up (path b). Cognitive reserve was significantly related to functioning at 2 years. Regarding baseline cognitive domain scores, after applying the Bonferroni correction, only attention and verbal memory were significantly related to functioning at 2 years (path c).

### The influence of social cognition as a mediator between cognitive reserve and cognitive domains on functioning

At baseline, as neither cognitive reserve nor cognitive domain scores were related to functioning (path c), the conditions for mediation were not satisfied.

**Table 3.** Means of cognition and FAST during follow-up (n = 186)

Variable	Baseline	2 years
<b>Attention</b>	<b>-0.38 (0.99)</b>	<b>-0.21 (0.87)</b>
<b>Processing speed</b>	<b>-0.96 (1.06)</b>	<b>-0.69 (1.00)</b>
<b>Verbal memory</b>	<b>-1.07 (1.24)</b>	<b>-0.65 (1.11)</b>
Working memory	-0.71 (0.76)	-0.67 (0.86)
Executive function	-0.43 (0.50)	-0.16 (0.62)
<b>Social cognition</b>	<b>-0.72 (0.89)</b>	<b>-0.58 (0.95)</b>
<b>Cognitive reserve</b>	<b>-0.45 (0.56)</b>	<b>0.04 (0.56)</b>
<b>FAST</b>	<b>26.01 (16.16)</b>	<b>19.40 (15.40)</b>

p value <0.05 except for the cognitive domains for which the Bonferroni correction was applied ( $p < 0.01$ ). Bold values represent the significant values.

At 2 years of follow-up, after including social cognition at baseline in the final model, all significant relations of baseline cognitive domains and cognitive reserve with FAST at 2 years remained significant. Further, their coefficients increased after the inclusion of social cognition, and therefore, baseline social cognition did not mediate in these relations. Nevertheless, relevant results were obtained after including social cognition at 2 years in the model. Social cognition at 2 years was a significant mediator between cognitive reserve and functioning at 2 years (Fig. 2). In fact, its association with functioning was significant (path b), while the relation between cognitive reserve and functioning became non-significant (path c'). These findings suggest a significant full mediation of social cognition, it mediating 20.8% of the effect (95% bootstrapped CI -10.215 to -0.337).

With respect to cognitive domains, after including social cognition at 2 years in the regression model between baseline attention and functioning at 2 years, social cognition had a significant association with functioning (path b) and attention remained significant, its coefficient increasing ( $b = 3.445$ ,  $p = 0.006$ ). Therefore, the conditions for the mediation of social cognition were not satisfied. Regarding baseline verbal memory, this domain lost its significant association with functioning at 2 years when including social cognition at 2 years in the model (path c'), while the latter had a significant association with functioning (path b) (Fig. 3). Thus, social cognition at 2 years appears to be a significant full mediator, it mediating 25.2% of the effect (95% bootstrapped CI -4.731 to -0.605).

## Discussion

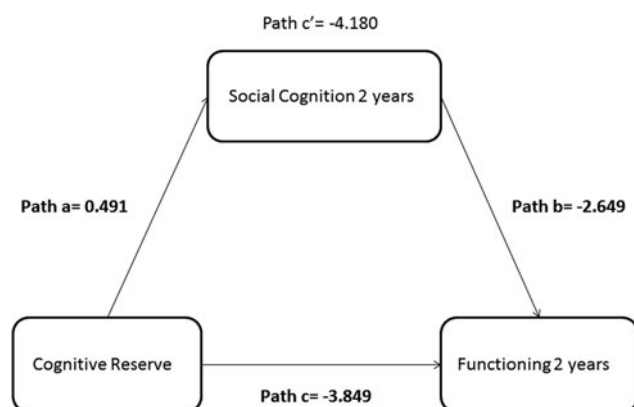
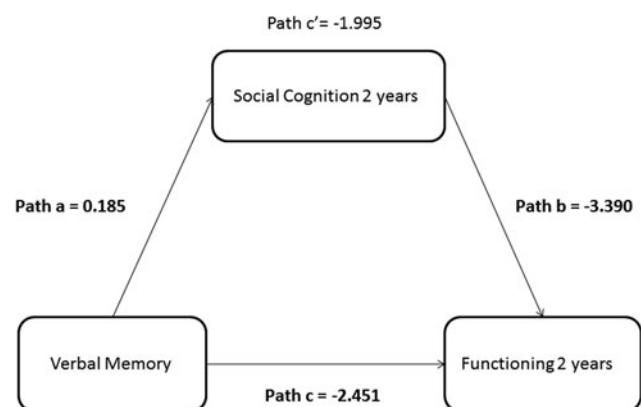
The main finding of this study is that social cognition acted as a mediator between cognitive reserve and functioning at 2 years of follow-up. Likewise, social cognition was a mediator between verbal memory and functional outcome. These findings suggest that social cognition is involved and indeed plays a crucial role in the functional outcome of FEP patients. There is little previous research on this mediation, although in a systematic review Schmidt *et al.* (2011) also found that social cognition mediated the relationship between neurocognition and functioning in schizophrenia. Our findings are from a sample of patients with FEP and take into account the role of cognitive reserve. Moreover, our study has a longitudinal design.

There is extensive evidence of social cognitive deficits in FEP (Horan *et al.*, 2012; Bora and Pantelis, 2013; Bliksted *et al.*,

**Table 4.** Statistical analyses of the paths a and b

Path a (cognitive domains/CR at baseline → social cognition)	Social cognition at baseline	Social cognition at 2 years
Cognitive reserve	<b><math>b = 0.462, p &lt; 0.001</math></b>	<b><math>b = 0.491, p &lt; 0.001</math></b>
Attention	$b = -0.016, p = 0.461$	$b = 0.057, p = 0.463$
Processing speed	<b><math>b = 0.159, p = 0.004</math></b>	$b = 0.138, p = 0.051$
Verbal memory	<b><math>b = 0.148, p = 0.002</math></b>	<b><math>b = 0.185, p = 0.003</math></b>
Working memory	$b = 0.146, p = 0.051$	$b = 0.206, p = 0.039$
Executive function	$b = 0.420, p = 0.059$	<b><math>b = 0.910, p = 0.006</math></b>
Path b (social cognition → FAST)	FAST at baseline	FAST at 2 years
Social cognition at baseline	$b = -1.862, p = 0.077$	<b><math>b = -3.410, p = 0.006</math></b>
Social cognition at 2 years	–	<b><math>b = -3.347, p = 0.007</math></b>
Path c (cognitive domains/CR at baseline → FAST)	FAST at baseline	FAST at 2 years
Cognitive reserve	$b = -2.105, p = 0.195$	<b><math>b = -3.849, p = 0.048</math></b>
Attention	$b = 0.125, p = 0.725$	<b><math>b = 3.257, p = 0.005</math></b>
Processing speed	$b = -1.347, p = 0.125$	$b = -0.961, p = 0.364$
Verbal memory	$b = -1.459, p = 0.059$	<b><math>b = -2.451, p = 0.007</math></b>
Working memory	$b = -2.881, p = 0.014$	$b = -2.062, p = 0.162$
Executive function	$b = -6.669, p = 0.057$	$b = -9.594, p = 0.016$

$p$  value  $< 0.05$  except for the cognitive domains for which the Bonferroni correction was applied ( $p < 0.01$ ). Bold values represent the significant values.

**Fig. 2.** Mediation of social cognition between cognitive reserve and functioning at 2 years.**Fig. 3.** Mediation of social cognition between verbal memory and functioning at 2 years.

2014; Langdon *et al.*, 2014; Ho *et al.*, 2015) and related conditions (Lahera *et al.*, 2015; Varo *et al.*, 2017). Impairments in social functioning have been related to deficits in emotion recognition, emotional intelligence/social perception, and social knowledge of FEP patients (Addington *et al.*, 2010), because of their difficulties managing specific situations and predicting social consequences, as well as in recognizing changes in behavioral meaning (Montreuil *et al.*, 2010). Moreover, it is considered that deterioration in social cognition has been related to poor functioning, especially poor work and social functioning as well as interpersonal and problem solving skills (Addington *et al.*, 2010; Fett *et al.*, 2011; Horan *et al.*, 2012).

A second major finding of our study is that social cognition may be a potential marker for predicting the functional outcome of patients. Previous studies have also found that social cognition may be a trait marker of psychosis (Bora *et al.*, 2008; Bora and Pantelis, 2013). In fact, it has been seen that social cognition

could be a better predictor of functional outcome than neurocognition with a significant impact on general functioning of patients (Fett *et al.*, 2011; Ohmuro *et al.*, 2016). In our sample of FEP patients, although baseline social cognition was related to functioning at 2 years, it was not a mediator between cognition and functional outcome; however, social cognition 2 years later was a significant mediator. This finding is probably related to the state and trait nature of social cognition. Social dysfunction has been described as a state marker related to symptoms of schizophrenia, and some studies suggest that it is not impaired after symptom recovery (Corcoran *et al.*, 1995; Drury *et al.*, 1998). Nonetheless, several studies have demonstrated deficits in social cognition in remitted patients (Herold *et al.*, 2002; Janssen *et al.*, 2003; Inoue *et al.*, 2006). It is possible that patients have social cognition deficits as a trait marker, and in addition, these deficits may worsen with symptoms, indicating that it is both a trait and a state variable. Moreover, social cognitive deficits are

already present even in patients at risk of psychosis (Thompson *et al.*, 2012; Ohmuro *et al.*, 2016; Zhang *et al.*, 2016) and deficits have even been found in unaffected first-degree relatives (Bora and Pantelis, 2013), this highlighting its trait properties.

On the other hand, another relevant finding of our study is that cognitive reserve and neurocognition predicted social cognition, both at baseline and 2 years. Bliksted *et al.* (2014) also found a correlation between social cognition and IQ, though they distinguish between a complex version of social cognitive deficits linked with IQ and another version related to simpler forms of social cognition that are independent of IQ. Regarding cognitive domains, processing speed and verbal memory showed a significant association with social cognition at baseline. At 2 years of follow-up, verbal memory and executive function were associated with social cognition. Previous studies have also found that cognitive functioning is related to social cognition. Theory of mind disabilities have been related to general cognitive impairment (Bora *et al.*, 2008) and more specifically, Fernandez-Gonzalo *et al.* (2013, 2014) showed that executive function was related to theory of mind in FEP patients. Further, Sachs *et al.* (2004) found a correlation between emotion discrimination and verbal memory and language processing cognitive domains.

As we hypothesized, at 2 years of follow-up there was an improvement in social cognition, cognitive functioning, and general functioning of patients. There is extensive evidence of the involvement of cognitive impairment in the functional outcome of FEP patients (Lepage *et al.*, 2014; Torgalsbøen *et al.*, 2015). Neurocognitive deficits have been identified as a core symptom of schizophrenia and may be more related to functional recovery of patients than clinical symptoms (Lepage *et al.*, 2014). It has been suggested that cognitive impairment appears soon after or even before the first episode of psychosis (Bora and Murray, 2014), and while some studies argue that there is deterioration in cognitive functions in the follow-up (Morrison *et al.*, 2006; Arango *et al.*, 2012), others postulate an improvement in cognitive function (Rodríguez-Sánchez *et al.*, 2008; Becker *et al.*, 2010). Our findings suggest that cognitive impairment in FEP is not progressive after the onset of illness, but rather improves in the follow-up, which lends support to the neurodevelopmental model of cognition. Although the effects of treatments were not analyzed in the study, it is possible that treatment effects and/or clinical stabilization contribute to cognitive improvements in FEP. In our sample of FEP patients, cognitive functioning improved significantly at the 2-year follow-up in all cognitive domains, except working memory and executive function. Similarly, Lepage *et al.* (2014) found that verbal and working memory are the cognitive domains most affected in FEP. This suggests that specific cognitive domains may be more sensitive than others for predicting long-term functional outcome, that is, some may be more impaired and may be more closely related to patient outcome. Specifically, in our FEP sample, verbal memory predicted the functional outcome at the 2-year follow-up. In relation to this, Torgalsbøen *et al.* (2015) found that attention/vigilance and verbal learning predicted remission at 6 months of follow-up, whereas attention/vigilance and working memory predicted functional outcome.

Cognitive reserve was also associated with functional outcome. This result has also been found in other studies. Specifically, it has been concluded that patients with a better cognitive reserve will have better outcomes in follow-up. Barnett *et al.* (2006) concluded that patients with better cognitive reserve could have better reasoning skills and functional

capacity to use compensatory forms and inhibit abnormal neural processing, which translates into greater insight and treatment adherence. Other studies also found that cognitive reserve can predict both the risk of illness as symptoms remit and functional improvement (Barnett *et al.*, 2006; Forcada *et al.*, 2015; Grande *et al.*, 2017). Amoretti *et al.* (2016) have shown that cognitive reserve predicts neuropsychological outcome, negative symptoms, and functioning at 2 years of follow-up, and hence, cognitive reserve can be used as a reliable indicator for the course of FEP patients (Amoretti *et al.*, 2018).

The findings of our study have important implications for clinical practice. An understanding of the association between neurocognition, cognitive reserve, and functioning and of the mediating role of social cognition is important for predicting the outcome of FEP patients after illness onset. It is essential to consider the relationship between illness outcome in patients and their IQ, both premorbid values and changes therein, to identify the need for a specific treatment to improve the functional outcome of FEP patients. Specially, patients with lower cognitive reserve may be more likely to need functional rehabilitation to help to improve their prognosis. In addition, people with higher cognitive reserve can be oriented to more 'normal' work or education. Likewise, it is essential that the treatment of these patients focuses on social cognitive deficits to improve their functional outcome.

This study has some limitations and strengths that should be considered. Our study had a follow-up of 2 years, and a longer follow-up period should be considered in future research. Another limitation is the variability across studies in how IQ is measured. Although we used criteria based on previous studies, there is no specific instrument validated to measure cognitive reserve. Finally, future studies should include a control group. The study also has important strengths. First, the results can be considered generalizable because it is a multicenter study that included a large sample of patients recruited in numerous Spanish psychiatric admission centers for acute psychosis. Second, the wide age range of the patients means that the sample is representative of the target population, with an average age ( $23.63 \pm 5.9$  years), lower than that for other studies with large FEP cohorts which did not include child and adolescent patients (OPUS trial:  $26.6 \pm 6.4$ ; Bertelsen *et al.*, 2008; and EUFEST trial:  $26 \pm 5.6$ ; Kahn *et al.*, 2008). Third, the neuropsychological battery used was extensive and covered the areas proposed by the NIMH-MATRICES consensus (except visual memory) (Kern *et al.*, 2008; Nuechterlein *et al.*, 2008; Nuechterlein and Green, 2009).

To conclude, although further studies are required, our findings suggest that cognitive reserve and neurocognition are related to functioning, and social cognition plays a mediating role.

**Supplementary material.** The supplementary material for this article can be found at <https://doi.org/10.1017/S0033291719002794>

**Acknowledgements.** We would like to thank the authors of the PEPs group who participated in the development of this manuscript.

**Financial support.** This work was supported by the Carlos III Institute of Health and European Fund for Regional Development (PI08/1213, PI11/01977, PI14/01900, PI08/01026, PI11/02831, PI14/01621, PI08/1161, PI16/00359, PI16/01164, PI18/00805), the Basque Foundation for Health Innovation and Research (BIOEF), the Secretaria d'Universitats i Recerca del Departament d'Economia i Coneixement (2017 SGR 1365), and R&D activities in Biomedicine, Madrid Regional Government and Structural Funds of the European Union (S2017/BMD-3740 (AGES-CM 2-CM)).

**Conflict of interest.** A González-Pinto has received grants from or served as consultant, advisor or CME speaker for the following entities: Almirall, AstraZeneca, Bristol-Myers Squibb, Cephalon, Eli Lilly, Glaxo-Smith-Kline, Janssen-Cilag, Ferrer, Johnson & Johnson, Lundbeck, Merck, Otsuka, Pfizer, Sanofi-Aventis, Servier, Shering-Plough, Solvay, the Spanish Ministries of Science and Innovation (through CIBERSAM) and of Science (through the Carlos III Institute of Health), the Basque Government, the Stanley Medical Research Institute, and Wyeth.

C Arango has been a consultant to or has received fees or grants from Abbot, Amgen, AstraZeneca, Bristol-Myers-Squibb, Caja Navarra, CIBERSAM, the Alicia Koplowitz Foundation, the Carlos III Institute of Health, Janssen Cilag, Lundbeck, Merck, the Spanish Ministries of Science and Innovation, of Health, and of Economy and Competitiveness, Mutua Madrileña, Otsuka, Pfizer, Roche, Servier, Shire, Takeda, and Schering-Plough.

E Vieta has received grants from or served as consultant, advisor or CME speaker for the following entities: AB-Biotics, Allergan, Angelini, Ferrer, Gedeon Richter, Janssen, Lundbeck, Otsuka, Sanofi-Aventis, Sunovion, the Carlos III Institute of Health, the European Union's 7th Framework Program and Horizon 2020, the Brain and Behaviour Foundation (NARSAD), and the Stanley Medical Research Institute.

R Rodríguez-Jimenez has been a consultant for, spoken in activities of, or received grants from: the Carlos III Institute of Health, the Spanish Health Research Fund (FIS), CIBERSAM, Madrid Regional Government (S2010/BMD-2422 AGES), JanssenCilag, Lundbeck, Otsuka, Pfizer, Ferrer, Juste, and Takeda

The other authors have no conflicts of interest to declare.

## References

- Addington J, Girard TA, Christensen BK and Addington D** (2010) Social cognition mediates illness-related and cognitive influences on social function in patients with schizophrenia-spectrum disorders. *Journal of Psychiatry and Neurosciences* **35**, 49–54.
- American Psychiatric Association** (1994) *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV)*, 4th Edn. Washington, DC: APA.
- Amoretti S, Bernardo M, Bonnin CM, Bioque M, Cabrera B, Mezquida G, Solé B, Vieta E and Torrent C** (2016) The impact of cognitive reserve in the outcome of first-episode psychoses: 2-year follow-up study. *European Neuropsychopharmacology* **26**, 1638–1648.
- Amoretti S, Cabrera B, Torrent C, Mezquida G, Lobo A, González-Pinto A, Parellada M, Corripio I, Vieta E, de la Serna E, Butjosa A, Contreras F, Sarró S, Penadés R, Sánchez-Torres AM, Cuesta M, Bernardo M and PEPsGroup** (2018) Cognitive reserve as an outcome predictor: first-episode affective versus non-affective psychosis. *Acta Psychiatrica Scandinavica* **138**, 441–455.
- Anaya C, Torrent C, Caballero FF, Vieta E, Bonnin CM and Ayuso-Mateos JL, CIBERSAM Functional Remediation Group** (2016) Cognitive reserve in bipolar disorder: relation to cognition, psychosocial functioning and quality of life. *Acta Psychiatrica Scandinavica* **133**, 386–398.
- Arango C, Rapado-Castro M, Reig S, Castro-Fornieles J, Gonzalez-Pinto A, Otero S, Baeza I, Moreno C, Graell M, Janssen J, Parellada M, Moreno D, Bargallo N and Desco M** (2012) Progressive brain changes in children and adolescents with first-episode psychosis. *Archives of General Psychiatry* **69**, 16–26.
- Barbato M, Liu L, Penn DL, Keefe RSE, Perkins DO, Woods SW and Addington J** (2013) Social cognition as a mediator between neurocognition and functional outcome in individuals at clinical high risk for psychosis. *Schizophrenia Research* **150**, 542–546.
- Barnett JH, Salmond CH, Jones PB and Sahakian BJ** (2006) Cognitive reserve in neuropsychiatry. *Psychological Medicine* **36**, 1053–1064.
- Becker HE, Nieman DH, Wiltink S, Dingemans PM, van de Fliert JR, Velthorst E and Linszen DH** (2010) Neurocognitive functioning before and after the first psychotic episode: does psychosis result in cognitive deterioration? *Psychological Medicine* **40**, 1599–1606.
- Benedict MJ and Alejandre MA** (1998) *Test de Aprendizaje Verbal Española-Completense (TAVEC)*. Madrid, Spain: Tea Ediciones.
- Bernardo M, Bioque M, Parellada M, Saiz-Ruiz J, Cuesta MJ, Llerena A, Sanjuán J, Castro-Fornieles J, Arango C and Cabrera B, PEPs Group** (2013) Assessing clinical and functional outcomes in a gene-environment interaction study in first episode of psychosis (PEPs). *Revista de Psiquiatría y Salud Mental* **6**, 4–16.
- Bertelsen M, Jeppesen P, Petersen L, Thorup A, Øhlenschlaeger J, le Quach P, Christensen TØ, Krarup G, Jørgensen P and Nordentoft M** (2008) Five-year follow-up of a randomized multicenter trial of intensive early intervention vs. standard treatment for patients with a first episode of psychotic illness: the OPUS trial. *Archives of General Psychiatry* **65**, 762–771.
- Bliksted V, Fagerlund B, Weed E, Frith C and Videbech P** (2014) Social cognition and neurocognitive deficits in first-episode schizophrenia. *Schizophrenia Research* **153**, 9–17.
- Bonnin CM, Martínez-Arán A, Reinares M, Valentí M, Solé B, Jiménez E, Montejo L, Vieta E and Rosa AR** (2018) Thresholds for severity, remission and recovery using the functioning assessment short test (FAST) in bipolar disorder. *Journal of Affective Disorders* **240**, 57–62.
- Bora E and Murray RM** (2014) Meta-analysis of cognitive deficits in ultra-high risk to psychosis and first-episode psychosis: do the cognitive deficits progress over, or after, the onset of psychosis? *Schizophrenia Bulletin* **40**, 744–755.
- Bora E and Pantelis C** (2013) Theory of mind impairments in first-episode psychosis, individuals at ultra-high risk for psychosis and in first-degree relatives of schizophrenia: systematic review and meta-analysis. *Schizophrenia Research* **144**, 31–36.
- Bora E, Gokcen S, Kayahan B and Veznedaroglu B** (2008) Deficits of social-cognitive and social-perceptual aspects of theory of mind in remitted patients with schizophrenia. Effect of residual symptoms. *The Journal of Nervous and Mental Disease* **196**, 95–99.
- Conners CK** (2004) *Continuous Performance Test II*. Toronto, Canada: Multi-Health Systems Inc.
- Corcoran R, Mercer G and Frith CD** (1995) Schizophrenia, symptomatology and social inference: investigating 'theory of mind' in people with schizophrenia. *Schizophrenia Research* **17**, 5–13.
- Cuesta MJ, Sánchez-Torres AM, Cabrera B, Bioque M, Merchán-Naranjo J, Corripio I, González-Pinto A, Lobo A, Bombín I, de la Serna E, Sanjuán J, Parellada M, Saiz-Ruiz J and Bernardo M, PEPs Group** (2015) Premorbid adjustment and clinical correlates of cognitive impairment in first-episode psychosis. The PEPsCog Study. *Schizophrenia Research* **164**, 5–73.
- Drury VM, Robinson EJ and Birchwood M** (1998) 'Theory of mind' skills during an acute episode of psychosis and following recovery. *Psychological Medicine* **28**, 1101–1112.
- Fernandez-Gonzalo S, Pousa E, Jodar M, Turon M, Duño R and Palao D** (2013) Influence of the neuropsychological functions in theory of mind in schizophrenia: the false-belief/deception paradigm. *The Journal of Nervous and Mental Disease* **201**, 609–613.
- Fernandez-Gonzalo S, Jodar M, Pousa E, Turon M, Garcia R, Rambla CH and Palao D** (2014) Selective effect of neurocognition on different theory of mind domains in first-episode psychosis. *Journal of Nervous & Mental Disease* **202**, 576–582.
- Fett AK, Viechtbauer W, Dominguez MD, Penn DL, van Os J and Krabbendam L** (2011) The relationship between neurocognition and social cognition with functional outcomes in schizophrenia: a meta-analysis. *Neuroscience & Biobehavioral Reviews* **35**, 573–588.
- First MB, Spitzer R and Gibbon M** (1997) *Structured Clinical Interview for DSM-IV Axis I Disorders*. Washington, DC: American Psychiatric Press Inc.
- Forcada I, Mur M, Mora E, Vieta E, Bartrés-Faz D and Portella MJ** (2015) The influence of cognitive reserve on psychosocial and neuropsychological functioning in bipolar disorder. *European Neuropsychopharmacology* **25**, 214–222.
- Frazier PA, Tix AP and Barron KE** (2004) Testing moderator and mediator effects in counseling psychology research. *Journal of Counseling Psychology* **51**, 115–134.
- Golden C** (1978) *The Stroop Color and Word Test: A Manual for Clinical and Experimental Uses*. Chicago: Stoelting Co.

- González-Ortega I, Rosa A, Alberich S, Barbeito S, Vega P, Echeburúa E, Vieta E and González-Pinto A (2010) Validation and use of the functioning assessment short test in first psychotic episodes. *The Journal of Nervous and Mental Disease* **198**, 836–840.
- González-Ortega I, de Los Mozos V, Echeburúa E, Mezo M, Besga A, Ruiz de Azúa S, González-Pinto A, Gutierrez M, Zorrilla I and González-Pinto A (2013) Working memory as a predictor of negative symptoms and functional outcome in first episode psychosis. *Psychiatry Research* **206**, 8–16.
- Grande I, Sanchez-Moreno J, Sole B, Jimenez E, Torrent C, Bonnin CM, Varo C, Tabares-Seisdedos R, Balanzá-Martínez V, Valls E, Morilla I, Carvalho AF, Ayuso-Mateos JL, Vieta E and Martínez-Aran A (2017) High cognitive reserve in bipolar disorders as a moderator of neurocognitive impairment. *Journal of Affective Disorders* **208**, 621–627.
- Green MF and Harvey PD (2014) Cognition in schizophrenia: past, present, and future. *Schizophrenia Research: Cognition* **1**, e1–e9.
- Heaton RK, Chelune GJ, Talley JL, Kay GG and Curtiss G (1993) *Wisconsin Card Sorting Test Manual (Revised and Expanded)*. Odessa, FL: PAR Psychological Assessment Resources, Inc.
- Hedman AM, van Haren NE, van Baal CG, Kahn RS and Hulshoff Pol HE (2013) IQ change over time in schizophrenia and healthy individuals: a meta-analysis. *Schizophrenia Research* **146**, 201–208.
- Herold R, Tenyi T, Lenard K and Trixler M (2002) Theory of mind deficit in people with schizophrenia during remission. *Psychological Medicine* **32**, 1125–1129.
- Ho KK, Lui SS, Hung KS, Wang Y, Li Z, Cheung EF and Chan RC (2015) Theory of mind impairments in patients with first-episode schizophrenia and their unaffected siblings. *Schizophrenia Research* **166**, 1–8.
- Hollingshead A and Redlich F (1958) *Class and Mental Illness*. New York: Wiley.
- Horan WP, Green MF, DeGroot M, Fiske A, Helleman G, Kee K, Kern RS, Lee J, Sergi MJ, Subotnik KL, Sugar CA, Ventura J and Nuechterlein KH (2012) Social cognition in schizophrenia, Part 2: 12-month stability and prediction of functional outcome in first-episode patients. *Schizophrenia Bulletin* **38**, 865–872.
- Inoue Y, Yamada K, Hirano M, Shinohara M, Tamaoki T, Iguchi H and Tonooka Y (2006) Impairment of theory of mind in patients in remission following first episode of schizophrenia. *Journal of European Archives of Psychiatry and Clinical Neuroscience* **256**, 326–328.
- Janssen I, Krabbendam L, Jolles J and Os JV (2003) Alterations in theory of mind in patients with schizophrenia and non-psychotic relatives. *Acta Psychiatrica Scandinavica* **108**, 110–117.
- Kahn RS, Fleischhacker WW, Boter H, Davidson M, Davidson M, Vergouwe Y, Keet IP, Gheorghe MD, Rybakowski JK, Galderisi S, Libiger J, Hummer M, Dollfus S, López-Ibor JJ, Hranov LG, Gaebel W, Peuskens J, Lindfors N, Riecher-Rössler A, Grobbee DE and EUFEST study group (2008) Effectiveness of antipsychotic drugs in first episode schizophrenia and schizophreniform disorder: an open randomized clinical trial. *Lancet* **371**, 1085–1097.
- Kaufman J, Birmaher B, Brent D, Rao U, Flynn C, Moreci P, Williamson D and Ryan N (1997) Schedule for affective disorders and schizophrenia for school-age children-present and lifetime version (K-SADS-PL): initial reliability and validity data. *Journal of the American Academy of Child and Adolescent Psychiatry* **36**, 980–988.
- Kern RS, Nuechterlein KH, Green MF, Baade LE, Fenton WS, Gold JM, Keefe RS, Mesholam-Gately R, Mintz J, Seidman LJ, Stover E and Marder SR (2008) The MATRICS consensus cognitive battery, part 2: co-norming and standardization. *American Journal of Psychiatry* **165**, 214–220.
- Kokkevi A and Hartgers C (1995) EuropASI: European adaptation of a multi-dimensional assessment instrument for drug and alcohol dependence. *European Addiction Research* **1**, 208–210.
- Kravariti E, Morris RG, Rabe-Hesketh S, Murray RM and Frangou S (2003) The Maudsley early onset schizophrenia study: cognitive function in adolescents with recent onset schizophrenia. *Schizophrenia Research* **61**, 137–148.
- Lahera G, Herrera S, Reinas M, Benito A, Rullas M, González-Cases J and Vieta E (2015) Hostile attributions in bipolar disorder and schizophrenia contribute to poor social functioning. *Acta Psychiatrica Scandinavica* **131**, 472–482.
- Langdon R, Still M, Connors MH, Ward PB and Catts SV (2014) Theory of mind in early psychosis. *Early Intervention in Psychiatry* **8**, 286–290.
- Leeson VC, Sharma P, Harrison M, Ron MA, Barnes TR and Joyce EM (2011) IQ trajectory, cognitive reserve, and clinical outcome following a first episode of psychosis: a 3-year longitudinal study. *Schizophrenia Bulletin* **37**, 768–777.
- Lepage M, Bodnar M and Bowie CR (2014) Neurocognition: clinical and functional outcomes in schizophrenia. *The Canadian Journal of Psychiatry* **59**, 5–12.
- Lin CH, Huang CL, Chang YC, Chen PW, Lin CY, Tsai GE and Lane HY (2013) Clinical symptoms, mainly negative symptoms, mediate the influence of neurocognition and social cognition on functional outcome of schizophrenia. *Schizophrenia Research* **146**, 231–237.
- Loonstra AS, Tarlow AR and Sellers AH (2001) COWAT metanorms across age, education, and gender. *Applied Neuropsychology* **8**, 161–166.
- Mayer JD, Salovey P and Caruso DR (2009) *Mayer-Salovey-Caruso Emotional Intelligence Test (Spanish Version)*. Madrid: TEA Ediciones.
- Montreuil T, Bodnar M, Bertrand MC, Malla AK, Joobar R and Lepage M (2010) Social cognitive markers of short-term clinical outcome in first-episode psychosis. *Clinical Schizophrenia & Related Psychoses* **4**, 105–114.
- Morrison G, O'Carroll R and McCreadie R (2006) Long-term course of cognitive impairment in schizophrenia. *The British Journal of Psychiatry* **189**, 556–557.
- Nuechterlein KH and Green MF (2009) MATRICS. Consensus cognitive battery, norwegian version. In Rund BR and Sundet KS Trans (eds), *MATRICS Assessment*. Los Angeles, LA: Inc.
- Nuechterlein KH, Green MF, Kern RS, Baade LE, Barch DM, Cohen JD, Essock S, Fenton WS, Frese III FJ, Gold JM, Goldberg T, Heaton RK, Keefe RS, Kraemer H, Mesholam-Gately R, Seidman LJ, Stover E, Weinberger DR, Young AS, Zalcman S and Marder SR (2008) The MATRICS consensus cognitive battery, part 1: test selection, reliability, and validity. *American Journal of Psychiatry* **165**, 203–213.
- Ohmuro N, Katsura M, Obara C, Kikuchi T, Sakuma A, Iizuka K, Hamaie Y, Ito F, Matsuoka H and Matsumoto K (2016) Deficits of cognitive theory of mind and its relationship with functioning in individuals with an at-risk mental state and first-episode psychosis. *Psychiatry Research* **243**, 318–325.
- Peña-Casanova J (1990) *Test Barcelona*. Barcelona: Masson.
- Pinkham AE and Harvey PD (2012) Future directions for social cognitive interventions in schizophrenia. *Schizophrenia Bulletin* **39**, 499–500.
- Reitan RM and Wolfson D (1985) *The Halstead-Reitan Neuropsychological Test Battery: Therapy and Clinical Interpretation*. Tucson, AZ: Neuropsychological Press.
- Rodríguez-Sánchez JM, Pérez-Iglesias R, González-Blanch C, Pelayo-Terán JM, Mata I, Martínez O, Sánchez-Cubillo I, Vázquez-Barquero JL and Crespo-Facorro B (2008) 1-year follow-up study of cognitive function in first-episode non-affective psychosis. *Schizophrenia Research* **104**, 165–174.
- Rosa AR, Sánchez-Moreno J, Martínez-Aran A, Salamero M, Torrent C, Reinares M, Comes M, Colom F, Van Riel W, Ayuso-Mateos JL, Kapczinski F and Vieta E (2007) Validity and reliability of the Functioning Assessment Short Test (FAST) in bipolar disorder. *Clinical Practice & Epidemiology in Mental Health* **7**, 3–5.
- Rosa AR, Reinares M, Amann B, Popovic D, Franco C, Comes M, Torrent C, Bonnin CM, Solé B, Valentí M, Salamero M, Kapczinski F and Vieta E (2011) Six-month functional outcome of a bipolar disorder cohort in the context of a specialized-care program. *Bipolar Disorders* **13**, 679–686.
- Rotger S, Richarte V, Nogueira M, Corrales M, Bosch R, Vidal R, Marfil L, Valero S, Vieta E, Goikolea JM, Torres I, Rosa A, Mur M, Casas M and Ramos-Quiroga JA (2014) Functioning Assessment Short Test (FAST): validity and reliability in adults with attention-deficit/hyperactivity disorder. *European Archives of Psychiatry and Clinical Neuroscience* **264**, 719–727.



- Sachs G, Steger-Wuchse D, Kryspin-Exner I, Gur RC and Katschnig H (2004) Facial recognition deficits and cognition in schizophrenia. *Schizophrenia Research* **68**, 27–35.
- Schmidt SJ, Mueller DR and Roder V (2011) Social cognition as a mediator variable between neurocognition and functional outcome in schizophrenia: empirical review and new results by structural equation modeling. *Schizophrenia Bulletin* **37**(suppl. 2), S41–S54.
- Stern Y (2002) What is cognitive reserve? Theory and research application of the reserve concept. *Journal of the International Neuropsychological Society* **8**, 448–460.
- Stirling J, White C, Lewis S, Hopkins R, Tantam D, Huddy A and Montague L (2003) Neurocognitive function and outcome in first-episode schizophrenia: a 10-year follow-up of an epidemiological cohort. *Schizophrenia Research* **65**, 75–86.
- Thompson A, Papas A, Bartholomeusz C, Allott K, Amminger GP, Nelson B, Wood S and Yung A (2012) Social cognition in clinical “at risk” for psychosis and first episode psychosis populations. *Schizophrenia Research* **141**, 204–209.
- Torgalsboen AK, Mohn C, Czajkowski N and Rund BR (2015) Relationship between neurocognition and functional recovery in first-episode schizophrenia: results from the second year of the Oslo multi-follow-up study. *Psychiatry Research* **227**, 185–191.
- Varo C, Jiménez E, Solé B, Bonnin CM, Torrent C, Valls E, Morilla I, Lahera G, Martínez-Arán A, Vieta E and Reinares M (2017) Social cognition in bipolar disorder: focus on emotional intelligence. *Journal of Affective Disorders* **1**, 210–217.
- Vieta E, Berk M, Schulze TG, Carvalho AF, Suppes T, Calabrese JR, Gao K, Miskowiak KW and Grande I (2018) Bipolar disorders. *Nature Reviews Disease Primers* **4**, 18008.
- Wechsler D (1997) *Wechsler Adult Intelligence Scale*, 3rd Edn. (Administration and Scoring Manual). San Antonio, TX, USA: The Psychological Corporation.
- Wechsler D (2003) *Wechsler Intelligence Scale for Children-IV (WISC-IV)*, 4th Edn. San Antonio, TX: Psychological Corporation.
- Zabala A, Rapado M, Arango C, Robles O, de la Serna González C, Rodríguez-Sánchez JM, Andrés P, Mayoral M and Bombín I (2010) Neuropsychological functioning in early-onset first-episode psychosis: comparison of diagnostic subgroups. *European Archives of Psychiatry and Clinical Neuroscience* **260**, 225–233.
- Zhang T, Yi Z, Li H, Cui H, Tang Y, Lu X, Xu L, Qian Z, Zhu Y, Jiang L, Chow A, Li C, Jiang K, Xiao Z and Wang J (2016) Faux pas recognition performance in a help-seeking population at clinical high risk of psychosis. *European Archives of Psychiatry and Clinical Neurosciences* **266**, 71–78.